School of Psychiatry

PHASE 3

MFAC3503: Psychiatry

Tutorial Readings for Staff 2011
Schizophrenia


Neurobiology of Schizophrenia

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With its hallucinations, delusions, thought disorder, and cognitive deficits, schizophrenia affects the most basic human processes of perception, emotion, and judgment. Evidence increasingly suggests that schizophrenia is a subtle disorder of brain development and plasticity. Genetic studies are beginning to identify proteins of candidate genetic risk factors for schizophrenia, including dysbindin, neuregulin 1, DAOA, COMT, and DISC1, and neurobiological studies of the normal and variant forms of these genes are now well justified. We suggest that DISC1 may offer especially valuable insights. Mechanistic studies of the properties of these candidate genes and their protein products should clarify the molecular, cellular, and systems-level pathogenesis of schizophrenia. This can help redefine the schizophrenia phenotype and shed light on the relationship between schizophrenia and other major psychiatric disorders. Understanding these basic pathologic processes may yield novel targets for the development of more effective treatments.

Introduction
Schizophrenia, affecting about 0.5 to 1.0 percent of the population worldwide with devastating consequences for affected individuals and their families, is the seventh most costly medical illness to our society (Freedman, 2003). The available symptomatic treatment is only partially successful, and therefore the development of rational therapeutics, based on an understanding of the etiology and pathogenesis of schizophrenia, is imperative. However, until recently, progress in schizophrenia has been painfully slow and limited by a number of factors, including the heterogeneity of the schizophrenia phenotype and the lack of clear pathological lesions like those that have provided reference points in the study of Alzheimer’s disease (AD), Parkinson’s disease (PD), and other neurodegenerative disorders (Ross and Margolis, 2005). Investigation into the mechanism of action of the drugs used to treat schizophrenia has not provided clear understanding of the pathogenesis of the disease. While schizophrenia is highly heritable (it has a heritability score of approximately 0.8), the genetics are complex and the interpretation of genetic data has proven difficult. Now, however, advances in phenotypic analysis, neuroimaging, genetics, and molecular pathology provide the basis for optimism. Schizophrenia can be understood, at least in part, as a subtle disorder of brain development (Arnold et al., 2005; Harrison and Weinberger, 2005; Rapoport et al., 2005). Evidence now supports an etiologic role for mutations or polymorphisms in a number of genes (Chen et al., 2006; Craddock et al., 2006; Owen et al., 2005; Riley and Kendler, 2006), as well as obstetrical and premorbid abnormalities of development and cognition. We argue in this review that a definitive study of the neurobiology of schizophrenia is now possible.

Lessons from Neurodegenerative Diseases
The success in understanding etiology and pathogenesis of neurodegenerative disorders such as AD, PD, and Huntington’s disease and related polyglutamine diseases suggests some potential lessons for schizophrenia. First, even for complex diseases, there can be tremendous benefit from understanding rare familial variants (Ross and Margolis, 2005). Schizophrenia is likely to be more complicated than the neurodegenerative disorders, since the search for Mendelian variants has been less rewarding. But possibly other chromosomal translocations (see below), as well as the identification of DISC1, suggests that this approach may yet be fruitful. Second, identification of more than one causative gene may help define a pathogenic pathway, and therapeutic targets, via the interaction of gene products. For instance, presenilin 1 and presenilin 2 mutations both cause familial AD through aberrant cleavage of the APP protein. Similarly, understanding the interactions of gene products mutated in genetic PD is beginning to elucidate the pathogenesis of familial, and potentially sporadic, PD (Smith et al., 2005). Third, with the identification of the genetic causes of neurodegenerative diseases, commonalities among the different disorders are now emerging, such as the presence of inclusion bodies and other deposits of aggregated protein (Ross and Poirier, 2005). Fourth, mutations that increase the risk of developing a disease but are not by themselves causative can also be illuminating. For instance, ApoE polymorphisms, which influence the risk for AD, appear to alter the metabolism of the A-Beta peptide, providing additional insight into AD pathogenesis. Finally, genetic changes need not be point mutations, frame shifts, or deletions. RNA as well as protein can be neurotoxic (Margolis et al., 2006). Diseases can also be caused by alterations in the dosage of genes, such as the duplications and triplications of α-synuclein that cause familial PD (Singleton et al., 2004). More subtle alterations in levels of expression may also increase susceptibility to PD (Singleton et al., 2004) and AD.

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Lessons from Developmental Diseases

Schizophrenia is increasingly viewed as a subtle disorder of neurodevelopment. A chromosome 22 microdeletion termed Velocardiofacial Syndrome (VCFS) is associated with schizophrenia. As described below, it may offer clues to schizophrenia’s pathogenesis.

We suggest that the severe disorders of cortical development, grouped together as the lissencephalies, may also provide clues to the etiology and pathogenesis of schizophrenia. Lissencephaly involves severe abnormalities of the normal “inside out” development of the cerebral cortex. Neurons migrate from the ventricular zone toward the pial surface, guided by radial glia, directed in part by secretion of Reelin by Cajal-Retzius or subpial granular layer cells. Migration of the neuronal cell body is mediated via microtubule-based transport organized by the centrosome. First the centrosome moves up the microtubules, followed by the nucleus and the cell body (D’Arcangelo, 2006; Hatten, 2002; Kato and Dobyns, 2003; Olson and Walsh, 2002; Tsai and Gleeson, 2005).

Reelin is believed to have a key role in directing cortical neuronal migration. Mutations in Reelin are one cause of lissencephaly. Other major genes whose mutations can cause lissencephaly are Lis1 and doublecortin (DCX), both of which are involved in regulation of microtubule-based transport. The potential roles of these molecules in the more subtle abnormalities of neuronal migration and positioning detected in schizophrenia and in models of DISC1 mutation are described below. Furthermore, the example of lissencephaly is another example, like that of familial AD, of the utility of knowing several genes, which, when mutated, lead to similar phenotypes. Also, the N-methyl-D-aspartate (NMDA) receptor has been shown to stimulate neuronal migration, so that impaired function of this receptor could contribute to the developmental phenotype. With mutations in several genes leading to the same phenotype, it becomes possible to identify relationships among their protein products and ultimately piece together the framework of a pathogenic pathway.

Schizophrenia Clinical Features and Therapeutics

Schizophrenia is a heterogeneous syndrome without any single defining symptom or sign and is unidentifiable with any known diagnostic laboratory tests. The diagnosis is applied to individuals with psychotic phenomena (hallucinations, delusions, and thought disorder) after other causes of psychosis, such as affective disorder or delirium, have been excluded. Many individuals with schizophrenia exhibit negative symptoms, including diminished emotional expression and reaction, diminished participation in interpersonal relationships, diminished production of speech, and apathy, with loss of energy, drive, and interests. While less striking than positive symptoms, negative symptoms may be more impairing and less responsive to treatment. The symptom profiles of bipolar disorder (which involves dramatic alterations of mood, with psychotic phenomena as a frequent accompaniment) and schizophrenia frequently overlap.

The success of genetic and neurobiological investigations of schizophrenia is likely to be dependent on understanding the heterogeneity of schizophrenia. One approach has been to divide patients into subtypes based on their predominant clinical manifestations. For instance, the 25%–30% of individuals with chronic schizophrenia who have predominantly negative symptoms (Kirkpatrick et al., 2001) have been defined as having “deficit” schizophrenia. However, other attempts to subtype schizophrenia in the past have not been very fruitful, so caution should be exercised. The use of dimensionally distinctive features, such as negative symptoms or cognitive abnormalities, as quantitative traits may be more productive.

The onset of schizophrenia most commonly occurs in the second or third decade of life, though onset age may vary from childhood to old age. Subtle abnormalities of cognition, social interaction, motor function, and physical morphology are frequently observed in individuals who later develop schizophrenia (Niemi et al., 2003), which is suggestive of a developmental vulnerability.

Clinical Features: Endophenotypes

An alternative approach to classification of heterogeneous disorders is to define endophenotypes (or intermediate phenotypes) (Cannon, 2005; Gottesman and Gould, 2003). These are heritable, and often quantitative, traits that may not be readily apparent in routine clinical examinations of affected individuals, yet may reflect neurobiological features underlying the disease and may be useful in genetic linkage studies. Ideally, endophenotypes in schizophrenia will reflect abnormalities of specific neural systems under relatively simple genetic control. Valid endophenotypes will associate with schizophrenia in population studies, will be present (though less prominent) in the first degree family members of probands with schizophrenia, and will be found at similar levels in both members of twins discordant for schizophrenia.

A variety of potential endophenotypes have been associated with schizophrenia, though none has yet been confirmed in large, unselected samples of at-risk individuals. For instance, disordered eye movements, which can be measured using quantitative methods, include antisaccade performance (associated with frontal-striatal function) (Ettinger et al., 2006) and abnormal smooth pursuit eye movements, especially the predictive pursuit component of this function (Hong et al., 2006). Attenuated inhibition of the P50 auditory event-related potential, a sensory motor gating task, may reflect deficits in attention and vigilance (Erwin et al., 1998). The P300 event-related potential, a measurement of cortical activity taken during stimuli discrimination tasks that also reflects attention and working memory, is attenuated both in individuals with schizophrenia and, to an intermediate extent, in their relatives (Bramon et al., 2006). Structural and functional neuroanatomic deficits, as revealed by imaging studies, have also been proposed as endophenotypes.

Clinical Features: Neuropsychology

While the psychotic phenomena of schizophrenia are striking, more subtle cognitive problems are increasingly recognized as central to the disease. Impairments in cognition include attention, working memory, learning, verbal fluency, motor speed, and executive functions. While positive and negative symptoms of schizophrenia can fluctuate, cognitive deficits remain relatively
stable, and are already apparent in first-episode pa-
tients who have never received antipsychotic medicines
(Harvey et al., 2003). Cognitive deficits are found in the
biological relatives of subjects with schizophrenia (Snitz
et al., 2006), suggesting that aspects of cognition im-
paired in schizophrenia may be under specific genetic
control, and therefore, serve as informative endopheno-
types in the genetic analysis of schizophrenia. Cognitive
dysfunction has been recognized as a core feature of
schizophrenia (Antonova et al., 2004; Gold, 2004), lead-
ing to impairment of skills and diminished functional
capacity (Bowie and Harvey, 2005).

The National Institute of Mental Health (NIMH)-spon-
sored Measurement and Treatment Research to Im-
prove Cognition in Schizophrenia (MATRICS) initiative
(Nuechterlein et al., 2004) is developing a consensus
around a cognitive battery for use in clinical trials in
schizophrenia. It incorporates seven cognitive domains,
including Speed of Processing, Attention/Vigilance,
Working Memory, Verbal Learning and Memory, Visual
Learning and Memory, Reasoning and Problem Solving,
and Social Cognition.

Working memory dysfunction in schizophrenia has
been linked to dysfunction of the dorsolateral prefrontal
cortex (DLPFC) (Goldman-Rakic, 1999). Even schizo-
phrenia patients with good performance on working
memory tasks are inefficient in their use of prefrontal net-
works. Behavioral strategies for cognitive improvement
can be effective in improving neurocognitive deficits.

Clinical Features: Neuroimaging
Recent advances in imaging technology (such as fMRI
and diffusion tensor imaging, or DTI) have enabled in-
vestigators to move beyond measures of isolated re-
gional abnormalities and instead begin the exploration
of the function and structure of the interconnected
neural networks that are implicated in schizophrenia.

The most consistent structural abnormalities found in
schizophrenia include lateral and third ventricular en-
largement; medial temporal lobe (hippocampal forma-
tion, subiculum, parahippocampal gyrus) volume reduc-
tions; and superior temporal gyrus (STG) volume reduc-
tions, particularly on the left (Figure 1). There is
also moderate evidence for frontal lobe volume reduc-
tion, particularly of prefrontal and orbitofrontal regions,
and parietal lobe abnormalities. Enlarged cavum septi
pellucidi, basal ganglia abnormalities, corpus callosum
abnormalities, thalamus abnormalities, and cerebellar
abnormalities are also evident (Antonova et al., 2004;
Honea et al., 2005; Niznikiewicz et al., 2003). Some, but
not all, studies have suggested that structural changes
may be progressive (Rapoport et al., 2005).

Structural neuroimaging suggests that abnormal pro-
cesses in schizophrenia occur at different stages of neu-
rodevelopment. There is evidence for an early neurode-
velopmental lesion (pre- or perinatal) that may render the
brain vulnerable to anomalous late neurodevelop-
mental processes (particularly postpubertal); these
anomalous late neurodevelopmental processes may in-
teract with other environmental factors associated with
the onset of psychosis (e.g., stress, substance use),
which together have neuroprogressive sequelae that
may be neurodegenerative (Pantelis et al., 2005; Rapo-
port et al., 2005). Abnormal brain structure may be
detectible via MRI prior to the onset of psychotic symp-
toms (Lymer et al., 2006).

Studies of executive function and memory using fMRI
have reported abnormalities of the DLPFC, medial tem-
poral lobe, hippocampus, parahippocampal gyrus, an-
terior cingulate, medial frontal and posterior parietal
cortex, striatum, thalamus, and cerebellum (Niznikie-
wicz et al., 2003). Recent fMRI studies have focused
on the integration of genetic and neuroimaging data
(for review see Turner et al., 2006). The fMRI studies sug-
gest that for any given task that is performed poorly by
individuals with schizophrenia, there is a network of
affected brain regions related to the abnormal function,
rather than a single abnormal brain region, raising the
issue of the state of the interconnections between
regions.

DTI, a technique based on the direction of water diffu-
sion, can probe white matter abnormalities in the brain.
Early studies with the technique, which is still under development, have raised the possibility of white matter disorganization in brain regions such as prefrontal and temporal white matter, corpus callosum, and uncinate fasciculus (Kanaan et al., 2005; Kubicki et al., 2005). More systematic and detailed confirmatory studies are now necessary. A potentially powerful approach may be to combine fMRI and DTI to probe potential brain circuit abnormalities in schizophrenia.

Neuropathology
Neuropathological investigations of schizophrenia (Arnold et al., 1998) have not found any evidence of the usual features of neurodegenerative diseases, such as inclusion bodies, dystrophic neuritis, or reactive gliosis. There is intriguing, though not always consistent, evidence of subtle cytoarchitectural anomalies in entorhinal gray matter (Arnold et al., 1997) and in other corticocortical, and an abnormally high frequency of aberrant neurons in the white matter underlying prefrontal cortex (e.g., Akbarian et al., 1996), temporal, and parahippocampal regions (Arnold et al., 2005). While these findings remain open to various interpretations (Arnold et al., 2005), together they provide suggestive evidence for subtle abnormalities in neurodevelopment in schizophrenia, such as disordered cortical neuronal migration, consistent with the observation of subtle behavioral, neurological, and morphologic abnormalities.

Another line of evidence suggestive of neurodevelopment abnormality derives from findings of a reduction in the volume of cortical neuropil without comparable neuronal loss (Selemon et al., 1995; Selemon and Goldman-Rakic, 1999). Many (though not all) ultrastructural, immunohistochemical, and other quantitative neuropathological studies suggest quantitative and qualitative deficits in neuronal processes and synaptic connectivity in schizophrenia (Honer et al., 2000). A summary of neuronal connections implicated in the pathology of the neuropil in schizophrenia is shown in Figure 2.

Gene expression array studies have compared the expression profiles, in a number of different brain regions, of schizophrenias and controls (Katsel et al., 2005). These studies have yielded inconsistent results and still need to overcome the difficulties inherent in the usage of postmortem brain tissue. Genes related to GABA neurotransmission, synaptic transmission, and metabolism have been implicated, though the significance remains uncertain. Several studies have identified abnormal expression of genes related to myelination, suggesting the possibility of glial and white matter abnormalities, which could be fundamental to the disease, given the imaging indications of white matter abnormalities noted above.

Pharmacology
Treatment for schizophrenia remains far from optimal. While psychosocial programs and various forms of reality-based therapy are helpful, the mainstays of treatment are medications tautologically termed “antipsychotics.” The antipsychotics, first introduced over 50 years ago with the serendipitous discovery that chlorpromazine was effective in reducing the “positive” symptoms of schizophrenia, all have as their primary mechanism of action blockade of dopamine D2 receptors (Snyder, 2006). This “first” generation of antipsychotics included chlorpromazine, haloperidol, and perphenazine, and, while clearly more effective than placebo, they had a propensity to cause acute and chronic neurologic symptoms, including tremor, rigidity, dystonia, and dyskinesia.

More recently, a “second” generation of antipsychotics, such as clozapine and olanzapine, have been developed that have reduced risk for these acute and chronic neurologic side effects, possibly because of their additional blockade of serotonin 5HT2A receptors. However, it is now apparent that these newer antipsychotics confer a much greater risk for obesity, hyperlipidemia, and type II diabetes. Furthermore, recent head-to-head comparisons between the older, off-patent perphenazine and the newer atypical antipsychotics did not disclose major differences in efficacy or tolerability by patients with schizophrenia (CATIE, 2005).

While the antipsychotics generally reduce positive symptoms, poor compliance and the lack of impact on negative and cognitive symptoms mean that most individuals with schizophrenia remain substantially disabled and unemployed, and require supervised housing arrangements for the rest of their lives. The one exception appears to be clozapine, which is significantly more effective, causes improvement in a subgroup of patients unresponsive to other antipsychotics, and can reduce negative symptoms (McEvoy et al., 2006).

Clinical trials (Coyle, 2006; Heresco-Levy et al., 2002; Lane et al., 2005; Tsai and Gleeson, 2005) with agents which modulate NMDA receptors, including glycine, D-serine, D-cycloserine, sarcosine, or D-alanine, have suggested improvement in negative and cognitive symptoms when these agents are added to either typical or atypical antipsychotics. However, the doses and
agents have not been consistent among the different trials, and larger, more definitive trials may be indicated. Other agents under investigation to enhance NMDA receptor function indirectly, thereby treating the negative and cognitive symptoms unresponsive to antipsychotics, include AMPA kinases, which prolong AMPA receptor open time, positive modulators of the metabotropic mGluR5 receptors, and mGlu2/3 receptor antagonists (Moghaddam, 2003).

Pathophysiology

Hypotheses regarding pathophysiology of schizophrenia originated from pharmacology (Snyder, 2006). The “dopamine hypothesis” derived, in part, from the identification of D2 receptor blockade as the mechanism for the action of antipsychotics, and was supported by the observation that stimulants acting via dopamine, such as amphetamines, can cause psychosis in normal individuals and can exacerbate psychosis in individuals with schizophrenia. Pharmacological and physiological studies indicate that dopamine modulates cognitive function in the prefrontal cortex, a finding of potential relevance to schizophrenia.

Evidence for a role of glutamate in schizophrenia also originated from pharmacology (Coyle, 2006). NMDA receptor antagonists, such as ketamine and phenycyclidine (PCP), can cause psychotic and cognitive abnormalities reminiscent of schizophrenia. In addition, subjects with schizophrenia appear to be especially sensitive to the psychotomimetic effects of these drugs. The extent to which these effects recapitulate schizophrenic pathophysiology remains uncertain. As noted above, treatment of schizophrenia with D-Serine, glycine, and sarcosine, which modulate NMDA receptors, has therapeutic benefit, particularly with regard to negative symptoms. Thus, hypofunction of the NMDA receptor, possibly on critical GABA interneurons, may contribute to the pathophysiology of schizophrenia (Coyle, 2006).

The potential role for GABA in the pathogenesis of schizophrenia derives mostly from neuropathologic studies (Lewis et al., 2005). A particular subtype of GABA interneurons, chandelier neurons, have decreased immunostaining for the GABA transporter (GAT), possibly related to reduced BDNF signaling or NMDA receptor hypofunction. Consistent with the inferred reduced GABAergic neurotransmission, ligand binding and immunocytochemical studies have revealed upregulation of the postsynaptic GABA-A receptors in these sectors. The extent to which these changes represent primary pathogenesis has yet to be determined.

Mouse Models of Pathogenesis

Functional hypotheses of schizophrenia can now be addressed using mouse models, aided by the recognition that observation of some aspects of the schizophrenia phenotype and endophenotype do not require the self-reports of affected individuals (Chen et al., 2006). Behaviors that have been used as outcome measures in mice, with varying resemblance to the clinical features of schizophrenia, include social interaction, prepulse inhibition, aggression, and locomotor activity. Mouse models associated with selected candidate genes are discussed below.

For instance, knock out of the dopamine transporter or overexpression of D2 dopamine receptors causes behavioral abnormalities, and overexpression of the D2 receptor in the forebrain causes cognitive changes reminiscent of those observed in schizophrenia (Kellendonk et al., 2006). Similarly, mice with alterations in molecules downstream of dopamine signaling such as DARPP-32 (dopamine and cyclic adenosine monophosphate-regulated phosphoproteins of 32 kDa) have behavioral phenotypes that may be relevant to schizophrenia. Targeted deletion of the calcineurin gene yields abnormal locomotion, decreased social interactions, and altered cognition, consistent with evidence of decreased cortical calcineurin. Caron’s group developed a mutant mouse line that expressed only 5% of normal levels of the NMDA receptor subunit NR1 (Mohn et al., 1999). These mice exhibited hyperactivity that responded to the typical antipsychotic haloperidol, but they also exhibited impaired social behaviors and mating that were partially reversed by the atypical antipsychotic clozapine.

These studies of candidate genes, based on functional hypotheses, provide interesting behavioral and pathophysiologic information, which is in many cases relevant to understanding the pharmacology of schizophrenia treatment, and in some cases of potential relevance to disease pathogenesis. However, we believe that the development of mouse models based on etiologic risk factors, such as the genes discussed below, will ultimately provide the most powerful tools for understanding the neurobiology of schizophrenia.

Genetic Etiologies: Genes Identified in Linkage or Association Studies

Linkage and association studies have now implicated several loci in the genome that appear likely to harbor genes conferring risk for schizophrenia (Figure 3, Table 1). Candidate genes identified by a genetic approach have the advantage over candidate genes chosen based on pharmacotherapies or pathological studies in that they are of necessity involved in the disease process, at least for the populations in which the genetic results were obtained. It should be kept in mind that schizophrenia genetics are complex, with multiple genes of modest effect interacting to produce the phenotype. Relative risk at the loci identified so far range between 1.5 to 2.0, indicating modest effect sizes. Simple mutations with Mendelian inheritance and complete penetrance have not yet been found using standard linkage and association methods, though study of chromosomal translocations provides a useful alternative.

Neuregulin 1

Neuregulin 1 was identified as a candidate gene via fine-mapping of a locus on chromosome 8p linked to schizophrenia (Harrison and Law, 2006; Stefansson et al., 2002). A number of studies have found association with schizophrenia within the neuregulin 1 region. The neuregulin 1 gene is very complex, with at least 25 exons spread over almost a megabase, with extensive alternative promoter usage and alternative splicing, resulting in multiple possible protein products. A region in the 5’ end of the gene appears to most consistently associate with disease. Unfortunately, no functional polymorphisms have been identified. Most neuregulin 1 isoforms
are transmembrane proteins, which can undergo pro-
teolytic cleavage to release extracellular fragments,
intracellular fragments, transmembrane receptors, or
membrane-bound signaling proteins.

Neuregulin 1 signaling, via ErbB receptors and regu-
lation of both NMDA receptors and postsynaptic density
95 (PSD-95), has been implicated in neuronal differenti-
ation and migration. In addition, a C-terminal fragment

![Figure 3. Locations of Linkage Findings and Genes](image)

Chromosomal regions with significant linkage to schizophrenia are indicated by vertical blue lines. Chromosomal deletions are shown with vertical red lines. The red arrows refer to the location of chromosomal abnormalities associated with schizophrenia. The yellow arrows and circles show the locations of the genes identified by linkage and association. The red arrows circles indicate genes identified via translocations. Adapted from Owen et al. (2005).

<table>
<thead>
<tr>
<th>Table 1. Candidate Schizophrenia Susceptibility Genes and the Strength of Evidence in Four Domains</th>
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<tr>
<td>Strength of evidence (0 to 5+)</td>
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<td><strong>COMT</strong></td>
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<td><strong>DISC1</strong></td>
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<td><strong>PPP3CC</strong></td>
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<td><strong>CHRNA7</strong></td>
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<td><strong>PRODH2</strong></td>
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<td><strong>AKT1</strong></td>
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<td><strong>GAD1</strong></td>
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<td><strong>ERBB4</strong></td>
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<td><strong>FEZ1</strong></td>
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<td><strong>MUTED</strong></td>
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<td><strong>MRDS1 (OFCC1)</strong></td>
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<td><strong>NPAS3</strong></td>
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<td><strong>GRIK4</strong></td>
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Adapted from Straub and Weinberger (2006).
of Neuregulin 1 can translocate to the nucleus and interact with transcription factors to enhance expression of genes, including PSD-95. The functional role of neuregulin 1 in schizophrenia is still uncertain, particularly since many different alleles and haplotypes have been implicated. However, recent biochemical experiments in human postmortem tissue suggest that neuregulin 1 signaling may be enhanced in schizophrenia, leading to suppression of NMDA receptor function (Hahn et al., 2006). This would be consistent with the glutamate hypofunction hypothesis of schizophrenia (see above). No consistent changes in the expression level of Neuregulin 1 itself have been detected in schizophrenia, and, in the absence of mutations changing protein sequence, it is unclear how this increased activation would come about. One possibility is the existence of polymorphisms that lead to alternative splice variants that encode protein products with enhanced function.

Mouse models with heterozygous deletions of the transmembrane domain of neuregulin 1 have altered activity and prepulse inhibition (Chen et al., 2006). However, the relation of the deletion in this model to changes in human schizophrenia is unclear. No coding mutations have been detected in schizophrenia. The region implicated in schizophrenia by haplotype analysis is upstream from the transmembrane domain, and includes the initial exon of the type II isoform (Falls, 2003), and mouse models with alterations in this region have not yet been described.

**Dysbindin**

*Dysbindin* (dystrobrevin binding protein I) was identified as a gene associated with schizophrenia through linkage to chromosome 6p (Straub et al., 2002). The association between this locus and schizophrenia has been replicated in several subsequent studies. Dysbindin co-localizes with dystrobrevin in both muscle and brain. It is widely distributed in brain, and has been detected both pre- and postsynaptically, including in synaptic terminals in the hippocampus (Benson et al., 2001). The function of dysbindin in brain is not well understood. It has been reported to influence glutamate neurotransmission (Numakawa et al., 2004). Mutations in *dysbindin* also cause Hermansky-Pudlak syndrome type 7 (Li et al., 2003), a complex genetic disorder related to lysosome biogenesis, which is not known to have a psychiatric phenotype. A deletion within the homologous gene in mice accounts for the phenotype known as “Sandy,” with albinism and bleeding disorders.

While the association of *dysbindin* with schizophrenia has been fairly well replicated, no protein coding mutations contributing to the risk for schizophrenia have been identified. Furthermore, many different alleles and haplotypes have been implicated in different studies (e.g., Burdick et al., 2006; Gomick et al., 2005). Reduced levels of expression of *dysbindin* message or protein have been found in schizophrenic brains (Bray et al., 2005), raising the possibility that polymorphisms in *dysbindin* associated with schizophrenia may modulate dysbindin expression level. In addition, knockdown of endogenous dysbindin with siRNA resulted in reduction of glutamate levels in neurons in culture, suggesting a possible synaptic consequence for reductions in dysbindin levels (Numakawa et al., 2004; Talbot et al., 2004) and connecting dysbindin with the glutamate hypofunction hypothesis of schizophrenia.

Two studies have independently described an association between *dysbindin* risk haplotypes and high levels of negative symptoms in schizophrenia (Fanous et al., 2005; DeRosse et al., 2006), supporting the importance of careful delineation of different domains of schizophrenia symptoms. This finding is consistent with other evidence that *dysbindin* haplotypes may influence prefrontal brain function (Fallgatter et al., 2006). Thus, further study of *dysbindin* genotypes in relationship to specific subtypes of schizophrenia and to cognitive endophenotypes appears warranted, as does detailed investigation of the role of dysbindin in glutamate neurotransmission and other neuronal functions. Further mouse models of *dysbindin* alterations would be very valuable.

### D Amino Acid Oxidase Activator

The chromosome 13 locus has strong linkage regions to schizophrenia. Among other genes, this locus contains G72, now called *D amino acid oxidase activator* (*DAOA*). Several individual replication studies and a meta-analysis have supported the association of *DAOA* with schizophrenia, though as with other loci, the associated alleles and haplotypes are not identical across studies, and some variants are located outside of the gene (Detera-Wadleigh and McMahon, 2006). Functionally, DAOA activates D amino acid oxidase (DAO). DAO oxidizes D-Serine, which is a coagonist at NMDA glutamate receptors. Thus, there is some biologic plausibility for *DAOA* as a candidate gene, based on the glutamate hypothesis. *DAOA* does not have a homolog in mice, so no knockout model has been made. Further explorations of this system may be of considerable interest, especially given the potential efficacy of D-Serine in therapeutic trials and reports of reduced D-Serine in blood and CSF in individuals with schizophrenia.

### COMT and Chromosome 22 Region

Another linkage region is on chromosome 22 (Harrison and Weinberger, 2005; Owen et al., 2005). It has been supported in many, though not all, linkage and association studies. In addition, strong genetic association between schizophrenia and the chromosomal microdeletion syndrome VCFS (Mendelian Inheritance in Man, MIM 192430), which is caused by deletion of approximately 1.5 to 3 Mb in chromosome 22q11, supplies strong evidence for a genetic contribution to schizophrenia from this region. Approximately 20% to 30% of patients with VCFS have schizophrenia or other major psychiatric disorders with psychosis (Murphy et al., 1999). Furthermore, patients with schizophrenia have increased frequency of the microdeletion compared with the general population (Karayiorgou et al., 1995). VCFS includes facial dysmorphism and other features, and presumably is caused by loss of one copy of several or many genes in this region. The VCSF region includes at least 27 genes. The Tbx1 gene may account for many of the physical features of VCFS (Li et al., 2003; Long et al., 2006). It is expressed in microvasculature in brain. Inactivating mutations in Tbx1 have been found in one small family with VCFS or Asberger’s syndrome (Li et al., 2003), but the relation of this gene to schizophrenia is still incompletely explored.
The gene on chromosome 22q11 that has received the most attention is catechol-O-methyltransferase (COMT). The protein product is an enzyme that participates in the clearance of dopamine from synapses, and thus could be involved in regulation of neurotransmission related to schizophrenia (Craddock et al., 2006; Tunbridge et al., 2006). A functional polymorphism, involving the presence of either valine or methione at codon 108 (in the soluble isoform of COMT, equivalent to codon 158 in the membrane-bound isoform of COMT) alters enzyme activity. The methione allele is less stable and thus has lower activity, suggesting the hypothesis that individuals with two copies of the methione allele, or with a deletion of one copy of COMT, would be expected to have higher dopamine levels in critical central synapses, perhaps especially in the prefrontal cortex.

In a seminal study combining genetics of the COMT valine/methione polymorphism with imaging methods, the valine allele, which would have lower synaptic dopamine, was reported to confer risk for schizophrenia via variation in cognitive function in contradiction to the dopamine hypothesis, which proposes increased synaptic dopamine as the risk mechanism (Egan et al., 2001). The relationship appears to be complicated (Craddock et al., 2006; Tunbridge et al., 2006), and the association between COMT alleles and schizophrenia appears to be less striking than the association between COMT and cognitive function. For instance, a relationship between the valine/methione polymorphism and longitudinal cognitive decline in patients with the 22q11.2 deletion syndrome has recently been reported, though not yet replicated (Gothelf et al., 2005). Variation at the COMT locus may provide the best studied example of the relationship between variation at a genetic locus and an endophenotype closely related to schizophrenia.

Other genes in the deletion syndrome region may also contribute to the risk for schizophrenia. For instance, genetic variation of the proline dehydrogenase (PRODH) gene influences the availability of glutamate, and mutant mice with a PRODH loss-of-function exhibit some behavioral abnormalities. A recent report has postulated an interaction between COMT and PRODH (Paterlini et al., 2005). However, association and follow-up linkage studies have not been strongly positive. ZDHHC8, also in the 22q deletion region, encodes a zinc finger domain protein. However, strong evidence in favor of this gene has not yet emerged (Harrison and Weinberger, 2005; Owen et al., 2005).

**Other Candidate Genes Based on Linkage Studies**

Other candidate genes are listed in Table 1 and described in recent reviews (e.g. Harrison and Weinberger, 2005; Owen et al., 2005; Straub and Weinberger, 2006).

**Genes Disrupted by Chromosomal Translocations**

Genes interrupted by chromosomal translocations so far appear to be very rare causes of schizophrenia. However, the advantage is that since translocations produce a definable genetic lesion, it may be possible to determine the effects of the mutation on the function of the gene product.

The Neuronal PAS Domain Protein 3 (NPAS3) gene codes for a transcription factor containing a basic helix-loop-helix (HLH) PAS domain involved in transcriptional regulation. NPAS3 was found to be disrupted by chromosomal translocation in two related individuals with schizophrenia (Pickard et al., 2005). Since HLH domain-containing proteins function as dimers, and because the translocation could produce a truncated protein without the transcriptional activation domains, the truncation might act via a dominant-negative mechanism. Since the family is so small, it is premature to conclude that there is a relationship between this gene and schizophrenia. However, deletions of NPAS transcription factors in mice cause behavioral phenotypes and altered hippocampal neurogenesis (Pieper et al., 2005), providing additional support for a role of NPAS in schizophrenia.

A translocation through GRIK4, which codes for one of the glutamate kainate receptors, has also been detected in an individual with schizophrenia (Pickard et al., 2006). Subsequent case control studies suggested an association of a haplotype within this gene to schizophrenia. A translocation through PDE4B, as discussed below, has also been detected in a small family with schizophrenia.

**DISC1: Interrupted by a Chromosome 1,11 Translocation**

DISC1, in our view, is emerging as the best supported candidate gene for schizophrenia (Hennah et al., 2006; Ishizuka et al., 2006; Porteous and Millar, 2006), with a great potential for future research. DISC1 was identified via a balanced (1:11) chromosomal translocation, segregating with schizophrenia, bipolar disorder, and other major mental illness in a large pedigree in Scotland, with LOD scores of 7 using a broad phenotype. The translocation is between exons 8 and 9 of the DISC1 gene on chromosome 1. No genes have been found at the chromosome 11 site.

The translocation has not been found in any other families. Another small family (Sachs et al., 2005), identified via a proband with schizophrenia, has a four-base deletion resulting in a frame shift and predicted C-terminal truncation of the DISC1 protein. However, the family is too small to clearly demonstrate segregation with disease, and the deletion has also been found in two presumably unaffected blood donors (Green et al., 2006).

A locus on chromosome 1 within the DISC1 gene was linked to schizophrenia in a Finnish population (Ekelund et al., 2001), and the DISC1 locus has emerged as a potential risk factor for both schizophrenia and affective disorder in different populations (Craddock et al., 2005; Hennah et al., 2006; Millar et al., 2003; Thomson et al., 2005; Porteous and Millar, 2006).

Study of the original Scottish phenotype suggested two distinctive features of the clinical phenotype. First, affected individuals have either schizophrenia or affective disorder. Consistent with this, recent linkage studies have implicated the DISC1 locus, especially for schizoaffective disorder (Hamshere et al., 2005). Second, reduced P300 amplitude and latency, an endophenotype, was associated with the translocation in both affected and unaffected translocation carriers (Blackwood et al., 2001). More recent imaging and neuropsychological studies have suggested that DISC1 haplotypes, including a putative functional polymorphism (S704C), are associated with altered hippocampal function, altered...
fMRI signals, and altered working memory and cognition in individuals with and without schizophrenia or affective disorder (Figure 4), consistent with an influence of DISC1 on cognitive endophenotypes (Callicott et al., 2005; Cannon et al., 2005; Porteous et al., 2006).

Variation at the DISC1 locus, via a deletion in exon 6, may also contribute to phenotypes in mouse substrain 129. The effects of this have not been conclusively demonstrated, but it appears to abrogate expression. On transfer of the DISC1 deletion allele to the BL/6 background, the deletion mice, but not littermate controls, have selective impairment in working memory (Koike et al., 2006).

The molecular mechanism of the DISC1 translocation mutation is uncertain. Most of the evidence points to loss-of-function effects, but the exact mechanism is controversial. Loss of function could result from loss of expression, and thus haploinsufficiency. Alternatively, it is conceivable that a truncated mutant protein is produced. No mutant protein expression was detected in lymphoblasts from the patients with the Scottish translocation (Millar et al., 2005), though techniques might not have been sensitive enough to identify low levels of expression, and expression of transcripts from the mutant allele could be detected. Biochemical studies have indicated that DISC1 protein has a self interaction domain and likely functions as a dimer. DISC1 protein with a C-terminal truncation, corresponding to the protein that would be produced from the translocation allele, disrupts the normal function and cellular localization of the full-length protein (Kamiya et al., 2005), suggesting the possibility of a dominant-negative mechanism. Future mouse model studies may resolve some of these issues in vivo. Whether via haploinsufficiency or dominant-negative interactions, loss-of-function mechanisms imply that understanding the normal function of DISC1 will be critical for understanding DISC1-related disease.

DISC1 appears to have roles in both brain development and adult neuronal functioning. Developmental roles include regulation of neuronal migration, neurite outgrowth, and neuronal maturation. Roles in the adult appear to include modulation of cytoskeletal function, synaptic transmission, and plasticity. The expression of DISC1 is increased during neuronal development, with peaks at E13.5 during late fetal development and at P35 in early postnatal periods (Schurov et al., 2004). Expression continues into adulthood, with the highest expression in hippocampus, olfactory bulb, lateral septum, cerebral cortex, and hypothalamus and other brainstem regions (Austin et al., 2003). DISC1 protein can be detected in many regions within cortical neurons, including presynaptic and postsynaptic locations (Kirkpatrick et al., 2001).

Studies of the protein interaction partners of DISC1, and the cell biology of these interactions, have greatly illuminated DISC1 functions and provided strong support for roles of DISC1 in brain development and adult neuronal function. Table 2 shows some of the protein interaction partners of DISC1 and their potential cellular roles. As indicated in Figure 5, the molecular and cellular interactions of DISC1 are critical for normal neuronal development and in the adult are implicated in normal neuronal signal transduction and plasticity.

DISC1 interacts with several proteins which themselves are implicated in neuropsychiatric diseases. For

<table>
<thead>
<tr>
<th>DISC1 Interactor</th>
<th>Interactor Function</th>
<th>DISC1 Binding Site</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>NudEL</td>
<td>Neuronal migration</td>
<td>727–854</td>
<td>Brandon et al., 2004; Morris et al., 2003; Ozeki et al., 2003</td>
</tr>
<tr>
<td>Lis1</td>
<td>Neuronal migration</td>
<td>727–854</td>
<td>Brandon et al., 2004</td>
</tr>
<tr>
<td>PDE4B</td>
<td>cAMP hydrolysis</td>
<td>219–283</td>
<td>Millar et al., 2005</td>
</tr>
<tr>
<td>Citron</td>
<td>Synaptic function</td>
<td>347–600</td>
<td>Ozeki et al., 2003</td>
</tr>
<tr>
<td>a-tubulin</td>
<td>Cytoskeleton</td>
<td>181–157</td>
<td>Branden et al., 2004</td>
</tr>
<tr>
<td>ATF4/5</td>
<td>Transcription factors</td>
<td>598–854</td>
<td>Morris et al., 2003</td>
</tr>
<tr>
<td>DISC1</td>
<td></td>
<td>403–504</td>
<td>Kamiya et al., 2005</td>
</tr>
<tr>
<td>FEZ1</td>
<td>Neurite extension</td>
<td>446–633</td>
<td>Miyoshi et al., 2004</td>
</tr>
<tr>
<td>Kendrin</td>
<td>Centrosome, microtubule</td>
<td>446–633</td>
<td>Miyoshi et al., 2004</td>
</tr>
<tr>
<td>eIF3</td>
<td>Translation initiation factor</td>
<td>2–231</td>
<td>Ogawa et al., 2005</td>
</tr>
<tr>
<td>MAP1A</td>
<td>Microtubule associating</td>
<td>1–292</td>
<td>Morris et al., 2003</td>
</tr>
<tr>
<td>MIPT3</td>
<td>Microtubule associating</td>
<td>293–696</td>
<td>Morris et al., 2003</td>
</tr>
</tbody>
</table>

Adapted from Porteous et al. (2006).
example, DISC1 interacts with NudEL. Its close homolog NudE may be genetically related to schizophrenia (Hennah et al., 2006). NudEL is part of a protein complex with Lis1, downstream of Reelin signaling (Brandon et al., 2004). As noted above, mutations of Lis1 cause lissencephaly, and the presence of DISC1 in the same complex as Lis1 is consistent with the idea that schizophrenia, as a relatively mild disorder of cortical development, is pathophysiology related to more severe disorders of cortical development. Reelin mutations also cause lissencephaly. The interaction between DISC1 and both NudEL and Lis1 would be disrupted by truncated protein expressed from the putative message produced by the chromosomal translocation. Finally, DISC1 interacts with PDE4B, which is itself interrupted by balanced chromosomal translocation in two individuals with schizophrenia or chronic psychiatric illness (Millar et al., 2005).

These interactions are relevant for the cellular functions of DISC1. DISC1 is part of a protein complex including, in addition to Lis1 and NudEL, dynein and dynactin, which is critical for neuronal migration (Hatten, 2002; Olson and Walsh, 2002; Tsai and Gleeson, 2005). Neuronal migration in the cerebral cortex involves movement along radial glial toward Cajal–Retzius cells and subpial granular layer cells, which secrete Reelin. Migration is driven by nucleokinesis, for which microtubule-based transport is critical. The DISC1 protein complex appears to have several important functions in this process. It appears to be critical for assembly of the centrosome and the organization of the cellular microtubule network. The nucleus is moved by microtubule-based transport toward the centrosome, and neurites extend distally from the centrosome, which is also based in part on microtubule-based transport. Cell biological studies in the Sawa laboratory indicate that DISC1 is important for maintaining a protein complex at the centrosome that is critical for these functions (Kamiya et al., 2005).

DISC1 also modulates neurite outgrowth (Miyoshi et al., 2003) (Ozeki et al., 2003). Either loss of normal DISC1 function or expression of the mutant allele caused abnormal neurite outgrowth in PC12 cells and cortical neurons. Furthermore, elegant in vivo studies in the Nakajima laboratory using in utero electroporation found delayed migration of cortical neurons expressing DISC1 siRNA or mutant truncated DISC1. In the adult cortex, affected neurons continued to have subtle disturbances of neurite orientation (Kamiya et al., 2005).

In addition to microtubules, the actin cytoskeleton is important for neuronal migration and neurite outgrowth. DISC1 associates with FEZ1, an actin binding protein that may have a critical role in anchoring microtubules.
near the cell membrane. Neurite outgrowth also appears to involve the DISC1/FEZ1 complex (Miyoshi et al., 2003). DISC1 also interacts with several transcription factors, including ATF4 and ATF5, suggesting that DISC1 mutations could potentially alter gene transcription (Morris et al., 2003). DISC1 also binds to Citron, a postsynaptic protein that interacts with PSD-95, suggesting a role for DISC1 in the regulation of synaptic function and synaptic plasticity.

Finally, as noted above, DISC1 has recently been shown to interact with PDE4B, with functional consequences for cAMP signaling. Release of PDE4B by DISC1 activates PDE4B, causing conversion of cAMP to adenosine monophosphate. cAMP is critical for regulation of protein kinase A, which in turn has many functions in neuronal signaling and plasticity in the cell. Furthermore, PDE4B is a target of the antidepressant rolipram, consistent with the postulated involvement of DISC1 in affective disorder as well as schizophrenia.

**Etiology: Environmental Interactions**

Environmental and genetic etiologies are both important in psychiatry (Caspi and Moffitt, 2006) and are believed to interact in most cases of schizophrenia. Recent immunologic, epidemiologic, and neuropsychiatric studies suggest infectious etiologies of several major neuropsychiatric diseases (Yolken et al., 2000). Infections that have been associated with schizophrenia include rubella, influenza, Herpes Simplex Virus-1 and -2, cytomegalovirus, poliovirus, and Toxoplasma gondii (Brown and Sussler, 2002). Patterson (2002) has developed evidence that it is not the virus itself that adversely affects fetal brain development, but rather the cytokine response mounted by the infected mother. Infections during pregnancy can affect brain development by releasing stress hormones, producing hypoxia, hyperthermia, or malnutrition, or by triggering proinflammatory cytokine responses of the mother, the placenta, or the fetus (Gilmore and Jarskog, 1997; Verdoux, 2004). The effects of infection in the perinatal and postnatal period can differ. There can be substantial individual difference in the response to infectious agents. Among other environmental insults implicated as risk factors for schizophrenia are obstetric complications, including premature birth, low birth weight, pre-eclampsia, rhesus incompatibility, resuscitation at birth, emergency Cesarean delivery, and prenatal nutritional deficiency (Cannon et al., 2002; Kyle and Pichard, 2006; St Clair et al., 2005).

**Conclusions and Possibilities for Future Research**

In conclusion, we now believe that the molecular genetics of schizophrenia are sufficiently advanced such that etiology-based studies of the neurobiollogy of schizophrenia are both justified and feasible. The field is still in its infancy, and we must struggle to integrate our rudimentary knowledge of schizophrenia genetics with our scarcely better developed understanding of normal human brain function. Additional genetic studies are indispensable in this effort, and will now be facilitated by genome-wide methods for study of association and methods to systematically investigate variations in genomic copy number. Epigenetic modification, such as methylation, may also prove relevant (Abdolmaleky et al., 2005). Mouse models will make it possible to test pathogenic hypotheses.

How to address the nature and contribution of environmental factors is more uncertain. One possibility may be to introduce proposed environmental factors, such as viral infections, to mouse models of identified mutations in genes such as DISC1 or NPAS3.

The mouse models generated to date have been based on the study of Mendelian disorders (Chen et al., 2006). The more subtle etiologies of schizophrenia and other psychiatric disorders may make more complex genetic models important. For instance, it may be important to generate models with splicing alterations in neuregulin 1 or with amino acid polymorphisms in COMT or DISC1. In addition, it may be important to use inducible or other conditional systems in order to mimic the effect of activation of the genetic lesion in particular tissues at particular times.

In addition to mouse models, genetic models in other organisms may be very useful. Unlike in neurodegenerative diseases, it may be difficult to use Drosophila or other invertebrates as models for the complexities of human psychiatric disorders. For understanding alterations of cortical development, zebrafish, in which development can be directly visualized, may prove suitable. Other species with more complex social behaviors and more complex cognition may ultimately be necessary. Perhaps genetically modified primates may become an important source of models. However, human patients must remain the gold standard. Studies of genetics and clinical and imaging phenotypes can increasingly be integrated. Future imaging studies may be able to combine fMRI with DTI to trace functionally identified circuits.

We propose that study of DISC1 may offer unique opportunities for inroads into understanding the biology of schizophrenia. DISC1 appears to act as a scaffold for protein interactions, and some of these interacting proteins have altered expression in schizophrenia (Lipska et al., 2006). These interactors will be helpful for understanding pathogenesis, and can themselves serve as potential candidate genes to test for mutations. Thus, a neurogenetic approach based on candidate genes (Ross and Pearson, 1996) may now become possible. The DISC1 interacting protein L1st is related to lissencephaly, highlighting the idea that schizophrenia, as a subtle disorder of cerebral cortical development, is related to more severe disorders of cerebral cortical development.

Study of the different genetic etiologies of schizophrenia will also improve understanding of the schizophrenia phenotype, and also understanding of affective disorder and potentially other related major psychiatric illnesses, just as study of the different genes causing lissencephaly has allowed a more careful classification of the phenotypes of lissencephaly (Kato and Dobyns, 2003). Some of the genes, such as dysbindin, appear to be related more specifically to schizophrenia, perhaps especially deficit schizophrenia, while others such as DISC1 and neuregulin 1 can relate to both schizophrenia and affective disorder.

The genes associated with schizophrenia may have a spectrum of different pathogenic effects, altering neuronal development, neuronal plasticity, and signal...
transduction. While undoubtedly a great oversimplification, it may be of heuristic value to postulate that variations in particular genes can affect particular neurobiological processes (Figure 6), in turn causing specific phenotypes. For instance, effects on neurodevelopment may be more closely associated with schizophrenia, while effects on signal transduction may be more likely to cause affective disorder. We suggest that DISC1 may serve as a kind of Rosetta Stone for schizophrenia research, helping to connect disparate domains. Testing these broader hypotheses will require integration of research in biochemistry and cell biology, mouse genetics, neuroimaging, and human genotype-phenotype correlations. These studies may allow us to reconceptualize our definitions of the psychiatric disorders, including schizophrenia, based on a better understanding of etiology and pathogenesis.

Ultimately, neurobiological study of schizophrenia, a remarkable disorder of brain function, may help illuminate the nature of normal thought, perception, and emotion. Thus, understanding of this most human disorder may help us better understand human nature itself.

Acknowledgments

NARSAD, Stanley Medical research institute, NIMH, NINDS, and Johns Hopkins Psychiatry provided support. We are indebted to many previous excellent reviews for information and perspective, including those by Cannon, Harrison, Lewis and Lieberman, Rapoport, and many previous excellent reviews for information and perspective, in addition to those by Cannon, Harrison, Lewis and Lieberman, Rapoport, and many previous excellent reviews for information and perspective. We thank Mike Owen for providing a copy of Figure 1 from Owen et al. (2005), which we have modified for our Figure 3. Some concepts from Figure 1 of Harrison and Weinberger (2005) were adapted for our Figure 6. We thank the anonymous peer reviewers for helpful comments and suggestions. We thank David Porteous and J. Kirsty Millar, Mike Owen, Akira Sawa, Bob Yolken, and Chris Walsh for comments.

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Schizophrenia: A Review

STEPHEN H. SCHULTZ, MD, STEPHEN W. NORTH, MD, MPH, and CLEVELAND G. SHIELDS, PhD, University of Rochester School of Medicine and Dentistry, Rochester, New York

Schizophrenia is a debilitating mental illness that affects 1 percent of the population in all cultures. It affects equal numbers of men and women, but the onset is often later in women than in men. Schizophrenia is characterized by positive and negative symptoms. Positive symptoms include hallucinations, voices that converse with or about the patient, and delusions that are often paranoid. Negative symptoms include flattened affect, loss of a sense of pleasure, loss of will or drive, and social withdrawal. Both types of symptoms affect patients’ families; therefore, it is important for physicians to provide guidance to all persons affected by the disease. Psychosocial and family interventions can improve outcomes. Medications can control symptoms, but virtually all antipsychotics have neurologic or physical side effects (e.g., weight gain, hypercholesterolemia, diabetes). There is a 10 percent lifetime risk of suicide in patients with schizophrenia. (Am Fam Physician 2007;75:1821-9, 1830. Copyright © 2007 American Academy of Family Physicians.)

This article exemplifies the AAFP 2007 Annual Clinical Focus on management of chronic illness.

► Patient information: A handout on helping a family member with schizophrenia, written by the authors of this article, is provided on page 1830.
onset of definitive symptoms. The risk of false-positive screening results is high, and screening is not yet accurate enough to warrant the cost and harms associated with misdiagnosis.10,11

**Diagnosis**

Schizophrenia is characterized by positive and negative symptoms that can influence a patient’s thoughts, perceptions, speech, affect, and behaviors (Table 1). Positive symptoms include hallucinations, voices that converse with or about the patient, and delusions that are often paranoid. Negative symptoms include flattened affect, loss of a sense of pleasure, loss of will or drive, and social withdrawal.

Schizophrenia is also characterized by disorganized thought, which is manifested in speech and behavior. Disorganized speech may range from loose associations and moving quickly through multiple topics to speech that is so muddled that it resembles schizophasia (commonly referred to as “word salad”). Schizophasia is speech that is confused and repetitive, and that uses words that have no apparent meaning or relationship to one another. Disorganized behavior may lead to difficulties in performing daily living activities, such as preparing a meal or maintaining hygiene. It also can manifest as childlike silliness or outbursts of unpredictable agitation.1

No single sign or symptom is pathognomonic of schizophrenia. To make a definitive diagnosis, signs and symptoms must be present for a significant portion of one month (or a shorter period if successfully treated), and some must be present for at least six months. These symptoms also must be associated with marked social and occupational dysfunction.

There are five types of schizophrenia: paranoid, disorganized, catatonic, undifferentiated, and residual.1 Paranoid type is characterized by a preoccupation with one or more delusions or frequent auditory hallucinations; cognitive function and affect remain relatively well preserved.1 Disorganized type is characterized by disorganized speech and behavior, as well as flat or inappropriate affect.1 Catatonic type has at least two of the following features: immobility (as evidenced by stupor or catalepsy); excessive, purposeless motor activity; extreme negativism (e.g., resistance to all instructions, maintenance of rigid posture, mutism); or peculiarities of voluntary movement (e.g., posturing, prominent mannerisms, grimacing).1 A patient is said to have undifferentiated schizophrenia if none of the criteria for paranoid, disorganized, or catatonic types are met.1 Residual type is characterized by the continued presence of negative symptoms (e.g., flat affects, poverty of speech) and at least two attenuated positive symptoms (e.g., eccentric behavior, mildly disorganized speech, odd beliefs). A patient is diagnosed with residual type if he or she has no significant positive psychotic features.1

Of note, this classic typing of schizophrenia can be limiting because patients often are difficult to classify. For that reason, an alternative three-factor dimensional model is given. The three factors are psychotic, disorganized, and negative (deficit). The symptoms are categorized as absent, mild, moderate, or severe.1

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**Table 1. Family History and Schizophrenia**

<table>
<thead>
<tr>
<th>Family history</th>
<th>Approximate lifetime incidence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None (e.g., general population)</td>
<td>1</td>
</tr>
<tr>
<td>Third-degree relative (e.g., first cousin)</td>
<td>2</td>
</tr>
<tr>
<td>Second-degree relative (e.g., niece or nephew)</td>
<td>2 to 6</td>
</tr>
<tr>
<td>First-degree relative (e.g., parent, child, sibling)</td>
<td>6 to 17</td>
</tr>
<tr>
<td>Dizygotic twin</td>
<td>17</td>
</tr>
<tr>
<td>Monozygotic twin</td>
<td>50</td>
</tr>
</tbody>
</table>

*Information from reference 2.*
TYPICAL PRESENTATION

The onset of schizophrenia can be abrupt or insidious. Most patients undergo a prodromal phase marked by a slow and gradual development of symptoms, such as social withdrawal, loss of interest in school or work, deterioration in hygiene and grooming, unusual behavior, or outbursts of anger. Family members can find this behavior disturbing and difficult to interpret. They may assume that the person is just “going through a phase.” Eventually, the appearance of active-phase symptoms (e.g., psychosis) marks the disturbance as schizophrenia.1

DIFFERENTIAL DIAGNOSIS

Table 3 outlines common diagnoses that produce symptoms similar to schizophrenia. Because substance abuse can mimic many signs and symptoms of schizophrenia, diagnosis should not be made if the patient is actively using illicit drugs. Patients who present with psychotic features should receive a drug screening as part of their initial evaluation. Those with severe depression or bipolar disorder also may present with psychotic features; however, the diagnosis of a mood disorder always takes precedence over the diagnosis of schizophrenia.

Despite the stability of the diagnostic criteria for schizophrenia, diagnosis often changes over time. In a study of 936 inpatients over seven years, 21.9 percent of those who were initially diagnosed with schizophrenia had their diagnosis changed during subsequent hospitalizations, and 32.8 percent of those who were initially diagnosed with another illness were later diagnosed with schizophrenia. Most diagnostic changes from schizophrenia were to either bipolar disorder or organic disorders. Organic disorders, psychotic disorders, and major depression were the diagnoses most commonly changed to schizophrenia.12

Delirium can have features that are similar to the active symptoms of schizophrenia (e.g., hallucinations, delusions). The etiology of delirium is extensive. The crucial difference between schizophrenia and delirium is the timing; signs and symptoms of schizophrenia generally develop over weeks to months, whereas delirium usually has a much more rapid onset. Because many medical illnesses can cause delirium, the diagnosis of new-onset schizophrenia should be made cautiously in patients who have an existing serious medical illness.
There also are racial disparities in the diagnosis of schizophrenia. For example, black persons are more likely than other racial groups to have symptoms attributed to schizophrenia, and Hispanics are more likely to be diagnosed with major depression when presenting with psychotic symptoms.

A complete history chronicling the development of signs and symptoms is crucial when diagnosing schizophrenia. Because the patient may have altered perceptions, obtaining a comprehensive history from at least one family member or close friend is essential to provide another perspective of the disease course.

**Drug Treatment**

Effective pharmacologic treatment of schizophrenia has been available since the 1950s. In the early 1950s, the term “neuroleptic” was introduced to denote the effects of chlorpromazine (Thorazine; brand no longer available in the United States) and reserpine on laboratory animals. It was intended to distinguish their effects from those of sedatives and other central nervous system depressants. Although “neuroleptic” is still used synonymously with “antipsychotic,” the term now usually refers to first-generation antipsychotics that confer an increased risk of extrapyramidal side effects, such as dystonic reactions (e.g., fixed upper gaze, neck twisting, facial muscle spasms), parkinsonian symptoms (e.g., rigidity, bradykinesia, shuffling gait, tremor), and akathisia (e.g., inability to sit still, restlessness, tapping of feet). Tardive dyskinesia, which is a chronic disorder of the nervous system characterized by involuntary jerking movements (primarily of the face, tongue, and jaw), often is considered an extrapyramidal side effect. However, it is actually a separate and mechanistically different phenomenon.

The term “atypical antipsychotic” refers to newer antipsychotics that confer less risk of extrapyramidal side effects than traditional antipsychotics. Table 4 lists antipsychotic agents currently available in the United States. Nonadherence to medications is a significant problem; in a recent study, 74 percent of patients discontinued their medication within 18 months. Nonadherence often leads to relapse of symptoms. Atypical antipsychotics were initially thought to help with adherence because of their lower rate of neurologic side effects. However, meta-analyses have found that drop-out rates and relapse prevention are no better with atypical antipsychotics than with neuroleptics. Meta-analyses also have found that in terms of symptom scores and drop-out rates, atypical antipsychotics are better than high dosages (i.e., more than 12 mg per day) of haloperidol (Haldol); there was no advantage when the dosage of haloperidol was less than 12 mg per day. In other words, many of the perceived benefits of atypical antipsychotics actually were a result of the lower neurologic side effect profile.

### Table 3. Differential Diagnosis of Schizophrenia

<table>
<thead>
<tr>
<th>Alternative diagnosis</th>
<th>Distinguishing features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brief psychotic disorder</td>
<td>Presence of delusions, hallucinations, disorganized speech, or grossly disorganized or catatonic behavior lasting at least one day but less than one month</td>
</tr>
<tr>
<td>Delirium</td>
<td>Multiple underlying etiologies; symptoms often similar to positive symptoms of schizophrenia but with a much shorter course</td>
</tr>
<tr>
<td>Delusional disorder</td>
<td>Delusions are not bizarre, and there are no other characteristics of schizophrenia</td>
</tr>
<tr>
<td>Medical illnesses</td>
<td>Illnesses that may cause schizophrenia-like symptoms include hepatic encephalopathy, hypoglycemia, electrolyte abnormalities (e.g., hyponatremia, hypercalcemia, hypocalcemia, hypomagnesemia), and sepsis; symptoms resolve with treatment of underlying condition</td>
</tr>
<tr>
<td>Medication-induced disorder</td>
<td>Medications that may cause schizophrenia-like symptoms include anticholinergics, anxiolytics, digoxin, phenytoin (Dilantin), steroids, narcotics, and cimetidine (Tagamet); symptoms resolve with discontinuation of medication</td>
</tr>
<tr>
<td>Mood disorders with psychotic features</td>
<td>No major depressive, manic, or mixed episodes have occurred concurrently with active phase symptoms; or, if they have occurred, their total duration has been brief relative to the duration of the active and residual symptoms</td>
</tr>
<tr>
<td>Pervasive developmental disorder</td>
<td>Recognized during infancy or early childhood; absence of delusions and hallucinations</td>
</tr>
<tr>
<td>Psychotic disorder NOS</td>
<td>This diagnosis is made if there is insufficient information available to choose between schizophrenia and other psychotic disorders</td>
</tr>
<tr>
<td>Schizoaffective disorder</td>
<td>Lasts one to six months; diagnosis does not require a decline in functioning</td>
</tr>
<tr>
<td>Schizotypal personality disorder</td>
<td>Pervasive patterns of social and interpersonal deficits beginning in early adulthood; accompanied by eccentric behavior and cognitive or perceptual distortions</td>
</tr>
<tr>
<td>Substance abuse</td>
<td>Multiple substances (e.g., hallucinogens, narcotics, alcohol) and withdrawal from these substances can cause delusions and hallucinations</td>
</tr>
</tbody>
</table>

NOS = not otherwise specified.
of the excessive doses of first-generation antipsychotics that were used for comparison in randomized trials.\textsuperscript{17}

Evidence suggests that delays in initiating therapy with antipsychotics may result in a lifetime deleterious effect on psychotic episodes and social adjustment.\textsuperscript{19,20} If initiation of antipsychotic therapy is delayed because of limited psychiatric resources, family physicians should consider starting medications instead.

ADVERSE EFFECTS

Prescribers should be aware of the potential adverse effects of antipsychotics and when the effects are likely to occur. The most concerning side effects of first-generation antipsychotics are neurologic (Table 5\textsuperscript{15}). The Abnormal Involuntary Movement Scale can be used to help monitor the development of involuntary movements associated with neurologic side effects.\textsuperscript{21}

Although newer atypical antipsychotics are associated with fewer neurologic side effects, they confer a higher risk of metabolic side effects such as diabetes, hypercholesterolemia, and weight gain. The comparative risk of diabetes-related side effects of several of the most common antipsychotics (atypical and conventional) are shown in Table 6.\textsuperscript{22}

Although atypical antipsychotics can cause weight gain, this effect is independent from the development of diabetes; the exact mechanism by which atypical agents might cause diabetes is unknown.\textsuperscript{22,23} In one retrospective cohort study of 3,015 patients comparing olanzapine (Zyprexa) with risperidone (Risperdal), both were associated with gaining weight in the first year but only olanzapine was shown to be associated with the development of diabetes.\textsuperscript{23} The diabetogenic potential of antipsychotics appears to be reversible if the medication is discontinued.

There have been no controlled trials on the effectiveness of long-term monitoring of biomedical markers (e.g., weight, blood sugar and cholesterol levels) in patients taking atypical antipsychotics, but the risk of metabolic side effects is high enough that regular monitoring is recommended by several consensus panels (Table 7\textsuperscript{24}).\textsuperscript{22,24} There are few or no data on the relative frequency that these tests should be performed, and no data to show that monitoring affects disease-specific or all-cause mortality rates.
Tardive dyskinesia is a common late side effect of prolonged treatment with antipsychotics. Stopping the causal antipsychotic does not diminish the chronicity and severity.25-28

To help manage side effects of drug treatment, family physicians should inquire about positive and negative symptoms at every patient visit, and they should regularly communicate with patients’ mental health professionals about changes in symptoms, new lab results, and prescribing and monitoring roles.

**Psychosocial Treatments**

Individual, group, and family treatments have been developed as therapies for persons with schizophrenia. Family interventions include therapy with individual families, psychoeducation with groups of families, and family group therapy.29 These interventions offer support, education about the illness, and options for reducing critical and emotionally overinvolved attitudes and behaviors toward the patients.

Family treatments have the most empiric support for improving symptoms and reducing hospitalizations.30 These treatments are based on early findings that family environments that were high in “expressed emotion” (either critical and rejecting or emotionally overinvolved) were associated with relapse in patients with schizophrenia.31-34 Multiple studies have shown that family interventions reduce relapse rates and improve symptoms, adherence to medications, and functioning.30 However, a recent review suggested that there are weaknesses in many family intervention studies, and that there is a need for additional investigation.35

There are several psychosocial rehabilitative interventions that have been shown to be effective in improving the quality of life in patients with schizophrenia. The Intensive Psychiatric Rehabilitation Treatment, which is a program that teaches living, job, and social skills to patients, has resulted in improvements in functioning.36 Social skills training has improved independent living skills37-40; supported employment programs have shown

**Table 5. Neurologic Side Effects of Antipsychotics**

<table>
<thead>
<tr>
<th>Side effect</th>
<th>Features</th>
<th>Time of maximal risk</th>
<th>Proposed mechanism</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute dystonia</td>
<td>Muscle spasms of the tongue, face, neck, and back; may mimic seizures; not hysteria</td>
<td>One to five days</td>
<td>Unknown</td>
<td>Antiparkinsonian agents are diagnostic and curative*</td>
</tr>
<tr>
<td>Akathisia</td>
<td>Motor restlessness; not anxiety or agitation</td>
<td>Five to 60 days</td>
<td>Unknown</td>
<td>Reduce dose or change drug; antiparkinsonian agents (benzodiazepines or propranolol [Inderal])† may help</td>
</tr>
<tr>
<td>Parkinsonism</td>
<td>Bradykinesia, rigidity, variable tremor, mask facies, shuffling gait</td>
<td>Five to 30 days (can recur even after a single dose)</td>
<td>Antagonism of dopamine</td>
<td>Antiparkinsonian agents helpful</td>
</tr>
<tr>
<td>Neuroleptic malignant syndrome</td>
<td>Catatonia, stupor, fever, unstable blood pressure, myoglobinemia; can be fatal</td>
<td>One or more weeks (can persist for days after stopping medication)</td>
<td>Antagonism of dopamine may contribute</td>
<td>Stop medication immediately; dantrolene (Dantrium) or bromocriptine (Parlodel)‡ may be helpful; antiparkinsonian agents not effective</td>
</tr>
<tr>
<td>Perioral tremor (i.e., rabbit syndrome)</td>
<td>Perioral tremor (may be a late variant of parkinsonism)</td>
<td>After months or years</td>
<td>Unknown</td>
<td>Antiparkinsonian agents often helpful</td>
</tr>
<tr>
<td>Tardive dyskinesia</td>
<td>Oral facial dyskinesia; widespread choreoathetosis or dystonia</td>
<td>After months or years (worse on withdrawal)</td>
<td>Excess function of dopamine hypothesized</td>
<td>Prevention crucial; treatment unsatisfactory</td>
</tr>
</tbody>
</table>

*—Many drugs have claimed to be helpful for acute dystonia. The most commonly employed treatments are diphenhydramine (Benadryl) 25 or 50 mg intramuscularly, or benzotropine (Cogentin) 1 or 2 mg intramuscularly or slowly intravenously, followed by oral medication with the same agent for a period of days to perhaps several weeks.

†—Propranolol often is effective in relatively low dosages (20 to 80 mg per day). Selective beta-α1-receptor antagonists are less effective.

‡—Despite the response to dantrolene, there is no evidence of an abnormality of Ca^2+ transport in skeletal muscle; with lingering neuroleptic effects, bromocriptine may be tolerated in large dosages (10 to 40 mg per day).

improvements in the number of hours worked and total wages earned; and in-home crisis intervention demonstrates promise by reducing treatment drop-out rates. Studies have shown that individual cognitive behavior therapy for schizophrenia reduces positive and negative symptoms, but currently there is no evidence that it reduces relapse rates.

Prognosis
Understanding the potential course of disease can help guide treatment. Patients with schizophrenia have a high rate of substance abuse, and those with substance abuse have their first hospitalizations at earlier ages, have more frequent hospitalizations, and have more interpersonal and family discord. The strength of patients’ commitment to their delusions is directly proportional to their likelihood of rehospitalization. Patients with poor executive functioning (i.e., skills involving problem solving, setting and attaining future goals, and decision making) use outpatient services at a higher rate and therefore may require increased support to maintain their independence.

Patients with severe psychotic disturbances have a higher likelihood of aggressive behavior than those with fewer psychotic symptoms. Patients with schizophrenia also have a low marital rate and high divorce rate. Accelerated heart disease is the most common cause of death in patients with schizophrenia; the risk of dying from cardiovascular disease is two to three times higher than in the general population. This risk is accelerated because their rate of cigarette smoking is two to four times higher than that of the general population.

Table 6. Ranking of Antipsychotics According to Risk of Diabetes-Related Conditions*

<table>
<thead>
<tr>
<th>Condition</th>
<th>Clozapine (Clozaril)</th>
<th>Olanzapine (Zyprexa)</th>
<th>Risperidone (Risperdal)</th>
<th>Conventional antipsychotics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes (prevalence)</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Hyperglycemia (fasting)</td>
<td>4</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Hyperinsulinemia (fasting)</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Elevated total cholesterol levels</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Elevated triglyceride levels</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Elevated BMI</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Elevated plasma uric acid levels</td>
<td>4</td>
<td>2</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td><strong>Sum of ranks†</strong></td>
<td><strong>26</strong></td>
<td><strong>21</strong></td>
<td><strong>12</strong></td>
<td><strong>11</strong></td>
</tr>
</tbody>
</table>

**NOTE:** 1 = lowest risk; 4 = highest risk.

BMI = body mass index.

*—Adjusted for diagnosis, duration of antipsychotic treatment, other medications, family history of diabetes, ethnicity, and smoking status.

†—The parameters are not equivalent in their contribution to the pathology of diabetes or its cardiovascular complications; no attempt has been made to weight the sums of rank orders. Low rank order equals low prevalence or risk.

Adapted with permission from Lean ME, Pajonk FG. Patients on atypical antipsychotic drugs: another high-risk group for type 2 diabetes. Diabetes Care 2003;26:1599.

Table 7. Physical Health Monitoring for Patients Taking Antipsychotics

<table>
<thead>
<tr>
<th>Disease process</th>
<th>Antipsychotics</th>
<th>Monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity</td>
<td>First- and second-generation</td>
<td>Calculate BMI at initiation of medication and every six months; encourage patients to monitor their weight; recommend weight-management program for patients with more than a 1-unit increase in BMI</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Second-generation</td>
<td>Perform baseline plasma glucose before initiation of medication; measure A1C four months after initiation of medication; inquire about polyuria and polydipsia at each visit</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>Second-generation</td>
<td>Perform a lipid screening at initiation of medication; repeat lipid screening every six months if abnormal and every two years if normal; follow NCEP recommendations for lipid management</td>
</tr>
</tbody>
</table>

BMI = body mass index; NCEP = National Cholesterol Education Program.

Information from reference 24.
Persons with schizophrenia also smoke more than patients with other mental disorders. In several studies, 90 percent of hospitalized patients with schizophrenia smoked. Nicotine has a possible positive effect on cognitive functioning in patients with schizophrenia, which may explain the high rate of smoking. Suicide is also a common cause of death in patients with schizophrenia; it has a 10 percent lifetime risk. The risk of suicide is strongly associated with depression, previous suicide attempts, drug abuse, agitation or motor restlessness, fear of mental disintegration, poor adherence to treatment, and recent loss. Overdose of treatment medications as a method of suicide is not common because antipsychotics have a high therapeutic index (i.e., lethal doses are much higher than the dosages that produce a therapeutic effect).

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Author disclosure: Nothing to disclose.

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Mood Disorders


18

Affect and Emotional Disorders

I wish to inform you that I have received the cake. Many thanks, but I am not worthy. You sent it on the anniversary of my child’s death, for I am not worthy of my birthday; I must weep myself to death: I cannot live and I cannot die, because I have failed so much. I shall bring my husband and children to hell. We are all lost: we won’t see each other any more; I shall go to the convict prison and my two girls as well. If they do not make away with themselves because they were born in my body.

A patient of Emil Kraepelin (1905)

Assessing and observing the state of, and changes in, mood is essential in psychiatry, but at the same time requires skill. Part of the problem has always been the conceptual confusion and lack of cohesive psychopathological theory that has traditionally been associated with disturbance of affect (Berrios, 1985). In a study of patients with unsolved diagnostic problems at the time of discharge from hospital, atypical psychotic depression was found, at follow-up, to be the condition most frequently responsible for doubt (Anstee and Fleminger, 1977). In another study depressed affect was a major cause of somatic problems without physical pathology (Brenner, 1979). However, the terms used are not standardized, nor mutually exclusive. Different languages, unlike the names given to physical objects, have an entirely different range of descriptions of mood so that one is left wondering whether it is just the terms which differ in different cultures or perhaps even the experience of emotion itself. So Angst cannot be translated exactly into English with a single equivalent word; neither can depression be precisely translated into German. The word feeling describes an active experience of somatic sensation, touch, as well as the passive subjective experience of emotion. Emotion, according to Whybrow (1997), ‘is actually memory and feeling intertwined’. Feelings are also personal convictions, predictive forecasts and social sensibilities. All these nuances of meaning are somewhat different from the associations of the word mood.

Traditionally, feeling has been used to describe a positive or negative reaction to an experience; it is marked but transitory. Affect is used to describe differentiated specific feelings directed towards objects. Mood is a more prolonged prevailing state or disposition. In practice, these terms are used more or less interchangeably. Similarly, emotion is often used with regard to the physiological and psychosomatic concomitants of mood, but this use is not exclusive.

Mood describes the state of the self in relation to its environment. There is an enormous range of variation of what could reasonably be called normal mood. Pathological mood, that is mood from which the patient suffers or mood which causes disturbance or suffering to others, also varies very greatly, and the extent to which it is acceptable to others in its expression is different in different social contexts. The clinician has to ask two questions concerning the mood of his patient, first is the person suffering? Second, is the expression of mood inappropriate in this social setting? Psychopathology of mood is confined to those situations where there is an affirmative answer to at least one of the questions, and treatment is directed towards improving the mood.

Like other human characteristics mood arises in the context of a diathesis. It is the physical constitution which forms the tendency for developing, for example, a prolapsed intervertebral disc; in the mental realm personality is closely associated with the type, quality and direction of mood. So, a person of cyclothymic personality is more prone to morbid states of elation and excessive activity, or taciturn depression and retardation.

In the phenomenological assessment of mood, Jaspers (1959) has concentrated on three main aspects: the involvement of self, the contrast of opposites, and the nature of the object of feeling. Feelings are a feature of self, but by a process of empathy they may be ascribed to other objects of awareness. So I experience my own feeling of sadness; I can also make a judgement that another person is sad because he looks sad, and even that a picture is sad because of its content and the affect that it evokes in me. A diagrammatic representation of some of the subjective experiences of mood is shown in Figure 18.1.

![Figure 18.1 Subjective evaluation of mood](image)

Affect is experienced in contrasts. This customary human characteristic of polarizing mood into opposites is made use of in the applications of Personal Construct Theory (Kelly, 1955), and in the use of Visual Analogue Scales (Aitken, 1969) in rating change of mood (Figure 18.2). Feelings may have a definite object, for instance a fear of cats, exposure to cats or to the idea of cats then evokes the emotion of anxiety. Feelings may also be without object, for example free-floating anxiety in which the sufferer is in no doubt about the reality of his somatic and psychological symptoms of anxiety but can ascribe it to no definite cause, precipitant or object.

Communication of Mood

‘No man is an island, entire of itself’ (John Donne, 1572–1631), and in no area of life is this more true than that of feelings. Our feelings are very much affected by those around us. They are observable and understandable to other people and this is not accidental; they are actually signalled as a non-verbal message. The affect itself is not directed towards another person, but the expression of the affect is conveyed both deliberately and unintentionally to others.
There can be vital feelings affecting the whole body in which an emotion is described subjectively as being physical but affecting the whole organism in a complete way. Psychological feelings are the emotions we commonly describe as sadness, joy and so on. Finally, Jaspers describes spiritual feelings, an expression which falls uncomfortably on the uncomprehending ears of our spiritually blunted secular culture.

3 It is possible to evaluate emotion according to its biological purpose. This would provoke a discussion of the theory of instinct.

4 Feeling state is a description of all the different feelings occurring at any one time and describes the affective state of the individual at that time, for example a state of arousal, feeling of anergia.

5 Emotion has been categorized traditionally according to duration and intensity. Thus, feeling is an individual emotional reaction. Affect is a complex but momentary emotional perturbation. Mood is a more prolonged emotional state which influences all aspects of the mental state.

6 There is an important distinction within the ambiguous word feeling which means both emotions and sensations. Emotion refers to a state of the self, whilst sensation refers to elements of perception.

**Pathological Changes in Mood**

**Changes in Mood**

Most often in psychiatric practice, subjective description of change in the experience of emotion is for the worse – a state of dysphoria, meaning the condition of ‘being ill at ease’; more rarely the patient may describe the onset of ecstasy or euphoria. The subjective experience of change of mood can be quantified approximately and represented graphically as in Figure 18.3, which shows part of a mood chart a previously depressed patient had recorded; he had noticed an association between an acute attack of bronchitis and exacerbation of depressive symptoms.

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**The Categorization of Emotion**

Jaspers has categorized feelings in the following ways.

1 According to the object of the emotion. This would include such diverse feelings as fear of snakes, patriotism, servile submission. The range of possible contents of emotion is, of course, limitless.

2 Feelings can be categorized according to their source. These may be localized feeling sensations: affect experienced in individual regions or areas of the body.

---

**Figure 18.2** Visual analogue scale

Research on facial expression indicates seven main groups of emotion that are discriminated by an observer (Argyle, 1975). These are happiness, surprise, fear, sadness, anger, disgust, contempt, and interest. Emotions are communicated non-verbally by different parts of the body, for example by the face (especially the eyes), gesture, posture, tone of voice and general appearance, especially the choice of clothes. In assessing the emotional state of others, those who are better at communicating emotion non-verbally are also better at reading it. The communicator assesses the other's affective response but he also in part evokes it. A person who is cheerful on meeting someone else will greet him cheerfully and induce a feeling of cheerfulness, even if transitory, which he then reads as the other person being cheerful also. This has important implications in the way that mood is assessed. It would seem that emotion is evaluated empathically. Without having to go through this elaborate argument in words, the observer says to himself ‘if I felt how I estimate the feelings of that person from his appearance, I would feel very unhappy; he is unhappy’. This is, of course, the empathic method as described earlier, and it takes place spontaneously and without deliberate training. Assessment of others’ mood does not need to become verbal to be acted on. It takes place rapidly and is followed by the appropriate behavioural response from the observer.

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**Figure 18.3** Mood chart kept by a depressed patient who had had acute bronchitis
Bodily feelings associated with mood

Mood and somatic symptoms have been discussed in Chapter 15. In a number of cultures and languages, depression is considered to have an anatomical location to such an extent that the mood state and the part of the body become synonymous. Melancholia literally means black bile; similarly in Urdu the word jee, meaning self, describes the hypochondrium anatomically and comes to mean depression, that is depression is a central assault upon the well-being of the self.

Changes in bodily feeling are important in a number of conditions. Physical illness frequently precipitates a loss of the accustomed sense of well-being. This is subjectively experienced as a generalized lowering of vitality and may be associated with other psychological abnormalities, for instance hypochondriasis or dissociation.

Feelings of capacity

There may be a loss of the normal feeling of self-sufficiency and the appropriate sense of self-esteem. Competence in any field of life is linked with a reasonably accurate knowledge of self-capacity – the ability to know one’s own limits and not attempt the impossible. Loss of the accustomed feeling of capacity to achieve what is known to be within that person’s capability may occur with psychotic depression; it may also be a neurotic development. The resultant feelings of helplessness and hopelessness have been described by Schmale (1958) and others in the ‘giving-up-give-up complex’.

Increased feelings of capacity may be experienced in mania. A young, unemployed lorry driver was an in-patient undergoing investigation on a medical ward. He developed a manic episode and conducted his own ward round late one night. Going to the bed of a particularly timid and sick patient he announced ‘I am a Professor of Medicine from the British Medical Association. With the treatment you are getting from Dr X you are going to die, but don’t worry. I’ll make sure you’re alright’.

Absence, blunting and flattening of feeling

Apathy is the absence of feeling; there may also be blunting or flattening of affective response. The patient himself is often not aware of his deficiency but when pointed out to him, may agree that there is a lack of any sort of emotional reaction. Apathy is often associated with anergia and lack of volition. The schizophrenic patient so afflicted has no desire or internal drive to work or find a job. He may be so apathetic that he does not dress or even feed himself properly. He will respond to continual persuasion or cajoling, but otherwise remains inert and passive.

The terms ‘blunting’ and ‘flattening’ are not identical, although both may occur in schizophrenia. Blunting implies a lack of emotional sensitivity, such as that displayed by the girl with schizophrenia who, with obvious relish for the sensational effect, took her visitors up to the bedroom to show them her mother who had been dead for 48 hours. Flattening is a limitation of the usual range of emotion and is often associated with anergia amongst people with schizophrenia. The individual does not express very much affect in any direction, although that which is expressed is appropriate in direction.

Feeling of a loss of feeling

This is subtly but significantly different from apathy. It is experienced by the patient as a loss, a deficiency and is all-pervasive – anger, love, pleasure and so on. The patient resists or does not understand it, suffers very greatly and often feels guilty about the feeling. It is a subjective experience of loss of feelings that were formerly present, rather than an objectively observed absence. A depressed young woman said ‘I have no feelings for my children. That is wicked. They are beautiful children.’ A person with religious belief may experience this loss of feeling with a religious content; they no longer believe in God. On more detailed eliciting of their subjective experience, they are likely to describe a loss of the feeling of assurance associated with their faith, rather than any actual change in the content of their beliefs. This effect occurs particularly in depressive psychosis, but also occasionally with personality disorders and schizophrenia. Milder forms are experienced as depersonalization or deaffectualization (see Chapter 14): the patient complains that his feelings are numbed, diminished, made remote from himself, to which is ascribed the unmelodious word, de-affectualization.

Anhedonia

In anhedonia there is a total inability to enjoy anything in life or even get the accustomed satisfaction from everyday events or objects. ‘A loss of ability to experience pleasure’ (Smillie, 1993). The term was originally introduced by Ribot (1896) and considered to be a prominent symptom of depressive illness by Klein (1974), probably the best clinical marker predicting response to treatment. This would seem to be a fundamental symptom of depressive illness. A highly intelligent and perceptive man suffering from psychotic depression said ‘I have a sort of uncanny feeling, I know what I am reading but I am not at all amused by it.’ Anhedonia is also described as a symptom in schizophrenia, in which it is especially likely to be social – absence of the ability to feel pleasure in relationships (Cutting, 1985).

Anhedonia is therefore one component of the loss of the capacity to feel feelings. Often the very way the patient describes the loss of the ability to feel feelings demonstrates that feelings are still there. A girl described how she no longer felt anything for her boyfriend: ‘It is nothing to do with him, it is me’. She did not feel she could love anyone and she was in a quandary because she thought it impossible to proceed with the affair, but could not break it up ‘because it would hurt him very much and I would hate to do that’ – thus demonstrating her continuing capacity for emotion.

Feeling of impending disaster

This experience of dread or apprehension is, of course, a common and normal emotion and would be quite appropriate, for example, for the idle student awaiting his examination results. However, a nameless dread without reasonable cause is seen with various morbid mental disorders. A graphic and terrifying example was written by the poet William Cowper (1822, published posthumously) during an acute episode of psychotic depression:

Hatred and vengeance, – my eternal portion.
Scarcely can endure delay of execution. –
Wait with impatient readiness to seize my
Soul in a moment.
Ecstasy

Ecstasy and euphoria occur in health; psychiatrists are only concerned with them when they occur inappropriately, or are prolonged excessively, or are present to an abnormal degree. Heightened states of happiness such as euphoria and ecstasy sometimes occur in people with mental illness or abnormality of personality. Understandably, most psychiatrists writing about the mood state of ecstasy have described its occurrence in psychotic patients, but with the pattern towards increased numbers of psychiatric out-patient referrals, patients with neurotic disorder, giving a previous description of less bizarre ecstatic symptoms are now seen. The patient may describe a calm exalted state of happiness amounting to ecstasy, although this tranquil mood state is relatively uncommon and usually short lived. In schizophrenia, ecstatic mood may be associated with exalted delusions, for example the chronic patient who sat placidly entrapped on a long-stay ward, knowing herself to be the Queen of Heaven and waiting for a messenger to inform her that she was to take over the rule of the world. Ecstatic states, usually with a histrionic flavour, may occur in dissociative disorder, and may be associated with religious stigmata (Simpson, 1984). Bizarre, mass hysterical phenomena, often with religious associations, are usually of this type, for example in the Devils of Loudon as described by Aldous Huxley (1952). The social, institutional and group psychological prerequisites for the development of epidemic or mass hysteria (Sirois, 1982) are usually present in these situations and mismanagement is usually responsible for the development from isolated hysteria in one individual to an epidemic. Ecstasy, solemn elation or excessive exuberant expansiveness may also be seen in epilepsy and in other organic states, for example in general paresis.

Characteristic of ecstasy is that it is self-referent; for example the flowers of spring ‘open for me’. There is an alteration of the boundaries of self so that the person may feel ‘at one with the universe’, or he may ‘empty myself of all will’ so that ‘I am nothing but feelings’. The change in ego boundaries does not usually have the aspect of interference with self that accompanies passivity experiences. In ecstasy, the abrogation of self is experienced as being voluntary.

Euphoria is a state of excessive unreasonable cheerfulness; it may be manifested as extreme cheerfulness as in mania, or it may seem inappropriate and bizarre. It is commonly seen in organic states, especially associated with frontal lobe impairment.

Feelings attached to the perception of objects

Objects may evoke an emotional response in a normal person, for instance a comfortable feeling of familiarity towards an armchair in which one rests after an energetic walk, or apprehensive dislike towards a dentist’s chair. This normal affective response may be exaggerated pathologically. Excessive feelings of fear amounting to terror may remain associated with objects, or, alternatively, profound and inappropriate happiness. The objects to which affect is attached may not only be physical, inanimate objects but also thoughts, and patterns of thoughts, and people. The occurrence of certain ideas may regularly be associated with specific pathological emotion, perhaps resulting in phobia. Any object of perception may be invested with idiosyncratic affect.

Feelings directed towards people

These may be disturbed in a number of different ways. Affect may be absent or deadened, increased, and excessive or distorted. It may also be ambivalent—both loving and hating, rejecting and over-protecting synchronously. A girl described in Chapter 15, suffering from anorexia nervosa, would take great care to cook enormous meals for her twin sister, to whom she was very close, the sister became grossly obese whilst the patient vanished almost to a skeleton. In answer to remonstrations about feeding her sister, she said: ‘I look horrible, so she should look horrible as well’.

Free-floating emotion

This is commonly described in psychiatric disturbance, and in his original description of anxiety neurosis Freud (1895) considered that the condition was characterized by free-floating anxiety. A powerful affect seems to have no goal and is associated with no object. The patient describes himself as feeling generally anxious, not anxious about anything in particular but just anxious. This free-floating anxiety has somatic and psychological concomitants. It may seem to be localized physically in certain areas of the body. Other free-floating affects occur such as restlessness, tension, gloom, despondency, euphoria, irritability, and so on.

Experience and expression of emotion

The experience and expression of emotion are separate, but closely linked. The expression of mood may be abnormal in a number of ways. Mood may be censored or denied so that it never gains expression. It may be altered, and this is the theoretical explanation of conversion with dissociation: that is an affect becomes unbearable intense and is therefore converted by a presumed unconscious mechanism into physical symptoms. The expression of mood may be impulsive and explosive, with inadequate control and a lack of empathic feeling.

Hypochondriasis is very commonly associated with disorders of mood, for example anxiety or depression. There is a background perception of sensations from many different parts of the body: skin surfaces, joints and viscera. A process of introspection and concentration of consciousness on individual organs or regions, coupled with the memory for past experience of the subject himself, or of the diseases of other people who he knows, causes him to experience these as morbid. When individual perceptions of the body become linked with unpleasant affect they become one of the bases of the multifaceted term hypochondriacal preoccupation.

A speculative hypothesis that clinicians have found helpful is the term alexitmyia which was coined by Sifneos (1972) to describe a specific disturbance in psychic functioning, characterized by difficulties in the capacity to verbalize affect and elaborate fantasies. This was originally introduced to describe psychosomatic disorders occurring in individuals with difficulty expressing their emotions. The link with absence or diminution of fantasy is a consistent finding (Nemiah and Sifneos, 1970). The communicative style shows markedly reduced or absent symbolic thinking so that inner attitudes, feelings, wishes and drives are not revealed; few dreams and a paucity of fantasies are reported (Taylor, 1984). Thinking is literal, utilitarian and concerned with the minutiae of external events. These individuals have great difficulty in recognizing and describing their own feelings and in discriminating between emotional states and bodily sensations. They show a stiff robot-like existence ‘almost
as if they are following an instruction book; there may be stiffness of posture and lack of facial expression. They show an impaired capacity for empathy in their interpersonal relationships. Alexithymic characteristics have been found, especially amongst patients with psychosomatic disorders, somatoform disorders, psychogenic pain disorders, substance abuse disorders, posttraumatic stress disorder, masked depression, character neuroses and sexual perversions.

Somatization in patients with mental disorder can be defined as the selective perception and focus on the somatic manifestations of the disorder with denial or minimization of the affective and cognitive changes (Katon et al., 1982). As a method of expression of emotion it is frequently reported in transcultural studies, especially in the Indian subcontinent, according to Luck (1982). Murphy and coworkers (1984) studied basic depressive symptomatology in thirty countries and showed how culture changes illness. The way dysphoria is expressed, Bavington (1982), studying depression in a predominantly Pathan culture in Pakistan, found somatization to be expressed in 45 per cent of cases; hypochondriasis was present in 55 per cent. Hysterical (disassociative) features in 60 per cent, feelings of guilt in 50 per cent, paranoid ideas in 18 per cent, suicidal thoughts in 75 per cent, diurnal variation in 18 per cent, retardation in 50 per cent, and irritability in 80 per cent of depressed patients. Bavington explains these somatic ideas by the presence of vital feelings rather than poverty of language. Mumford (1992) found that patients with psychiatric disorders originating from India and Pakistan typically communicate their distress in terms of somatic symptoms; somatic presentation was common in general hospital settings where psychiatric disorders were often unrecognized and untreated. The use of somatic symptoms and somatic metaphors to communicate emotional distress is found in all languages and cultures. Complaining of emotional dysphoria in terms of somatic symptoms may reflect the limitation of the medical profession in listening to complaints rather than a poverty of language or paucity of verbal expression of the patient.

Vital Feelings

Vital feelings was a term used by Wernicke (1906) to describe certain somatic symptoms occurring in the affective psychoses. The word vital comes from the concept of the vital self which describes the close relationship of the body to awareness of self, the way we experience our bodies and the impression we consider our physical presence makes on others. So, vital feelings are those that make us aware of our vital self. These are the feelings of mood which appear to emanate from the body itself; localized and somatized affect. For example, depressed patients commonly complain of headache. On more informed enquiry the patient may say "it's not exactly a pain, but more an unbearable feeling of pressure like a tight band around the head", 'a feeling of misery, like a black cloud pressing on my head'. The head is the commonest site for vital feelings, but they may also occur in the abdomen 'I have a dull feeling in my bowels, they are slowing down and blocking', in the chest 'it feels like a weight bearing down on my chest, stopping me breathing', in the eyes 'everything looks black, dark and dull; my eyes are heavy, I cannot see properly', in the legs 'my legs are terribly heavy; I cannot walk, I feel so exhausted'. They may occur in other regions of the body, for instance the bladder: the feet, the hair and so on. The features which appear to be constant are the association of the localized body sensation with the prevailing depressed mood, the sensation of weight, tension, heaviness, even depression, in the particular organ, and a consequent loss of function 'I cannot think properly... my bowels are blocked'.

Schneider (1920) considered vital feelings to be of paramount diagnostic significance in depressive illness, equivalent to the first-rank symptoms in schizophrenia, the core of cyclothymic depression and automatic in origin. He considered these feelings to be common in depression. It would seem that Dupre (1913), writing about what he called coenestopathic states, was describing the same symptom: "Coenestopathic states are, indeed, so common as to figure among the most frequent features of the psychoses". He described coenesthesia as the 'deep but more or less indefinite awareness that we have of our own bodies and the general tone of functional activity'. Coenestopathic states are 'the distressing feelings which emanate from one or other of the coenesthetic areas... a change in the normal quality of physical feeling in certain parts of the body'. They are localized but there is no local pathologic lesion. Dupre claimed that coenestopathic states were autonomous, not associated with other psychiatric disorders; but, in describing the affects with which they are associated, he appears to describe affective disorders. The mood of depression may be described as a global loss of vitality in which all functions are affected and all performances depressed.

A change in vital feelings does not only occur in depression. The bizarre feelings that the schizophrenic patient has about his body is a change in the way he expresses himself, often further elaborated by delusions. It should be noted that the term vital is used rather differently in vital anxiety states. These states have been described (Lopez Ibor, 1966) in which the anxiety is thought to be endogenous, developing relatively acutely in people of stable personality.

The depressive content of what phenomenologists would consider to be vital feelings varies very greatly: for example 'I have turned to stone... I have a feeling of depression in my chest... it is a pain, a knot, a weight... I have a cloud on my head, a feeling of nothingness.' Burns (1971) commented with regard to respiratory vital feelings 'A striking feature of the breathlessness described by the patients with depression was its fairly sudden onset and cessation, corresponding exactly with the onset and resolution of the depressive illness'.

Trehovani (1979) has considered that lowering of vitality is fundamental to the experience of depressive illness. He has described this as 'a lowering of vitality which is all-pervasive and leads to a marked loss of ability of the subject to function as he did before he became ill in terms of both mind and body'.

Religious Feelings

Expert knowledge of the abnormal does not preclude ignorance of the normal and the psychiatrist can never generalize from the sample of people selectively referred to him to the whole of mankind. This discrepancy can become very obvious in the area of ecstatic and religious experience. There is a need to acknowledge, take into account, have respect for, and use in treatment the patient's own subjective experience in this area (Sims, 1994). The psychiatrist sees a most unrepresentative group of those having some form of religious experience, which has been considered to amount to over 40 per cent of the adult population of the USA, more of whom are males than females, more are stable than unstable, and more happy than unhappy.
The anthropology of ecstasy (Lewis, 1971) can be traced through Christian and other cultures and only makes contact with recognizable mental illness at a few points. William James (1902), in The Varieties of Religious Experience, demonstrated the vast extent of the phenomenology of religion, and showed how unwise it would be to equate the surprising with the pathological. Once again, the phenomenological dichotomy of form and content is important. For a person whose predominant thinking in health is religious, the content of their mental illness, if they become psychiatrically disordered, may well be religious also; but it is the form of the condition by which the doctor will decide whether they are ill or not and, if so, what is the precise nature of their illness. Thus, although a religious person with manic-depressive illness in manic phase will describe bizarre religious flights of ideas, pressure of talk and ecstatic mood, there is no such entity ipso facto as 'religious mania'.

Accounts vary as to the extent of psychopathology amongst converts to religious groups and sects; it is probably associated with the nature of the group. Thus Ungerleider and Wellisch (1979) found no evidence of severe mental illness in one study, while Galanter (1982) described evidence of emotional problems amongst adherents to Divine Light, the Unification Church, Baba and Subud.

Suggestive indicators for establishing a religious experience as probably associated with psychiatric morbidity are
1. the phenomenology of the experience conforms with psychiatric illness
2. there are other recognizable symptoms of mental disturbance
3. the lifestyle, behaviour and direction of personal goals of the person subsequent to the event are consistent with the natural history of mental disorder, rather than with an enriching life experience
4. such behaviour is consistent with disorders in the person's personality.

With the following signs, the experience is more likely to be intrinsic to the person's belief and less likely to denote psychiatric illness
1. the person shows some degree of reticence to discuss the experience, especially with those he anticipates will be unsympathetic
2. it is described unemotionally with matter-of-fact conviction and appears 'authentic'
3. the person understands, allows for and even sympathizes with the incredulity of others
4. he usually considers that the experience implies some demands upon himself
5. the religious experience conforms with the subject's recognizable religious traditions and peer group.

In his clinical practice the psychiatrist is likely to come across patients describing religious experience: in some patients he will feel this is symptomatic of mental illness, but in others it is clearly intrinsic to the values of the patient and independent of illness even though illness may also be present.

Other Mood States and Feelings
There are many other possible mood states, some of them of clinical significance. Further discussion of anxiety and irritability and related psychiatric symptoms and conditions is found in Chapter 19. Phillips et al. (1997) have made a case for considering 'disgust' as a significant emotion for psychiatric consideration. The subjective experience is different from other unpleasant emotions and it often forms a prominent part of symptomatology in obsessive-compulsive disorder, phobias, depression, eating disorders and dysmorphic phobia. Disgust or towards oneself is also a frequent complaint, for example with some sexual disorders and other socially deviant behaviour.

Manic-Depressive Mood
Certainly since the writings of Kraepelin, the apparently opposite mood states of mania and depression have been recognized as occurring in the same illness - frequently at different times and stages of the illness in the same patient, more rarely at the same time in the same patient. The affective disorders are now recognized as a very considerable part of psychiatry with an impressive body of research in most relevant areas (Paykel, 1992). Although depression occurs more frequently than mania in bipolar illnesses, the first attack of mania may occur even after the age of 60 (Shulman and Post, 1980).

Although they are described separately, it is important to realize that these mood states may occur together. Mania and depression are not opposite mood states; they are both pathological, and the opposite of either would be freedom from morbid emotion. Agitation and overactivity may occur with depression, irritability and a feeling of frustration with mania. It is usual for a person to go through a depressive phase before becoming manic, and again on the return from mania before reaching a state of normal mood. A patient, now depressed, having previously been manic, described this 'The first fine careless rapture has disappeared. I feel more tired and moody.'

Depression of Mood
Core experience: psychological and physical
Depression of mood is very common, and depression of such persistence and intensity as to be regarded as illness frequently occurs. There is considerable discussion as to what is the central core of depression. Of course, arguments advocating biochemical, psychodynamic, or conditioning factors as initiating causes are mutually exclusive. Depression affects virtually all physical and psychological functions, for example, using a tachistoscopic method, Powell and Hemsley (1984) were able to show that depression even influenced perception.

The word depression is a misnomer; as depressive illness may occur without the patient making a complaint of depression as a symptom (depressio sine depressione), for this reason the term melancholia may be preferred: although this literally means black bile it has come to be accepted as a medical illness. It was the term used by Lewis (1934) in his classical description of depressive states in a detailed study of sixty-one cases; this has influenced all subsequent investigation of the condition. However, there is some nosological confusion as this term is used in DSM IV (American Psychiatric Association, 1994) to describe just one aspect of major depressive disorder (major depressive episode with melancholic features). Melancholia is the preferred term for Whybrow (1997) who considers that it 'better captures the
"veritable tempest in the brain" that marks the experience of inner turmoil and confused thinking as harmony and emotion drain away, often to be replaced by a withered imitation of life. The subjective symptoms of depression are very variable. The mood varies from indifference and apathy to profound dejection, despondency and despair. Anhedonia, the complete inability to experience pleasure, is a constant feature. It is experienced as joylessness and revealed in facial expression, speech, behaviour, lifestyle and in the patient's account of personal experience.

A slowing down of the ability to initiate thought or action is noted by the observer as retardation. A patient, describing this after recovery, said 'it feels as if treacle has been poured into my head through your ears'. Psychotic retardation is experienced subjectively as an inability to fulfil normal obligations. As loss of coping. The proneness to self-blame often results in the patient describing himself as lazy and good for nothing. There is a catastrophic lowering of self-esteem as a prominent cognitive component.

Beck (1967; Beck et al., 1979) considered that there is a pre-depressive constellation of attitudes or assumptions developed from previous experiences. The patient has a negative view of himself, he interprets his ongoing experiences in a negative way and he has a negative view of the future.

Brown and Harris (1978), in a study of depressed women in inner London, considered that provoking agents such as adverse life events potentiated already existing vulnerability factors to promote depression. These latter included lack of a close confidant, loss of mother before the age of 11, three or more children under the age of 14 at home, and no employment outside the home. Ingham et al., (1986), in Edinburgh, studied the relationships between self-esteem, vulnerability and psychiatric disorder in the community. These associations are complex, but certain life circumstances influence feelings of self-esteem independent of illness. Anxiety, as well as depression, has effects upon self-esteem.

Depression is associated with many definite life periods and changes. For example, Kitamura et al., (1993) have shown that affective disorder frequently develops during pregnancy, 68 per cent of those affected experienced onset in the first trimester. Adverse psychosocial factors such as early loss of either parent, crowded accommodation and negative attitude towards the pregnancy by the husband were more likely to be followed by depression, which occurred more often in first pregnancies.

Agitation and purposeless restlessness add to the discomfort, and to the inability of the depressed person to achieve anything. This anxiety and preoccupation with gloomy thoughts impairs concentration. Diurnal variation of mood is often prominent with the patient feeling at his worst, and perhaps most suicidal, when he wakes early in the morning or, alternatively, somewhat later in the morning. The degree of depression and misery may sometimes successfully be concealed; this is the presentation of depressio sine depressione (smiling depression) in a patient who appears not to be depressed in the consulting room but may, much to his doctor's dismay, kill himself. The concealment is probably conscious and may be associated with habitual masking of the expression of emotion or alternatively aimed at avoiding treatment.

Concentration, application and decision making become difficult, painful and sometimes impossible. The person describes difficulty or impossibility in fantasy and recollection of emotion. This is described as loss of memory and loss of feeling. Often this loss of mental function makes the patient believe he is 'going mad' or 'losing his mind'; a sort of mental hypochondriasis. Physical retardation may become the focus for hypochondriacal beliefs about the body: 'I am constipated . . . my bowels are totally blocked'. A very depressed middle-aged woman described her bodily feelings thus: 'I have a feeling like having an injection at the dentist's. My face feels numb, but at the same time painful all over.'

Anxiety is a common concomitant with depression and may completely obscure the latter. In agitated depression, agitation and restlessness is extreme and the patient carries a serious risk of suicide. Histrionic behaviour may also obscure the underlying depressive illness. A patient who was actually profoundly depressed kept picking his skin and pulling her hair, saying, 'look, I can't feel anything when I do this to myself'.

The affect of depression may be localized somatically in vital feelings (see above). It may take the form of profound misery or depression. There is usually a feeling of loss of capacity, helplessness and a feeling that the patient cannot cope. Absence of feelings is often described, or it may be described as an inexplicable loss of feelings 'that ought to be there'.

Feelings of guilt and unworthiness are prominent in depressive illness of endogenous type. This has long been known, for example Plutarch, in the first century AD, described a person 'He looks on himself as a man whom the gods hate and pursue with their anger . . . "Leave me," says the wretched man, "I am the impious, the accursed, hated of the gods, to suffer my punishment" ' (Zilboorg and Henry, 1941). On the other hand Shepherd (1993) considers that guilt feelings did not feature predominantly in depressive states described in pre-Puritan England. The patient may blame himself for having allowed himself to get into this state of mind. He is full of self-reproach and retribution for all sorts of pecadilloes from the distant past. For all that goes wrong around him he takes personal blame: this may be of delusional intensity. Using a scale for the evaluation of feelings of guilt, it was possible to identify two separate components 'delusional' guilt or shame (experienced in relation to one's actions), and 'affective' guilt (a more general feeling of unworthiness) (Berrios et al., 1992). As well as delusions of guilt and unworthiness, hypochondriacal and nihilistic delusions are relatively common in depression, especially when it occurs in the elderly.

Delusions occur in psychotic depression. It is important to make the distinction between a belief about the state of the world coloured by current mood - 'I feel that I must have done something to my brain as I can't think properly', from an actual delusional belief - 'I can't think at all, it is impossible, my brain is dead'. The former is a metaphorical statement, the latter a belief held with conviction. In practice, there is often a grey area between frank depressive delusions and emotionally laden views of the world.

Table 18.1 shows the frequency of syndromes, however slight, in depressive illness that were recorded quantitatively using a rating scale in 239 men and 260 women (Hamilton, 1989). It is seen that anxiety is a frequent symptom in depressive illness.

Suicidal thoughts
'I feel as though I want to destroy myself. There is no point in going on.' Suicidal ideas, ruminations and impulses are common. Alvarez (1971) has written a detailed study of suicide from a literary point of view. He is concerned with the background
and the reasons for suicide and attempted suicide in many well-known writers, especially poets. He writes about suicide as 'letting go'.

I have to admit that I am a failed suicide... Seneca, the final authority on the subject, pointed out disdainfully that the exits are everywhere: each precipice and river, each branch of each tree, every vein in your body will set you free... Yet despite all that, I never quite made it.

The intertwined threads of artistic creativity, manic-depressive illness and suicide have been explored by Goodwin and Jamison (1990).

Both the muse and madness as the gift of the gods has been a recurring theme from earliest times through such nineteenth-century poets as Browning, Shelley, Coleridge and Byron to the modern American poets, amongst whom there was found to be a very high prevalence of manic-depressive illness, and many suicides. In her enlightening study of manic-depressive illness and the artistic temperament, Touched with Fire, Jamison (1993) demonstrates differential rates for depressive illness and suicide in poets, artists and other writers, and comments upon this.

Extreme mood swings are frequent, with enthusiasm and creativity during elation and stark despair when the poet finds himself or herself lacking in inspiration. Poets and also creative musicians (Schumann, Wolf, Rachmaninov, Tchaikovsky, etc.) show this pattern especially frequently, whilst it is much less common amongst biographers – and presumably writers of textbooks. In the same way that depression may occur without suicide or suicidal ideas, suicide may be carried out without predisposing pathological depressive mood.

Depression is regarded as the final common pathway leading to suicide (Van Heeringen et al., 2000). These authors imply depression the emotion and not the diagnostic category. They consider that psychological, social, and biological aetiological factors, and the increased rates associated with many psychiatric disorders are all mediated through hopelessness resulting in suicidal behaviour. This emotion of hopelessness arises from feeling defeated in some important area of life and feeling closed in with no possible escape or rescue. Suicidal behaviour is then a 'cry of pain', an attempt to escape these feelings of entrapment.

Plans for suicide may not be carried out, solely because of the degree of retardation – occasionally electroconvulsive therapy may lessen retardation after three or four treatments and thereby increase the risk of suicide, because improvement from depression of mood and lowered self-esteem because of guilt feelings has not yet occurred. Death is often welcomed with a sense of relief. A psychotically depressed patient, when offered admission to hospital, accepted with resignation 'I will come in and there you will kill me. It is what I desire.' It is frequently described afterwards by the relatives of suicides that in the days or hours preceding their death they were happier and more tranquil than they had been for a long time.

Homicide of one or more of those close to the patient followed by suicide is a real danger with a small minority of sufferers from depressive illness. A profoundly depressed man felt that life was not worth living, that he had failed completely and the world was intolerable. The only person he cared for was his 5-year-old son and he did not want to condemn him to what he anticipated would be a lifetime of misery. He put his son on the handle-bars of his bicycle and rode over the quay into the harbour intending to kill them both. The boy was drowned but the father was rescued, resuscitated and charged with murder. Subsequently, he responded to treatment for his severe depressive illness.

Depersonalization, which is common in depression, may be manifest as loss of feelings or the ability to feel. This is a milder form of what may progress in severe illness to nihilistic delusions: 'my body has been changed to water', or 'I am dead: I have no feelings and no will'. Depersonalization is described in Chapter 14. When it occurs in healthy people it is noticed but associated with little emotion, but when it is complained of as a symptom, it is described as being extremely unpleasant. The patient usually finds it very difficult to describe the experience but is quite adamant that it is one of the most unpleasant experiences he has ever had.

| Table 18.1 Frequency of symptoms in depressive illness (after Hamilton, 1989) |
|---------------------------------|---------------------------------|
| Symptoms % of subjects Males | Symptoms % of subjects Females |
| Depressed mood 100.0 | Depressed mood 100.0 |
| Loss of interest 99.6 | Loss of interest 98.8 |
| Anxiety, psychic 97.1 | Anxiety, psychic 97.8 |
| Anxiety, somatic 87.4 | Somatic, general 94.2 |
| Insomnia, initial 83.7 | Somatic, somatic 87.3 |
| Suicide 82.0 | Somatic, gastrointestinal 83.5 |
| Somatic, general 82.0 | Suicide 80.4 |
| Somatic, gastrointestinal 80.3 | Insomnia, initial 77.7 |
| Insomnia, delayed 74.1 | Guilt 72.7 |
| Guilt 71.5 | Insomnia, delayed 71.9 |
| Insomnia, middle 71.5 | Weight loss 68.8 |
| Weight loss 69.0 | Agitation 68.1 |
| Agitation 68.1 | Libido 49.5 |
| Libido 59.8 | Retardation 43.5 |
| Retardation 52.3 | Hypochondriasis 33.1 |
| Hypochondriasis 52.3 | Loss of insight 28.0 |
| Loss of insight 28.0 | Paranoid symptoms 25.1 |
| Paranoid symptoms 25.1 | Depersonalization 21.1 |
| Obsessional symptoms 13.3 | Obsessional symptoms 20.7 |
| Depersonalization 10.9 | Paranoid symptoms 13.8 |
| Diurnal variation | Diurnal variation 60.1 |
| worse in morning 61.4 | worse in morning 65.5 |
| worse in evening 30.7 | worse in evening 25.0 |
| worse in afternoon 7.9 | worse in afternoon 9.5 |
SYMPTOMS IN THE MIND

That internal restlessness and Disorder in Man, which has been the Complaint of All Ages was part of the title of James Vere's book (1778) in which restlessness of the soul is associated with instinctual conflict in a way that anticipates Freud's theory of anxiety—the resultant conflict from the opposing forces of the super ego and id. Mood is a variable expression of the self; it may be a transient feeling reactive to a certain situation or it may be a more long-lasting, sustained, inexplicable mood that is regarded as endogenous.

Internal restlessness also describes the emotions of neurotic disorder—anxiety, irritability and the situational fears of phobic state. These, with obsession disorders, are discussed in Chapter 19.

Cyclothymia and related conditions

As well as the major episodes of mania and depression occurring in manic-depressive disorder, for which admission to hospital will often be indicated, there are also recurrent and cyclical conditions with episodes of depression and hypomania of mild to moderate severity which rarely lead to hospitalization (Akiskal and Mallya, 1987); the symptoms are manifested as abnormalities of personality, such as cyclothymia, rather than as symptoms of mood disorder. These conditions are common in the general population. These authors would see such conditions as including contributions from the hyperthymic temperament, subaffective dysthymic temperament, irritable temperament and cyclothymic temperament. There are, therefore, both bipolar spectrum disorders, which are characterized by abrupt biphasic shifts in mood, cognition, behaviour and circadian rhythms.

These conditions are described as cyclothymia (F34.0) in ICD 10 and cyclothymic disorder (301.12) in DSM IV (American Psychiatric Association, 1994). The patient shows mood swings over many years in both directions, that is, during periods of depression and hypomania, but severity does not amount to that seen in manic-depressive illness. Disorder of cyclothymia should always be considered in the differential diagnosis.

Depression and Loss

Any social situation of transition is associated with some disturbance of emotion (Parkes, 1971). Depression is the affect associated with experience of loss. It is not the intention here to enter into theoretical aspects but to discuss the subjective experience. Parkes (1976) has demonstrated how loss of a person, loss of a limb and even loss of a home are stressful in similar ways, and that there is a mental process going on in which the person is 'making real inside the self events which have already occurred in reality outside'. This process is associated with marked psychic pain and unhappiness. An example of depression associated with the threat of loss of a loved object was a taxi driver who owned his own car which was the only thing he valued in life. During an episode of profound depression he polished the taxi to perfection, took it into the garage, connected a pipe to the exhaust of the car, started the engine, and killed himself.

The dysphoric mood associated with the experience of loss is always exacerbated if there is any sense of guilt or self-blame attached to the circumstances of the loss: "if only I had called the doctor in to see Mother earlier, I shall never forgive myself". Byatt (1985) comments about this in relation to crime fiction: 'Detective stories, like the belief in Original Sin, console and comfort men for death, because someone always is responsible for bringing it into the world (of the novel) and all our woe goes out with retribution or atonement. One of the many unpleasant aspects of grief is the need to feel responsible or guilty'.

Grief

The immediate experience of loss is shock and numbness. The suddenly bereaved person may say that he cannot believe that it has happened to him. He just feels numb and empty. He may describe depersonalization feelings. There is a tendency to deny that the loss has happened. A woman was referred to a surgeon for a lump in the breast. At operation the mass was found to be malignant and the breast was amputated. For several days after the operation she was unable to accept that the painful area under the dressing signified the loss of her breast rather than a minor excision.

Following initial shock and denial come the pangs of grief. This is an acute feeling of loss with anxiety prominent as well as grieving—'anxious searching'. The implications of the experience of loss begin to be realized and this may cause the person feelings of anxiety amounting to panic: 'However am I going to cope without him?'. The somatic symptoms of anxiety may be present as well as the psychological.

Three distinct patterns of "morbid grief' have been observed (Lieberman, 1978)

1. Phobic avoidance of persons, places or things related to the deceased, combined with extreme guilt and anger about the deceased and his death
2. A total lack of grieving with anger directed towards others and over-idealization of the deceased
3. Physical illness and recurrent nightmares of the deceased.

These patterns have relevance for treatment using the behavioural method of "forced mourning".

When the experience of loss has been accepted as a reality, depression, the affect appertaining to loss, occurs. The person feels very low and hopeless, perhaps with the lowering of vitality and apathy of depression. He becomes resigned to his situation but sees no way out: 'there is simply no future for me now'. Not surprisingly this state is often associated with suicidal ideas and impulses, and there is an increased mortality from suicide and other causes in the 6 months subsequent to bereavement (Parkes et al., 1969). As the state of grieving is resolved the person gradually overcomes this despairing hopelessness. There is an attitude of mind that results in reorganization and redirection. He gradually makes decisions and carries out activities that demonstrate his emotional and intellectual acceptance of the loss and intention to continue his life as congenially as possible, although still remembering the loss. This stage of 'resolution may be postponed for many years, as with Queen Victoria's grieving for Prince Albert.

Parkes (1976) differentiates between the subjective experience of "external loss and internal change. The external loss is shown by pining for the lost object. Anxiety following loss occurs both in bereaved people and in amputees, and is associated with anxious searching: a bereaved person was walking up and down the street wondering if she would see her husband whom she knew to be dead. In these
circumstances misperception of strangers as being the lost relative may happen. A man whose father had died some long time before, thought he heard his father’s voice in another room and then realized it was his son. People return to places associated with the lost person or keep articles which belonged to them sacrosanct. The pseudohallucinations of bereavement described by Rees (1971; Chapter 7), would seem to be a feature of this anxious searching. These are usually experienced as pleasant and reassuring but may occasionally be a horrific memory of the person when ill and suffering. In a small proportion of amputees phantom limb pain is incapacitating. Parkes considered that this was at least in part psychologically determined and an equivalent of separation anxiety. Confirmatory evidence was the account from a man who said that he only experienced pain when he took his prosthesis off at night, when he put it on again the pain would always disappear. Parkes described similar affective experiences in those compulsorily rehoused from the slums of Boston.

Internal change, with a sense of mutilation, is common to people with different types of loss. Amputees feel themselves to be badly damaged both in their function and in their self-image. Because a man has lost his leg, he will be unable to carry out his previous activities as before and may feel himself to be less of a man. Similarly, the woman with an amputated arm may prefer a cosmetic but useless prosthesis rather than a more functional hook. She may feel the affect to her self-image of a mutilated arm more than the loss of function. Parkes and Napier (1975) stress the social associations of loss in their discussion of prevention and alleviation of the problems resulting from amputation. Widows also describe a feeling of loss within themselves due to their bereavement; there is, of course, often a real loss of status. Those rehoused often described an internal change on moving: ‘something of me went when I left the old home’.

**Helplessness and hopelessness**

Engel (1967) has studied the life setting in which illness develops, observing that the onset of illness often coincides with a time when the patient was assailed with real or threatened loss or separation, and was experiencing great difficulty with coping. Patients have described this affect as ‘discouragement’, ‘despair’, ‘giving up’ or ‘depression’ (Schmale, 1958; Schmale and Iker, 1966). These authors considered that the affect contributes to the emergence of somatic disease when the necessary predisposing factors are present. The different facets of this emotional set have been described as the giving-up given-up complex and five subjective characteristics have been delineated (Engel, 1968).

5 There is a painful remembering of times when his self-esteem and sense of well-being were lowered. This reactivation of memory especially concerns past failures, embarrassments and griefs. Feelings of giving-up may be directed towards an idea (a cherished ambition) or an object, as well as towards a person or even himself.

These affects of helplessness and hopelessness have been described as a psychological disturbance preceding becoming ill (Schmale and Iker, 1966).

Low self-esteem is an important facet of the vulnerability theory in the social origins of depression. Thus, it is not just feelings of loss but feelings of hopelessness that also result in depressive mood: the self is construed as worthless and the world meaningless, and the social situation is such that no amelioration is possible. This is frequently the soil in which situational (neurotic) depression grows.

**Mania**

Mania is a word with a long history. Hare (1981) considers that the early descriptions of intellectual deterioration with excitement were made because of the association with organic deterioration from poor general health during the nineteenth century. As the physical health of the population improved, it was possible to describe separate conditions with different natural histories. However, mania still forms a much higher proportion of affective psychoses occurring peripherally than of affective disorders occurring at other stages of life (Beer and Kendall, 1969).

Mania refers to elation of mood, acceleration of thinking and over-activity. Subjectively, although it may be described as a different state from normal, it is rarely complained of by the patient, as a symptom. A young manic in-patient described his internal state thus: ‘I feel hypersuscetible with experience... I am developing a close secretarial relationship with Camilla Brown (another young patient)... I feel like a rocket with the blue paper lit, standing in a bottle and just ready to take off’.

It has become conventional to refer to all but the most severe cases as suffering from hypomania. This is unfortunate as one does not refer to ‘hypodepression’, and the person using the term hypomania often gives the impression that wrong diagnosis is permissible to a greater extent than if the term mania had been used.

The early stages of mania may be experienced as enjoyable, even ‘wonderful’, and an enormous relief from the depression that preceded it. A patient quoted by Whybrow (1997) put it this way: ‘In the early stages of mania I feel good—about the world and everybody in it. There’s a faster beat; a sense of expectation that my life will be full and exciting.’ For this reason the patient may be reluctant to take medication or to report his condition to his doctor. Later on in manic illness the patient’s experience is usually described as unpleasant and even frightening.

In pure form it is characterized by excessive cheerfulness, rapid speech and association of thought, and over-activity. The speed of thinking and the ready ability to form associations results in rapid and apparently sparkling conversation (see Chapter 9). Puns and clanging associations abound, for example in a case quoted by Bingham (1841).

A fine bold lady, well dressed and well known to the officers of a certain house; a regular mudlark, as they called her, was brought thither by her friends. She was no sooner
SYMPTOMS IN THE MIND

announced than every missile and instrument of attack was carefully removed out of her way. She opened the conference by a familiar address to the physician under whose care she had been before and was going to remain, by saying to him, 'Well, Doctor Morrison,' but I beg pardon, I forgot whom I was speaking to - it is Sir Alexander. Well, Sir A.-, since I had the pleasure of seeing you last, I have been benighted, and you have been knighted.

An excellent ward sister, Sister Boddy, with whom I once had the privilege to work was greeted by a young manic patient as 'Sister Boddy, Sister Anatomy!'

Manic thinking

Exceptional distractibility is shown in the way external events, such as a noise in the street outside, are immediately incorporated into conversation. The rapid association of thought is called flight of ideas (see Chapter 9) and the incessant need to talk and express these ideas pressure of speech. Behavioural changes result from this elevation of mood and acceleration of thinking and activity. Restless activity associated with grandiose schemes is often seen: a patient buried several mattresses in his garden because he felt it would improve the quality of his vegetables by making compost. Manic patients often go on spending sprees well beyond their means, or get involved in sexual affairs in a manner unlike their normal character. A manic patient said 'my thinking feels hot'. Their overactivity and super-abundance of energy involves other people, who find themselves having to cope with the elaborate projects that the manic patient sets in operation. An example of this is the case of folle a deux described by Ropschitz (1957) in Chapter 20.

Usually, the manic patient looks at his world with complete, unshakable and wholly unjustified optimism. However, he is intolerant of authority, when relatives and doctors try to curtail his restless overactivity, showing itself in manic irritability, he feels all his brilliant ideas are frustrated by the lack of vision of everyone else. He has no insight into his illness but feels better, more alert, more healthy than ever before. He believes his thinking to be greatly improved and feels other people are dull and slow in comparison.

Kraepelin considered that there were three fundamental components to the symptomatology of manic-depressive psychosis: level of mood, psychic activity and motor activity. Characteristically, in mania these functions are elevated whilst in endogenous depression (bipolar affective disorder, current episode depression in ICD 10), they are depressed. In agitated depression the mood is depressed whilst activity is increased and the patient moves restlessly and purposelessly about, wringing his or her hands. Thinking may be increased showing what Mapother and Lewis (1937) have called 'a ceaseless roundabout of painful thought'. These changes are represented diagrammatically in Figure 18.4. In affective disorders restlessness and retardation of body or mind may occur in the same patient and sometimes, rather surprisingly, at the same time. It was an observation of great significance by Kraepelin to demonstrate how these apparent polar opposites of mental disorder should in fact be part of the same condition. Mixed affective states are those in which features of mania and depression are seen during the same episode of illness. Thus the features of Kraepelin's triad can be mixed in any combination in the affective disorders.

A Third Functional Psychosis?

Not uncommonly, the mental state of a patient is clearly psychotic, but not obviously revealing either manic-depressive psychosis or schizophrenia. Quite often such conditions show features of both major psychoses: more rarely the presentation is quite different. A variety of different terms have been used to describe a third functional psychosis (Table 18.2). Reference is only made here to some of the syndromes described. For more information the reader is recommended to consult a textbook of psychiatry, for instance Gelder et al. (2000), especially 'Schizoaffective and schizotypal disorders' (Tsuang et al., 2000). The term schizoaffective psychosis, introduced by Kasanin in 1933, is used in several quite different ways: delusions, hallucinations and thought disorder are described. The International Classification of Diseases, ICD 10 (World Health Organization, 1992), places them diagnostically within schizophrenia. However, Procacci (1976), in his thorough survey of the literature, emphasizes the scant resemblance of a well-defined minority to nuclear schizophrenia, and sees them as more related to affective psychoses. He focuses on the group of young, acute onset patients with good premorbid history, presence of external precipitants, affective features and heredity coupled with positive criteria for Bleulerian schizophrenia; this dilemma demonstrates the over-inclusiveness of Bleuler's description of schizophrenia (1911).

Not infrequently, as has been demonstrated by Carlson and Goodwin (1973), in the acute phase of a bipolar affective psychosis a patient will show symptoms suggestive of schizophrenia, such as thought disorder, bizarre behaviour, ideas of reference, delusions and hallucinations. It has been described how a proportion of people who have had schizophrenic features in one episode develop manic features of an affective psychosis in a subsequent episode (Lipkin et al., 1970). Some patients with remitting schizophrenic illnesses subsequently present with clear-cut affective disorders (Sheildrick et al., 1977). There are also some who are initially diagnosed manic-depressive and subsequently reclassified schizophrenic (Hoch and Richlin, 1941).

<table>
<thead>
<tr>
<th>Mood</th>
<th>Psychic activity</th>
<th>Motor activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>Retardation</td>
<td>Motor retardation</td>
</tr>
<tr>
<td>Elation, hilarity</td>
<td>Loss of thoughts</td>
<td>Overactivity, restlessness</td>
</tr>
<tr>
<td>Flight of ideas</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 18.4 Kraepelin's triad: the components of manic-depressive psychosis
The psychosis seems to be somewhere between schizophrenic and manic–depressive psychosis and the doctor is not sure which diagnosis is correct.

It is recommended that diagnosis should be made only after careful elucidation of the phenomenological symptoms.

Helmen and Hippius (1967) found that half a sample of 120 admissions for schizophrenia also manifested depressive symptoms at the time of admission to hospital; and this finding has been subsequently confirmed. The possible explanations for this association of schizophrenic and depressive symptoms have been discussed by Hirsch (1986); he considers that depressive symptoms are an integral part of the schizophrenic syndrome (Knights and Hirsch, 1981).

### Table 18.2 The third functional psychosis

<table>
<thead>
<tr>
<th>State</th>
<th>Author</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophreniform states</td>
<td>Langfeldt</td>
<td>Schizophrenics who recover, acute onset, psychogenic precipitants, depressive symptoms and family history, confusion during acute episode, extroversion of personality, pyknic body build.</td>
</tr>
<tr>
<td>Schizo-affective psychosis</td>
<td>Kasanin</td>
<td>Good premorbid function, mixed schizophrenic and affective features, recovery after a few months.</td>
</tr>
<tr>
<td>Dementia praecox</td>
<td>Kraepelin</td>
<td>Obsolete term.</td>
</tr>
<tr>
<td>Dementia praecox with cyclical course</td>
<td>Dunton</td>
<td>Obsolete term.</td>
</tr>
<tr>
<td>Catatonic syndrome</td>
<td>Kiley</td>
<td>Acute onset of catatonic symptoms, no premorbid personality changes, good prognosis.</td>
</tr>
<tr>
<td>Benign stupor</td>
<td>Hoch</td>
<td>Aged 15–25, acute onset of catatonic symptoms, inactivity, intellectual interference, affectless, negation.</td>
</tr>
<tr>
<td>Recovered schizophrenics</td>
<td>Kant</td>
<td>Acute onset, clouding of consciousness, extroversion, pyknic build, affective features and family history, young, often prior diagnosis of affective psychosis.</td>
</tr>
</tbody>
</table>

Kuriansky and colleagues (1974) demonstrated that there was a shift in diagnosis in the USA from 1932 to 1956 towards schizophrenia. Baldessarini (1970) showed a shift, that is, diagnosis of affective psychoses from 1960 to 1968, and he linked this with the introduction of the successful treatment of mania and schizo-affective psychosis.

Slater and Roth (1969) regarded the controversy over schizo-affective psychosis as largely unnecessary. They considered that careful diagnosis of either schizophrenia or manic–depressive psychosis will include most cases. This dictum still holds good in the main, although there are difficult cases. The term schizo-affective psychosis has been used loosely to imply any of the following four situations:

1. a patient has previously had an attack of schizophrenia with first-rank symptoms and now has a quite definite episode of affective psychosis
2. a patient who had a previous manic–depressive illness, now has a definite schizophrenic illness
3. a patient appeared to be suffering from schizophrenia and manic–depressive psychosis simultaneously, and both illnesses can be clearly delineated.
AFFECT AND EMOTIONAL DISORDERS


The management of bipolar disorder in general practice

Philip B Mitchell, Jillian R Ball, James A Best, Bronwyn M Gould, Gin S Malhi, Geoffrey J Riley and Ian G Wilson

It has been estimated that 1 in 200 Australians experiences an episode of bipolar disorder in any 12-month period, and that lifetime prevalence is between 1% and 4%. The Australian National Study of Low Prevalence (Psychotic) Disorders included an analysis of 112 people meeting the criteria for bipolar disorder of the International classification of diseases, 10th revision (ICD-10). Most were already in treatment, and might therefore reflect the more severe or acute segment within the continuum of severity.

The mean age of onset was 25 years for men and 26 years for women. In addition to episodes of mania, 79% had experienced at least one clinically significant episode of depression. A large proportion (86%) reported experiencing delusions at some time in their lives.

The Australian National Study of Low Prevalence (Psychotic) Disorders included an analysis of 112 people meeting the criteria for bipolar disorder of the International classification of diseases, 10th revision (ICD-10). Most were already in treatment, and might therefore reflect the more severe or acute segment within the continuum of severity.

Difficulty with relationships was common. Just over half the men and a quarter of the women were single and had never been in a long-term relationship, and 64% of the men and 42% of the women did not have children. Half the men and a third of the women lived alone. Many had achieved well educationally, but their work performance was impaired: only 30% of both men and women with bipolar disorder had no formal secondary school qualifications, but 79% were receiving a pension or benefit, and 67% of those seeking work were unemployed at the time of interview; 9.8% were classified as severely disabled.

Nature and course of illness

- Bipolar I disorder is characterised by the occurrence of at least one lifetime episode of mania and, usually, episodes of depression.
- Bipolar II disorder is characterised by episodes of both hypomania and depression, but no manic episodes.
- Recognised patterns of illness also include mixed states and rapid cycling between depression and mania.

Depression is often the predominant mood and has been associated with the greatest burden of disability. One study found that patients with bipolar I disorder experienced 32% of their weeks of follow-up in depression and 9% in mania or hypomania. For those with bipolar II disorder, 50% of their follow-up period occurred in depression but only 1% in hypomania.

Suboptimal function between discrete bipolar episodes, characterised by symptoms such as mild anxiety or depression, is common and tends to be unrecognised. The suicide rate in people with bipolar disorder is about 15 times that of the general population, and 80% of suicides occur during episodes of depression. At least 25% of patients will attempt suicide and 10%–20% will complete suicide. Comorbid conditions including anxiety disorders (52% of patients in one Australian study) and substance misuse (39%) are prevalent.

Role of general practitioners

GPs are well placed to coordinate the care of patients with bipolar disorder as they continue to provide other aspects of general medical care and develop an understanding of the patient’s circumstances and progress.
Background and evidence base for recommendations
The original concept for these recommendations arose out of discussions between one of us (P B M) and Eli Lilly Australia. They were prepared by an expert working group with expertise in the diagnosis and management of bipolar disorder in both the general practice and psychiatric settings. The disciplines represented include general practice, psychiatry, psychology and medical education. This outline for the management of bipolar disorder in general practice has been formally endorsed by the Royal Australian College of General Practitioners.

Most of the pharmacological and psychological treatments recommended are supported by National Health and Medical Research Council (NHMRC) levels of evidence I or II; that is, there is evidence from a systematic review of all relevant randomised controlled trials (level I) or from at least one correctly designed randomised controlled trial (level II). The evidence is derived from patients with bipolar I disorder in inpatient or outpatient psychiatric treatment settings. Currently, there are no controlled trials of treatments for bipolar disorder in primary care.

More than a third admitted symptoms such as reckless behaviour, spending excessively and increased sexual interest or activity.6 The age of the patient influences the differential diagnosis. In younger patients, conditions such as attention deficit hyperactivity disorder and conduct disorder need to be considered. In patients older than 40 years — an age when initial presentation in the manic phase of bipolar disorder is relatively uncommon — possible organic causes should be addressed.

Some “clues” to the presence of bipolar disorder are summarised in Box 1. Note that longitudinal monitoring is often necessary to make or refine the diagnosis.

Management of bipolar disorder

Medication
The main tasks for GPs in managing medication for patients with bipolar disorders are:
• monitoring the efficacy and side effects of maintenance therapy;
• in consultation with a psychiatrist, implementing treatment during acute episodes of mania or depression; and
• supporting medication adherence.

Clinical practice guidelines released by the Royal Australian and New Zealand College of Psychiatrists in 2004 provide an authoritative guide to treatment.7 Some principles of treatment for bipolar disorder are listed in Box 2, and recommendations for laboratory monitoring during maintenance therapy are summarised in Box 3. Most research has been conducted in bipolar I disorder and extrapolated to bipolar II disorder when required: there is relatively little direct evidence on medications for bipolar II disorder.

While several guidelines on bipolar disorder have been published internationally, there is considerable unanimity between these. The major distinctions appear to be stronger recommendations for use of lithium in the Australian and European guidelines, with more emphasis on valproate in the United States.8 Furthermore, US guidelines strongly support the use of mood stabilisers alone at an early stage in the treatment of bipolar depression.

The term “mood stabiliser” is used to describe medications that are effective in both acute and maintenance phases of therapy. “Traditional” mood stabilisers are lithium, valproate and carbamazepine, but there is evidence that atypical antipsychotics and the newer anticonvulsants are also effective in at least some phases of bipolar disorder.

Antidepressants in bipolar disorder
The role of antidepressants in bipolar disorder is controversial. A systematic review concluded that antidepressants were effective in symptom management during at least some acute episodes of depression in bipolar disorder. However, the potential to trigger rapid cycling and other adverse effects is important.

Duration of symptoms
The criteria of the Diagnostic and statistical manual of mental disorders (DSM-IV) require that symptoms of hypomania are present for at least 4 days and symptoms of mania are present for 7 days before a diagnosis is made. However, these thresholds are somewhat arbitrary.

1 Clues to bipolar disorder
Symptoms and signs of mania and hypomania can include the following types of behaviour which are out of character for the individual:
• feeling energised and “wired”
• excessively seeking stimulation
• overly driven in pursuit of goals
• needing less sleep
• irritable if stopped from carrying out ideas
• disinhibited and flirtatious
• offensive or insensitive to the needs of others
• spending money in an unusual manner or inappropriately
• indiscreet and disregarding social boundaries
• having poor self-regulation
• making excessively creative and grandiose plans
• having difficulty discussing issues rationally or maturely
• reporting enhanced sensory experiences

2 Principles of medication for bipolar disorder*

Acute manic episodes
• Use a mood stabiliser (lithium, valproate, carbamazepine or an atypical antipsychotic, such as olanzapine, aripiprazole, quetiapine or risperidone) for elevated mood
• Also use an antipsychotic (if not already chosen as a mood stabiliser), a benzodiazepine, or a combination of an antipsychotic and a benzodiazepine, to calm or sedate the mood until the mood stabiliser takes effect (about 1 week)

Acute bipolar depression
• In de novo depression, in the absence of pre-existing mood stabiliser therapy, use a mood stabiliser either alone or in combination with an antidepressant. Mood stabilisers with proven antidepressant effects in bipolar depression include lithium, lamotrigine, quetiapine and olanzapine
• In breakthrough depression during mood stabiliser therapy, optimise the mood stabiliser by ensuring compliance and checking serum levels. If this fails, add an antidepressant or a second mood stabiliser. Selective serotonin reuptake inhibitors and venlafaxine are generally preferred, although monoamine oxidase inhibitors and tricyclic antidepressants are sometimes necessary

Prophylaxis
• Consider lithium, valproate, olanzapine, lamotrigine (the latter agent is most effective for prevention of bipolar depressive episodes) or carbamazepine

* Adapted from the clinical practice guidelines of the Royal Australian and New Zealand College of Psychiatrists, 2004.7
the short-term treatment of bipolar depression and that switching to mania was not a common early complication of treatment. However, another recent study reported the common occurrence of switching to mania, which was observed in 19% of acute and 37% of long-term antidepressant courses, but the study was limited by the lack of a placebo control group.

Patients who stopped taking antidepressants within 6 months of remission from bipolar depression were more likely to relapse within the following year than those who continued treatment. This suggests that for many patients continuation of antidepressants beyond the usual 2–3 months recommended in bipolar depression may be preferable.

### Initiating therapy

Ideally, patients should be reviewed by a psychiatrist before a diagnosis of bipolar disorder is confirmed and therapy commenced. However, access to specialist services may be delayed, particularly when the patient's symptoms are not severe. Telephone advice from specialist services can assist GPs in these situations, and guide the initiation of appropriate treatment. More frequent review of the patient can also assist in monitoring progress and checking that symptoms, risks and disability are not progressing more rapidly than anticipated.

### Medication adherence

Poor treatment adherence in patients with bipolar disorder is common. GPs can provide verbal and written information on the advantages and disadvantages of prescribed medications, and encourage patients to give their own views and discuss their experiences. Symptom history can be used to review the effects of medication, identifying the costs and benefits of adherence, and highlighting the active role of the patient in the analysis. Patients should feel confident about discussing the pros and cons of medication and their own concerns.

### Psychological therapies

There is growing evidence for the benefits of psychological therapies in bipolar disorder. Psychological therapies include:

- education about the condition and its treatment;
- basic cognitive behavioural techniques (eg, identifying triggers and planning how to minimise or avoid them; accurately labelling emotions; identifying thoughts and reframing them into more positive rational responses; and dealing with adjustment/self-esteem issues and long-term vulnerabilities);
- interpersonal and social rhythms therapy teaches patients to be more effective in handling relationships and make graded lifestyle changes to increase stability, and highlights the importance of routine and sleep;
- supportive psychotherapy is particularly useful in identifying interpersonal triggers that affect the patient's mood. It is most useful in the maintenance stage of treatment, once the skills of cognitive behavioural therapy have been learned; and
- exercise, yoga, relaxation therapy and similar “mind–body” interventions, individualised to the needs and lifestyle of the patient, may also be beneficial in the maintenance stage.

### Indications for referral

Indications for referral to specialist services are given in Box 4.

### Relapse and emergency care

#### Acute episodes

An episode of acute mania is a medical emergency. Patients have the capacity to destroy their reputations, relationships and finances within hours or days. Insight and judgement are usually impaired early, even in the absence of delusions, and involuntary hospitalisation is frequently required to protect the patient. However, the decision to admit may be traumatic for the patient and family members, all of whom will need support.

If outpatient treatment occurs, it is essential to monitor risky behaviour, such as financial indiscretion or potential harm to others from, for example, hazardous driving. A financial power of attorney may be necessary. Outpatient attendance is often erratic, and a legally enforceable community treatment order may be required.

It has been said that patients with acute mania are “always worse than they seem”. An apparently reasonable level of function during a brief assessment may mask more serious dysfunction. Reports from family and friends should be taken seriously, but interpreted with an understanding of the patient’s normal function and the nature of these relationships.

Similarly, active depression is associated with a high risk of suicide and self-harm. Risk management, particularly the recognition of suicidality, is a crucial responsibility of the treating doctor.
Relapse profile

It may be possible to develop a “relapse profile” for individual patients and list how the patient should respond. Common triggers for hypomanic or manic episodes are changes to everyday rhythms (eg, sleeping and eating), and stressful life events.

Possible early warning signs include destructive or impulsive behaviour after being sleepless or irritable, looking haggard, speaking in a caustic manner, telephoning friends indiscriminately regardless of the time, stopping medications, and impulsive, self-destructive threats and gestures.

Strategies to offer the patient when early warning signs of mania occur include the following:

- establish a regular routine for eating and sleeping;
- spend nights in your bedroom even if you are not sleeping — lie down and rest as much as you can;
- prioritise and reduce the number of tasks you are involved in;
- modify excessive behaviour — slow down;
- engage in calming activities and be aware of how you are thinking, feeling and behaving;
- carefully follow through the consequences of your actions — consider the costs and benefits;
- delay impulsive actions — if it is still a good idea in a few days time, it might really be a good idea;
- spend time on your own to reduce stimulation, for example by avoiding crowds, busy shops, intense movies and parties;
- find a quiet, restful place to spend your time;
- keep a diary of your moods and reactions;
- reframe your overly inflated thoughts as symptoms;
- recognise if you are getting into destructive situations;
- talk to someone you can trust;
- avoid drinking tea, coffee, cola or other drinks that contain caffeine;
- avoid alcohol, marijuana or other drugs; and
- see a doctor to review your medications and current state.

Possible medical and psychological responses to signs of relapse include:

- intensify psychological therapies, including stress reduction, normalising sleep patterns;
- adjust medication, for example adding a benzodiazepine or antipsychotic; and
- advise patients to minimise sleep disruption and other stressors and triggers.

The family

Bipolar disorder can be extremely taxing for the family and carers of the patient. Families need and want information and education about the illness, as well as continuing support during times of crisis. They can become active participants in elements of treatment such as encouraging appropriate medication, regulating lifestyle and monitoring the patient for signs of relapse (Box 5).

Insight, consent and confidentiality

Loss of insight during acute episodes of bipolar illness poses particular problems for families and carers, as patients may deny their illness and resist treatment. Treating doctors must balance the need to respect patients’ privacy, while fulfilling their obligation to provide adequate care.

Decisions may need to be made about the legal competence of patients and the point at which action should be taken to declare them incompetent and a danger to themselves and others. “Dangers” are not always physical, and can include damage to reputation, income and relationships. Dependants normally under the care of the patient may also be at risk, and treating doctors should be aware of their legal and ethical responsibilities to protect children from harm.

Support for patients and families

Although there is not a single national bipolar disorder support organisation for patients and families, a range of state-based services and organisations exist (Box 6).
6 Resources for bipolar disorder

Services directory

Treatment guidelines

Books for patients, families and carers

Websites
beyondblue: the national depression initiative — http://www.beyondblue.org.au
Black Dog Institute — http://www.blackdoginstitute.org.au
SANE Australia — http://www.sane.org
ORYGEN Youth Health — http://www.orygen.org.au
National Institute of Mental Health (USA) — http://www.nimh.nih.gov
Mental Health Council of Australia — http://www.mhca.org.au

Conclusions
Bipolar disorder is a challenging illness for patients, families and carers, as well as for health care professionals. Increased understanding of the disorder can facilitate early and accurate diagnosis, effective short-term and long-term pharmacological and psychological treatment, and the development of effective support mechanisms.

GPs have a central role in facilitating diagnosis, accessing specialist care, and providing continuing monitoring and support.

Acknowledgements
The contributions of Associate Professor Kay Wilhelm, Mr Tony James, Ms Sarah Reed and Ms Laila Chaama are gratefully acknowledged.

Competing interests
The development of these guidelines was supported by an untied educational grant from Eli Lilly Australia. Eli Lilly facilitated the meetings of the group and provided administrative support. Eli Lilly had no editorial role in the content of this document. The Chair of the expert working group (Professor Philip Mitchell) and other members of the working group received no remuneration, either directly or indirectly, for involvement in this task. In the past 3 years, Philip Mitchell has served on an advisory board for Eli Lilly Australia and has received honoraria for lectures or consultations from some of the manufacturers of compounds detailed in these guidelines including AstraZeneca, Eli Lilly, GlaxoSmithKline and Janssen-Cilag.

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References
Anxiety Disorders


6.4 ANXIETY DISORDERS

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Anxiety is a normal response to threatening situations. It is useful in that it can alert to danger and provide the means to take the necessary action. At lower levels, anxiety can improve performance and this alerting function is also useful. Anxiety disorders however, are not the same as being too anxious. Anxiety disorders are characterised by specific fears, ideas that symptoms and events that would be of concern to others, might prove catastrophic, result in excessive and unreasonable levels of anxiety in response to these specific events or circumstances. They affect a significant proportion of the population (1) and cause significant distress and disruption to functioning (2, 3).

Although recent decades have seen an accumulation of research into the nature, cause and treatment of anxiety disorders, there is no widespread agreement in regard to the more prominent causal factors. Deficit models of anxiety disorders draw upon findings of neurobiological and neuropsychological differences between persons with anxiety disorders and normal controls (e.g. 4). More specifically, causation models which focus on neurobiological deficits rely primarily on evidence of the effectiveness of drug therapy for the anxiety disorders; in particular the efficacy of the SSRIs has been used to support the involvement of the serotonergic system in panic disorder, social phobia and obsessive compulsive disorder. This raises a number of questions. Could neurobiological and neuropsychological differences be merely correlates of normal cognitive processes? Furthermore, can the differences observed adequately explain the specific phenomenology of the disorders in question? Finally, how valid is the use of treatment response as evidence of causation?

There is growing empirical support for models of anxiety that give prominence to psychological factors, in particular diathesis-stress models which specify a role for both dispositional and environmental factors. Certainly the growing support for genetic factors in the aetiology of anxiety disorders (5) is consistent with the role of a trait vulnerability factor that is common across the anxiety disorders. Andrews, Stewart, Morris-Yates, Holt & Henderson (6), found that receiving any anxiety or depressive disorder diagnosis is significantly associated with a high neuroticism score, and that the neuroticism score increases with the number of additional diagnoses satisfied. This finding was taken to support the concept of a general neurotic syndrome which depends, in part, on the presence of the predisposing personality factor of neuroticism. Other evidence in support of this general neurotic syndrome was that no significant pattern comorbidity or variation in scores on predisposing factors was evident between the diagnostic groups.

Another similar model, the tripartite model of anxiety disorders, depressive disorders and neuroticism (7) is based on numerous studies that report a significant relationship between neuroticism and the symptoms and diagnosis of anxiety disorders. The tripartite model allows for the differentiation of anxiety and depressive disorders on the basis of additional distinctive features: the physiological arousal that is specific to anxiety disorders and the low positive affectivity (or low extraversion) that is specific to depressive disorders. Barlow (8) has also hypothesised that anxiety and depressive disorders share a
common diathesis (or biological vulnerability) but differ on a number of dimensions that are critical to each specific disorder (such as focus of attention, or degree of psychological vulnerability to unpredictable or uncontrollable events). What is the status of these models given the lack of prospective studies to demonstrate that a vulnerability factor, such as neuroticism, antedates the development of disorder?

**Panic Disorder and Agoraphobia**

A 33 year old woman presents to the emergency department of her local general hospital. She is in a high state of distress and reports palpitations, choking sensations, shortness of breath, a tightness across her chest, and a feeling that she was about to faint or have a heart attack. After having been calmed by medical staff, she underwent investigations including an electrocardiogram, which revealed no abnormalities.

Panic disorder is characterised by sudden attacks of fear or anxiety accompanied by the expectation that loss of control, collapse or death will follow. What further investigations or information would be necessary to confirm a diagnosis of Panic Disorder in this case?

The following features were noted from the woman's history. Over the past 6 years, the woman has attended an emergency department on many occasions with similar presenting complaints, believing she was suffering a heart attack. She also regularly visits her local medical practitioner, where she gets temporary relief by the reassurance that she has no heart disease. However, over the last few months these attacks have become more frequent (at least 2 a week). She clearly remembers her first attack, which took her by surprise while on a long-weekend in the country following a particularly stressful time at work. Since her first attack she has found it impossible to travel to places where medical help would not be available. She now makes sure that she always knows the nearest medical centre or emergency department, and is never without her mobile phone.

Agoraphobic avoidance may develop when, in order to prevent these feared outcomes, the sufferer avoids places in which escape would be difficult or help not available. In the case above, the avoidance of places where medical help might not be available has grown out of the belief that the sensations experienced are signs of a life threatening heart attack.

Catastrophic misinterpretations of bodily sensations lie at the heart of current cognitive models of the development and maintenance of panic disorder (9). The notion of anxiety sensitivity, as a trait construct, was developed to explain why not everyone who is exposed to bodily sensations interpret them catastrophically and develop panic disorder (10). How might the catastrophic cognitions about having a heart attack have developed in the case vignette? Are the constructs of anxiety sensitivity and trait anxiety sufficiently distinct for the concept of anxiety sensitivity to be useful in understanding the aetiology or maintenance of panic disorder? (11, 12).

Following her most recent attendance at the emergency department she was referred back to her local GP who commenced her on alprazolam 1mg/day. In the following weeks she did not experience a panic attack, but still could not travel without her mobile phone and felt apprehensive while not in the vicinity of a hospital. She also reported increasing concerns about her need to rely on long term medications to deal with her anxiety.

The use of high potency benzodiazepines has been proposed for panic disorder (13) but this medication can cause sedation and potential dependency and, upon withdrawal, is associated with significant relapse of symptoms for 70-90% of patients (14). What is the role of benzodiazepines in the treatment of panic disorder, and can they be used as an adjunct to Cognitive Behavioural Therapy?
What management issues are remaining in this case? What would need to change for this woman to have overcome her panic disorder? In terms of treatment, there is growing support for the efficacy of cognitive behavioural treatments (15). What are the essential components of CBT treatment for this disorder? How would you apply the specific cognitive behavioural strategies in the case described?

Social Phobia

A 24 year old university student visits his local doctor with a complaint of anxiety about attending lectures and presenting at tutorials. The symptoms of pounding heart, shaking, blushing, nausea and difficulty concentrating had caused him to avoid these situations, to the extent that he now faces possible failure in most subjects. When questioned, he worried that he might behave in a way that would be foolish or embarrassing, and that others in his classes would think him stupid. He also worries that others would notice his anxiety, seeing him shake or blush, and think that he was weird.

The essential feature of social phobia is a fear of embarrassment or fear of negative evaluation which leads to the avoidance of situations where such scrutiny may occur. In DSM-IV there is provision for the diagnosis of a generalised subtype of social phobia in which fears are related to most social situations.

When asked about other difficult situations, the patients admitted that he also experienced similar anxiety in most social situations and in fact any other situation where he might be open to the scrutiny of others. He was extremely uncomfortable sitting in cafes or restaurants, or travelling on buses or trains. He attended parties only with the help of a few drinks beforehand to alleviate his anxiety.

The above vignette typifies generalised social phobia, as anxiety appears to occur in any situation where the patient may be open to the scrutiny of others. One issue for social phobia, specifically the generalised subtype, is the significant comorbidity and overlap in clinical features with avoidant personality disorder (APD). What additional features would need to be present for the patient to meet criteria for APD? Are social phobia and APD different disorders or do they lie on a continuum? Most studies comparing the two disorders across a number of domains have concluded that APD appears to be a more severe variant of generalised social phobia (16-18). However most, but not all, patients with a diagnosis of APD have a diagnosis of social phobia. Should individuals with APD without comorbid social phobia, be considered a sub-group distinct from individuals who have both diagnoses? What implications does this comorbidity have for treatment?

The patients history revealed that the difficulties with social anxiety had begun in adolescence and had steadily got worse. He describes a normal and happy childhood, not particularly shy or anxious until high school. He had a close group of friends at school with whom he still keeps in contact, and he currently lives with his girlfriend in a share house with two others. He reported that he likes people and wanted to feel more comfortable in social situations so he could widen his social network. Apart from his social anxiety, he felt good about himself and his academic performance at university. He did not anticipate rejection or humiliation in most situations.

These features indicate that he is unlikely to meet criteria for an avoidant personality disorder.

In some patients, social avoidance may result from an underlying paranoid personality style in which the main concern is the expectation of harm from others. Questioning the patients beliefs about others will help to determine if this is the case.
For example, the patient was asked by the clinician:

"So what happens when you're sitting in a tutorial?"
"I feel like everybody's watching me, seeing me shake. When I see them laughing I think that they're poking fun of me, criticising me, saying he's really strange"
"Do you really think they are?"
"Well I don't really know, although it feels like it at the time, I mean, that's what I think at the time"
"So when you think about it now, what do you think is really going on?"
"I guess it's not really true, they all seem like nice people, there's no reason to believe they don't like me"

Questioning will also establish if the beliefs are of delusional intensity, or over valued ideas. In what ways can irrational beliefs, over-valued ideas and delusions be differentiated?

Cognitive behavioural therapy is the treatment of choice for social phobia (15). Fear of negative evaluation is an important feature of social phobia, and it has been suggested that the disorder might be resistant to treatment that does not specifically address this cognitive element (19). So how then can medication be an effective treatment for social phobia? To date there is little evidence to support the increased efficacy of this medication over CBT, nor to suggest that the use of this medication as an adjunct to CBT offers any advantage (15). While anxiolytic drugs may reduce both state and anticipatory anxiety, if they do little to reduce the core fears specific to each of the anxiety disorders, what role do they play in the treatment of these disorders?

**Generalised Anxiety Disorder**

A 30 year old woman presents with long-standing symptoms of generalised anxiety which had worsened in previous months. She complained of pounding heart, shaking, breathlessness, and of feeling tense, irritable and "on edge" most of the time. She worried a lot about her work as a secondary school teacher, in particular worrying that she was a poor teacher, used the wrong methods, and that sooner or later her students would fail, hence showing up her incompetence. Worry about responsibility for her students would often keep her awake at night, particularly at assessment times or before parent-teacher meetings. In the past two months, anxiety had interfered with her ability to prepare lessons, as she would procrastinate in order to avoid the anxiety involved in the preparation and also to avoid the failure the task would inevitably bring about. She also worried about her family, fearing that her husband or children would develop a serious illness. She would present her primary school-aged children at the doctors weekly for minor complaints, as this was the best way she had found to stop the worry.

Generalised anxiety disorder (GAD) is characterised by generalised and persistent feelings of anxiety which are driven by worry. Since DSM-III-R this worry has been the defining feature of GAD. The worry is out of proportion to the feared event, pervasive, difficult for the individual to control, and the content usually covers several domains.

She described herself as having always been a worrier and could not recall a time in her life where there had not been some degree of anxiety. She would state that if there was no prominent worry in her thoughts, she would search for something to worry about. She feared that if she did not continue to worry about her children's health, then they might be more likely to develop a serious illness.
The apparent need and conscious decision to worry is a feature of many patients with GAD. This observation has led some researchers to hypothesise that worry may have a reinforcing effect through its effect on attentional processes in anxiety (20). Attention has also been drawn to the trait-like nature of GAD particularly in the light of its insidious onset, its stability across time, and the lack of a specific focus to the anxiety (20).

The patient was able to gain a degree of control over her worry and procrastination at work following a course of cognitive behavioural treatment. She had also been able to stop visiting her GP for reassurance about her children’s health. However, several months later she represented with symptoms consistent with a major depressive episode. At this time, the fears about her family’s health and beliefs about her lack of competence at work re-emerged.

Not only have questions been raised as to whether GAD represents anything more than high trait anxiety, but also whether the disorder can be clearly differentiated from depressive disorders. With the significant overlap in clinical features between GAD and depression/dysthymia, the high rate of comorbidity, and evidence of shared genetic factors (21), should GAD be regarded as a distinct and separate disorder?

Obsessive Compulsive Disorder

The central feature of Obsessive Compulsive Disorder (OCD) is the experience of persistent, unwanted and intrusive thoughts, images or impulses (obsessions) of harm to self or others, blasphemy, inappropriate sexual thoughts, or contamination. Sufferers generally attempt to control their fear by engaging in compulsions, such as washing or checking. For example, washing to avoid contamination follows thoughts about possible contamination.

A 35 year old man presents with compulsive rituals involving checking. He spends two to three hours each day checking that electrical appliances are switched off, and doors and windows are locked. He believes that if he does not engage in this behaviour a fire might start, someone might get electrocuted, or someone will break into the house. He knows that these thoughts are ridiculous but is unable to stop the behaviour. The thought of something terrible comes into his head and he feels the need to continue to check things over and over again.

As with GAD, there is debate as to how OCD should be classified. This has centred on whether OCD belongs to the grouping of anxiety disorders and is within the ‘general neurotic spectrum’ or whether OCD is better related to a range of other disorders (including body dysmorphic disorder, hypochondriasis, dieting disorders, trichotillomania, impulse control disorders, Tourette’s disorder and some monosymptomatic delusional disorders) which share certain phenomenological features, have similar course of illness, comorbidity, family history patterns, and show similar treatment responses (22). Do the shared characteristics of these disorders warrant their grouping into so-called ‘OCD spectrum disorders’? Are there other disorders that could be similarly classified? What implications does the way OCD is classified have for our understanding of the disorder?

In respect to aetiology, specific vulnerability factors have been proposed for OCD (23, 24). Salkovskis’s model proposes that while intrusive thoughts are common among the general population, the difference in OCD is the way in which the thoughts are interpreted as an indication that the individual may actually be responsible for harm or its prevention. Therefore, it is the interpretation of the obsessive thoughts, rather than the thoughts themselves, that cause anxiety or discomfort and cause the individual to engage in ‘neutralising’ responses in an effort to prevent harm or avoid responsibility. Such responses include compulsive behaviours, cognitive rituals, avoidance, or reassurance seeking. These neutralising responses in turn reduce the sense of responsibility, but prevent the natural extinction of the anxiety and increase the salience and frequency of the initial intrusive thoughts. Central to Salkovskis’s model is the activation of pre-existing beliefs of excessive responsibility when the individual experiences the intrusive thoughts.
A 25 year old woman presents with obsessional fears of contamination and reports that she spends several hours each day washing her hands following contact with ‘contaminated’ objects. She recognises that these concerns are unreasonable but states that she feels anxious and uncomfortable if she feels that she has not cleaned or washed her hands sufficiently. Despite thorough questioning, she does not report that any harm would come to herself or those around her if she did not clean or wash, but that she is driven by a sense of urgency to complete her cleaning routine.

Can Salkovskis’s model account for compulsive washers who present as above - those who do not fear the consequences of contamination per se, but who experience significant distress when in contact with the contaminant? (25).

Treatment of OCD involves exposure to situations that produce the urge to perform a ‘neutralising’ response to obsessive thoughts, and prevention of the ‘neutralising response’ (15). How might this treatment, ‘exposure and response prevention’ work?

Post-Traumatic Stress Disorder

Distressing and lasting psychological responses to trauma have been described throughout history, but post-traumatic stress disorder (PTSD) was first recognised in the psychiatric nomenclature in 1980 (26). Critical to the diagnosis of PTSD is the occurrence of a traumatic event followed by the persistent re-experiencing of the event in the form of intrusive recollections or dreams, acting or feeling as if the event were recurring, and the experience of intense psychological distress or physiological reactivity when exposed to stimuli which trigger memory of the event. Avoidance of these stimuli, a pervasive numbing of responsiveness and increased arousal are also key features of the disorder.

A 27 year old man presents to his doctor 6 months after having lost his house in a bushfire. He, his wife, and children managed to escape the fire unharmed, but one of his neighbours died. He states that he can’t get the fire out of his mind, is unable to sleep properly, and when he does sleep, dreams about almost getting caught in the fire. When asked what happens in the dreams he states: “We’re back in the fire. I can hear the kids screaming, crying, then I see Joan running towards us, from her house, burning ... I wake up in a pool of sweat, certain that we are going to be next”. He also relates a number of instances where similar memories have been triggered, such as hearing fire engine sirens, hearing about stories of fires on the news, and when attending a local bonfire. When he experiences these memories, he feels and acts as if the trauma was happening all over again, needing to escape from the situation. Since the fire, he has felt helpless, hopeless, and is unable to concentrate on much at all. His wife complains that he is not the same person she married, having become withdrawn and emotionally detached.

Central to the controversy over the diagnosis of PTSD is the definition of the traumatic event, particularly as the event is a necessary criterion for the diagnosis. The stressor criterion has been described as a ‘gatekeeper to PTSD’ and accordingly if the criterion is loosely defined the prevalence of the disorder is likely to increase, but if it is too tightly defined, the prevalence will reduce (27). DSM-IV stressor criterion requires that “the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others” as well as requiring that “the person’s response involved intense fear, helplessness, or horror” (28, pp. 427-428). Does the emphasis on physical threat exclude individuals from meeting criteria for the disorder who otherwise meet all the remaining disabling features of PTSD?
A 40 year old clerk presents with persistent somatic symptoms of anxiety, sleep disturbance and irritability following a series of distressing incidents at his workplace. The incidents, which took place over a period of several months, included official complaints against his work, verbal abuse by his colleagues and obscene notes and drawings anonymously left at his desk. The man has not been able to return to work for some weeks and stated that he regularly experienced intrusive images and memories of these experiences which cause him great distress and anxiety.

How would the current diagnostic systems conceptualise the patient’s presenting problem? Should a diagnosis of PTSD be required in order for this man to be compensated for the harassment suffered at the workplace? What is the best treatment in this case? Should pharmacotherapy be considered?

Only a minority of people who experience a traumatic event as defined by DSM-IV will develop PTSD, but there is no conclusive evidence regarding the factors that might influence the severity of post-trauma reactions. Even traumas most commonly associated with PTSD such as combat exposure in men, and rape and sexual assault in women, do not always result in the disorder (29). The answer is likely to be complex and multifactorial, but a number of pre-existing vulnerabilities such as a prior psychiatric history, and characteristics of the trauma are likely to be important (30, 31). Even individuals who experience severe trauma may be aided in their recovery if good social support is available (32).


A New Unified Treatment Approach for Emotional Disorders Based on Emotion Science

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ABSTRACT—Research on the nature of emotional disorders points toward a common set of diatheses (underlying psychological vulnerabilities) and functionally (but not superficially) similar expression of pathological emotional responding (e.g., Barlow, 2002). Successful drug treatments for different emotional disorders are very similar, with selective serotonin reuptake inhibitors or related compounds in wide use. Successful psychological treatments, on the other hand, are currently specifically targeted to each individual disorder. Distilling common principles among existing empirically supported psychological treatments, and giving attention to new findings on emotion regulation and dysregulation from emotion science, we propose and describe a new unified psychological treatment for emotional disorders.

KEYWORDS—emotion; regulation; treatment; unified; avoidance

Psychological treatments emerging mostly from cognitive-behavioral traditions have been developed to accommodate the range of emotional disorders, defined here as the anxiety and unipolar mood disorders. A number of specific psychological treatments have gained empirical support in the treatment of these disorders, surpassing in most instances the efficacy of pharmacological treatments over the longer term (see Barlow, 2004). Despite this evidence, there are a number of limitations to current psychological treatments. Although approximately 50 to 80% of patients undergoing cognitive-behavioral psychological treatments for one or more of the emotional disorders achieve “responder” status (attainment of clinically significant treatment gains)—a higher figure than those typically achieved through alternative or “placebo” psychological treatments in anxiety and related disorders—this leaves a significant number of people who achieve less than optimal response or do not respond (see Barlow, 2002). In addition, successful psychological treatments are often complex and target individual disorders, making them numerous but narrowly construed (e.g., separate treatments for panic disorder, generalized anxiety disorder, major depressive disorder, etc.). These factors raise barriers to dissemination, as the separate protocols and materials for each disorder can require a significant amount of training, thus limiting clinician access to empirically supported treatments for the emotional disorders.

A solution may lie in the development of a common treatment approach applicable to a range of emotional disorders, one that distills hypothetically active procedures shared by current disparate treatments. Newly discovered commonalities within and between the anxiety and mood disorders, as well as the current emotion science literature, provide additional rationale for such a unified approach.

COMMONALITIES AMONG THE EMOTIONAL DISORDERS

Emerging evidence suggests considerable overlap among the anxiety and mood disorders. This is perhaps best seen in the high rates of current and lifetime comorbidity (presence of more than one disorder; e.g., Kessler, Chiu, Demler, Merikangas, & Walters, 2005). A large-scale study of 1,126 patients found that

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55% of patients diagnosed with a principal anxiety disorder had at least one additional anxiety or depressive disorder at assessment (Brown, Campbell, Lehman, Grisham, & Mancill, 2001). This number increases to 76% if one considers lifetime diagnoses.

Possible explanations for these high levels of comorbidity have been reviewed extensively (Barlow, 2002) and include overlapping definitional criteria, varying base rates of occurrence in different study settings, and a possible sequential relationship among the disorders such that features of one disorder serve as risk factors for another. Another possible explanation for this comorbidity is the presence of a “negative affect syndrome” (NAS). The collective symptoms of emotional disorders have been theorized as merely variable responses emerging from a more fundamental disorder (Barlow, 2002). Among evidence for the existence of NAS is the common observation of generalizable effects in treatment: Psychological treatments targeting a specific anxiety disorder result in significant improvement in co-morbid anxiety or mood disorders not specifically targeted by the treatments (reviewed in Barlow, Allen, & Choate, 2004). For example, successful treatment of panic disorder with or without agoraphobia (PDA) also impacts positively on comorbid diagnoses of generalized anxiety disorder (GAD) and depression, and these changes are largely maintained at follow-up. Although it cannot be determined from treatment outcomes if the treatment elements addressed the individual features of both disorders or if the disorders themselves contained similar underlying features that responded to treatment, the fact that a treatment for one disorder generalizes its effects to other comorbid disorders suggests the utility of a unified treatment approach.

In addition, research using confirmatory factor analysis to examine the factor structure (classification of variables into factors by the relationships between them) of the anxiety and mood disorders finds negative affect and positive affect to be higher-order factors to the symptom-specific disorder factors in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994), with significant pathways from negative affect to all of the disorder factors and autonomic arousal emerging as a lower-order factor (Brown, Chorpita, & Barlow, 1998). In other words, the model shows that negative affect influences all of the emotional disorders, which suggests a common diathesis (underlying psychological vulnerability). This study also found that certain mood disorders, such as major depressive disorder, overlap more strongly with some anxiety disorders (such as GAD) than with other mood disorders. These results again illustrate overlap both within and between the diagnostic categories of the anxiety and mood disorders. Much as our systems of classification appear to be moving away from “splitting” similar slices of symptoms into different disorders and toward a more dimensional approach (Krueger, Watson, & Barlow, 2005), it may be time to consider common principles of treatment.

EMOTION REGULATION AND EMOTIONAL DISORDERS

Although the scientific study of emotion has not, thus far, substantially influenced research on the nature and treatment of emotional disorders, attention is now turning to the process of emotion regulation (and dysregulation) as one facet in the development and treatment of anxiety and mood disorders. Reflecting Gross’s definition (Gross & Thompson, in press), Barlow and colleagues have defined emotion regulation as strategies used by an individual to manipulate an emotion’s occurrence, experience, duration, intensity, and expression (Campbell-Sills & Barlow, in press). Emotion regulation can be performed internally (e.g., trying not to think about an unpleasant feeling) or externally (e.g., smiling although feeling sad).

Goals of emotion regulation change depending on the context in which they are occurring. Emotion regulation is typically employed to decrease negative emotions and increase positive emotions. However, it may also be used to increase negative emotions and decrease positive emotions if doing so is situationally beneficial, such as masking joy at times when that emotion would be inappropriate to express, such as at a funeral or while empathizing with a sad friend (Gross & Thompson, in press).

Of course, emotion regulation serves an important purpose. Unregulated strong emotions might keep us from focusing on daily tasks or might result in socially undesirable behaviors such as emotional outbursts. However, maladaptive strategies for regulating emotion—particularly attempts to avoid emotion or to downregulate emotion during or after emotional provocation—can have unintended and negative consequences. For example, numerous studies have shown that use of emotional suppression, although intended to reduce emotional response, has little effect on emotional experience but can produce deleterious biological and cognitive effects such as increased sympathetic nervous system activation and impaired memory (Campbell-Sills, Barlow, Brown, & Hofmann, in press; see Gross & Thompson, in press).

Maladaptive emotion regulation seems to be a component of emotional disorders. In one study, individuals who tended to naturally suppress were more likely to be obsessional, anxious, and depressed (Marcks & Woods, 2004). From our own laboratory, a comparison of participants with anxiety and mood disorders to control participants found that the clinical participants were more likely to utilize maladaptive emotional-regulation strategies such as avoidant or suppressive behavior when viewing an emotion-provoking film (Campbell-Sills & Barlow, in press). Also, individuals with panic disorder fall back on emotional suppression or avoidant strategies in response to CO₂ challenge, a procedure that uses CO₂-enriched air inhalation to induce the physiological sensations associated with panic (Levitt, Brown, Orsillo, & Barlow, 2005).
A UNIFIED TREATMENT PROTOCOL

We have now developed a treatment protocol to target what we hypothesize to be the three main components of the major emotional disorders (Barlow et al., 2004). This approach distills and incorporates the most salient components of the currently empirically supported individualized treatments for various specific anxiety and mood disorders—namely, restructuring faulty cognitive appraisals, changing action tendencies associated with the disordered emotion, and preventing emotional avoidance and facilitating emotional exposure. The result is a conceptually sharper and (potentially) more widely applicable approach that incorporates advancing knowledge of modern learning theory, cognitive neuroscience, and emotion regulation (as reviewed above). We refer to this protocol as “unified” because it is designed to be applicable to all anxiety and unipolar mood disorders.

The protocol begins with a psychoeducation phase that describes emotions, their functions, and how they become disordered. The treatment then focuses on (a) altering antecedent cognitive appraisals, (b) modifying emotion-driven behaviors, and (c) preventing emotional avoidance.

Altering Antecedent Cognitive Appraisals

One adaptive antecedent-focused emotion-regulation strategy emerging from emotion science and cognitive-behavioral practice involves providing the patient with new attributions and appraisals to bring with them to their emotional experiences. In our view, there are two misappraisals common to the emotional disorders: Overestimating the probability of a negative event occurring and catastrophizing the consequences of a negative event should it occur. This strategy then utilizes standard cognitive restructuring techniques to help patients reappraise situations in a more adaptive manner. Importantly, these skills are practiced prior to emotion provocation and are not to be used to suppress or distract from emotional experience.

The positive benefits of antecedent reappraisal have been demonstrated in a study using CO₂ challenge for patients with PDA who typically panic in response to the procedure (e.g., Sanderson, Rapee, & Barlow, 1989). In this study, all patients were told that they would be able to adjust the mixture of CO₂ by turning a dial when a light was illuminated, thereby creating a sense of control over the biological challenge for this group of individuals. For the first 5 minutes, all patients received compressed air, followed by 15 minutes of 5.5% CO₂-enriched air. For 10 of the patients, the light was illuminated throughout the 15-minute CO₂ procedure, while for the other 10 patients, the light was never illuminated. The dial did not, in fact, control the flow of CO₂, and patients never actually resorted to using it, but patients in the perceived-control condition reported fewer panic attacks and less negative emotions than the patients in the no-control group did. Antecedent appraisals of control and familiarity prior to emotion-inducing stimuli have also been shown to reduce biobehavioral stress responses in the hypothalamic-pituitary-adrenal axis, a component of the neuroendocrine system that controls stress reactions (Abelson, Liberzon, Young, & Khan, 2005), and lead to attenuation of responding in the amygdala to anxiety-provoking stimuli (Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003).

Modifying Emotion-Driven Behaviors

Emotion-driven behaviors (EDBs), often referred to as “action tendencies” in the literature of emotion science (Barlow, 2002), are motivated behaviors that naturally occur in response to emotional states. EDBs ordinarily serve an adaptive purpose in specific situational demands (e.g., running away in response to fear triggered by actual danger) but can contribute to emotional disorders when emotions occur at inappropriate times (e.g., running away from a social gathering in response to fears of rejection). EDBs are initially reinforcing as they reduce emotional intensity, but they can be ultimately maladaptive. For example, withdrawal in response to fear of rejection produces further social exclusion and prevents new positive associations (e.g., learning that social situations are not often associated with rejection and embarrassment). A century of tradition in emotion science suggests that one of the most powerful methods for modifying emotions is to change EDBs and thereby change feelings (see Barlow, 2002).

This portion of the protocol teaches patients to recognize their usual EDBs and the resultant (ineffective) consequences. This often involves taking the patient through emotion-induction exercises that elicit cognitive, physiological, and emotional responses. The patients are then instructed to practice responses that are incompatible with their EDBs. Some examples of EDBs are presented in Table 1.

Preventing Emotional Avoidance

Whereas EDBs are initiated to reduce (escape) emotion intensity once it has occurred, emotional avoidance is used to prevent the full experience of emotion in the first place. There are several forms of emotional avoidance. Subtle behavioral avoidance typically occurs when a person encounters a situation he or she associates with strong emotions, such as the interoceptive avoidance (avoidance of somatic sensations) sometimes seen in individuals with panic disorder. Such individuals begin to avoid activities that may cause them to experience the physiological sensations associated with panic attacks, such as breathlessness caused by walking up a flight of stairs. Another form of emotional avoidance is cognitive avoidance. One example is distraction, in which the individual shifts attention to an alternative focus to avoid engaging in the emotional experience. The third form of emotional avoidance is use of safety signals. Individuals may carry objects that serve as talismans, such as a cell phone, toy animal, or empty pill bottle, because these objects convey
superstitious feelings of security in the event of a strong emotional experience such as a panic attack. Some examples of emotional avoidance are presented in Table 2.

The patient must be made aware of the relationship between emotion avoidance and EDBs, for even a patient successful at modifying their EDBs may still be preventing full emotional arousal during exposure through emotional avoidance, creating difficulties in identifying and replacing faulty emotion regulation strategies with more adaptive techniques.

**TABLE 1**

<table>
<thead>
<tr>
<th>Emotion-driven behavior</th>
<th>Disorder most usually associated</th>
<th>Incompatible behaviors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calling relatives to check on safety</td>
<td>Generalized anxiety disorder (GAD)</td>
<td>Restricting contact/calling relatives</td>
</tr>
<tr>
<td>Perfectionistic behavior at work or home appliances</td>
<td>GAD</td>
<td>Leaving things untidy or unfinished</td>
</tr>
<tr>
<td>Checking locks, stove, or other appliances</td>
<td>Obsessive-compulsive disorder</td>
<td>Repeatedly locking/unlocking and turning on/off until memory is unclear</td>
</tr>
<tr>
<td>Leaving (escaping from) a theater, religious service, or other crowded area</td>
<td>Panic disorder with agoraphobia</td>
<td>Move to the center of the crowd; smile or produce nonfearful facial expressions</td>
</tr>
<tr>
<td>Social withdrawal</td>
<td>Depression</td>
<td>Behavioral activation</td>
</tr>
<tr>
<td>Leaving (escaping) a social situation</td>
<td>Social phobia</td>
<td>Staying in situation and approaching people</td>
</tr>
<tr>
<td>Verbally/physically attacking someone when in an argument</td>
<td>Posttraumatic stress disorder</td>
<td>Remove self from situation and/or practice relaxation techniques</td>
</tr>
<tr>
<td>Hypervigilance</td>
<td>All disorders</td>
<td>Focus attention on specific task at hand; meditation; relaxation</td>
</tr>
</tbody>
</table>

**TABLE 2**

<table>
<thead>
<tr>
<th>Emotional-avoidance strategy</th>
<th>Disorder most usually associated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subtle behavioral avoidance</td>
<td></td>
</tr>
<tr>
<td>Avoiding eye contact</td>
<td>Social phobia</td>
</tr>
<tr>
<td>Avoiding drinking caffeine</td>
<td>Panic disorder with or without agoraphobia (PDA)</td>
</tr>
<tr>
<td>Attempting to control breathing</td>
<td>PDA</td>
</tr>
<tr>
<td>Avoiding exercise and other forms of physiological arousal (interoceptive avoidance)</td>
<td>PDA/depression</td>
</tr>
<tr>
<td>Avoiding touching sink/toilet</td>
<td>Obsessive-compulsive disorder (OCD)</td>
</tr>
<tr>
<td>Procrastination (avoiding emotionally salient tasks)</td>
<td>Generalized anxiety disorder (GAD)</td>
</tr>
<tr>
<td>Cognitive avoidance</td>
<td></td>
</tr>
<tr>
<td>Distraction (reading a book, watching television)</td>
<td>Depression/PDA</td>
</tr>
<tr>
<td>“Tuning out” during a conversation</td>
<td>Social phobia</td>
</tr>
<tr>
<td>Reassuring self that everything is okay</td>
<td>GAD</td>
</tr>
<tr>
<td>Trying to prevent thoughts from coming into mind</td>
<td>OCD</td>
</tr>
<tr>
<td>Distraction from reminders of trauma</td>
<td>Posttraumatic stress disorder</td>
</tr>
<tr>
<td>Forcing self to “think positive”</td>
<td>Depression</td>
</tr>
<tr>
<td>Worrying</td>
<td>GAD</td>
</tr>
<tr>
<td>Rumination</td>
<td>Depression</td>
</tr>
<tr>
<td>Thought suppression</td>
<td>All disorders</td>
</tr>
<tr>
<td>Safety signals</td>
<td></td>
</tr>
<tr>
<td>Carrying a cell phone</td>
<td>PDA/GAD</td>
</tr>
<tr>
<td>Carrying empty medication bottles</td>
<td>PDA</td>
</tr>
<tr>
<td>Holding onto “good luck” charms</td>
<td>OCD</td>
</tr>
<tr>
<td>Carrying items that are associated with positive</td>
<td>GAD/depression</td>
</tr>
<tr>
<td>experiences (e.g., teddy bears, pictures)</td>
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<tr>
<td>Having mace at all times</td>
<td>PTSD</td>
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<tr>
<td>Carrying a water bottle</td>
<td>PDA</td>
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<tr>
<td>Having reading material/prayer books on hand</td>
<td>GAD</td>
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<tr>
<td>Carrying sunglasses or items to hide face/eyes</td>
<td>Social phobia</td>
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</tbody>
</table>
DIRECTIONS FOR FUTURE RESEARCH

All treatments utilized in health care systems around the world must now be based on evidence, but interventions for emotional disorders will evolve as our understanding of the connection between emotion development, emotion regulation, and the emotional disorders continues to grow. Thus, researchers must try to clarify how and why disordered emotion develops and why it leads to maladaptive emotion regulation. Increased knowledge in this area would lead to further treatment development and, perhaps, to strategies for primary prevention. More fundamentally, deeper knowledge of emotion development and regulation could result in a reorganization of our system of classification for emotional disorders that would be more functional and dimensional. For example, the DSM-V or DSM-VI might eliminate separate anxiety, mood, and related (e.g., dissociative) disorders and instead specify presence of negative affect with notational references to presence and severity of associated features such as (low) positive affect, intrusive thoughts, rumination or worry processes, panic attacks, and emotional and situational avoidance (or dissociative) behavior—possibly organized like a Minnesota Multiphasic Personality Inventory profile. This characterization could lead to more individually tailored treatments.

Recommended Reading


REFERENCES


5 Treatments that work in anxiety disorders

Gavin Andrews and Caroline Hunt

Anxiety disorders are the commonest mental disorders in community practice, and there are practical psychological therapies that do help

On March 12 this year the Federal Minister for Health released the results of a national survey of mental health and well-being conducted by the Australian Bureau of Statistics.1 It showed that 9.7% of adults in Australia reported symptoms in the past year that met criteria for one of the six main anxiety disorders (Box 1). In comparison, 5.8% met criteria for depression and 7.7% for a substance-use disorder. The prevalence of anxiety disorders was stable across the 18 to 55 age range but reduced progressively thereafter — we do seem to get calmer as we get older. People with anxiety disorders reported being unable to carry out their usual roles or tasks one day in 12, almost at the same level as people with chronic physical disorders like heart trouble, asthma or arthritis, yet only 28% reported using health services, and three quarters of the time this meant seeing a general practitioner. Thus, the first question is why people disabled by an anxiety disorder do not seek help, or are not recognised when they do.

The answer is, in part, related to the normality of being anxious and, on occasions, too anxious. Anxiety is a normal emotion and a powerful motivator. Mild to moderate levels of anxiety improve the ability to cope, reactions become faster, understanding is better and responses are more appropriate. This sense of increased mastery is usually pleasurable. It is good to be aroused, tense, and anxious before important events, but care should be taken to see that the anxiety does not become so severe that it impairs performance. Just as moderate levels of anxiety facilitate coping, high levels reduce the capacity to plan, to make accurate judgements or to carry out skilled tasks, or even to comprehend useful information.

Patients who are stressed complain of being disabled by anxiety, yet some level of anxiety is required to make them keen to work on their problems. This facilitating effect of moderate anxiety and the debilitating effect of high anxiety makes the prescribing of anxiolytics difficult. Sufficient medication to produce the calm that patients seek will usually result in impaired performance. There is now considerable concern about the dependency-producing potential of the benzodiazepines and many doctors are looking to alternative treatments.2

In this article, alternative and useful drug therapies will be mentioned because they tend to be the treatments used most often in general practice (details of drug therapies can be found in chapters 4 and 6).2,3,4 But, like Tyrer,3,4 we would encourage doctors “to introduce patients to psychological treatment whenever possible”. The results of the national survey reinforce this advice. Of people with any mental disorder (and anxiety disorders were the most common diagnoses), three quarters expressed a need for counselling

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Insects, marine creatures and spiders in their webs are frequently painted by people with anxiety disorders. Reproduced with permission from the Cunningham Dax Collection of Psychiatric Art in the Mental Health Research Institute of Victoria.
or other “talking therapy”, significantly more than the number who expressed a need for medication. In this respect, the public are right. In the anxiety disorders, the cognitive behavioural range of treatments are mostly superior to therapy with drugs, both in short term effectiveness and in the long term, after treatment has concluded.6,7 These psychological treatments can restore the mental health of anxious people and overcome the debilitating effects of excessive anxiety.

Doctors must divide their time between attending to crises, caring for the chronically ill, and putting energy into people who they can cure and need never see again. The anxiety disorders are in the last category, potentially curable by psychological techniques, given a skilful practitioner and a hard-working patient.

Diagnosis of anxiety disorders

The presentation of most anxiety disorders is stereotyped and should therefore be simple to recognise. Patients with panic/agoraphobia fear collapse, insanity or death during a panic attack; patients with social phobia fear being negatively evaluated while under the scrutiny of others; unwanted and intrusive obsessions are largely confined to fears of contamination or harm; generalised anxiety disorder means irrational worry about things that may go wrong or one’s inability to cope; post-traumatic stress disorder is characterised by the continual triggering of intense and fearful memories of a previous trauma. Specific phobias are limited to irrational fears of harm in very circumscribed situations (e.g., heights, insects, furry animals, snakes, still water, closed spaces, blood and injury) that probably once had survival value. Specific phobias are seldom of clinical importance and will not be discussed further apart from saying that graded exposure (discussed later) is the treatment of choice.

Most disorders have signs as well as symptoms. Anxiety is different. The experience of being chronically anxious is a private one and there is little to observe, except perhaps some tremor and an occasional burst of unexpected panicky behaviour. One central feature of all anxiety disorders is that patients complain of the physical symptoms of the “flight or fight” response — rapid heart rate, need to overbreathe, tremor and shaking, nausea, sweating and focusing of attention — that would be normal if there was a significant physical exertion required to deal with a danger. Because hyperventilation is part of this response most patients also complain of dizziness and light-headedness, tingling and numbness.

Complaints of anxiety that differ from the stereotypes — especially in patients over 40 with no previous history of anxiety — should alert the physician to the need for skilful interviewing to discover the nature of the underlying condition. Of all such conditions, a depressive disorder with secondary anxiety is the most common, and the symptoms of depression will have to be specifically elicited — depression, loss of interest, loss of energy, loss of self esteem, being prey to morbid thoughts, disturbed sleep, and weight loss. Antidepressant drugs, especially the selective serotonin reuptake inhibitors (SSRIs), are the treatment of choice.

Physical conditions such as hyperthyroidism and other mental disorders such as schizophrenia, delirium or dementia can also present with untoward anxiety. In such cases, management of the underlying disorder is the top priority and first step towards controlling anxiety.

<table>
<thead>
<tr>
<th>Prevalence of mental disorders in Australia, 1997</th>
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<tbody>
<tr>
<td>Any anxiety disorder</td>
</tr>
<tr>
<td>Panic disorder</td>
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<tr>
<td>Agoraphobia</td>
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<tr>
<td>Social phobia</td>
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<tr>
<td>Generalised anxiety disorder</td>
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<td>Obsessive–compulsive disorder</td>
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<tr>
<td>Post-traumatic stress disorder</td>
</tr>
<tr>
<td>Any affective disorder</td>
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<tr>
<td>Any substance-use disorder</td>
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<tr>
<td>Any mental disorder</td>
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</tbody>
</table>

Management

Core symptoms

Treatment should deal with the flight or fight symptoms of anxiety, as many patients misinterpret them as evidence of an underlying physical problem. Thus, doctors have a responsibility to educate their patients about the flight or fight response and about these readily treatable disorders. Most patients seen at our specialist clinic have already been to their local bookstore seeking help for their symptoms, and the quality of such books is, in general, good. Isaac Marks’ Living with fear is exceptional, in that it was shown to be an effective treatment in a controlled trial.8 Andrew Page’s Don’t panic is an inexpensive and locally available self help book.

Anxiety secondary to threat or crisis

When a patient who is not normally nervous complains of anxiety, the first step is to delineate the nature of the threat or crisis. Most, it is not “things that go bump in the night” that produce anxiety, but the meaning that is given to them. If you are awakened during the night by a creaking door and decide that the cat has caused it, it is easy to drift back to sleep. However, if you decide that the noise was caused by intruders, you instantly become alert and anxious and, with a pounding heart and dry mouth, you rehearse what to do. Once it has been established that the noise was caused by the cat, it is easy to go to sleep again. It is not the event, but the thoughts about the event, that generate anxiety. The best way to reduce anxiety is to evaluate the situation, decide what to do, and then carry out what has to be done. In the consulting room, with the support of their doctor, patients can look at the ramifications of the problems causing their anxiety and begin to work out how they might cope.

It is often useful to identify four steps in helping such patients cope with a crisis:11

1. Ensure that they are certain the problem is theirs; many people get needlessly upset over distressing events that do not actually threaten their own well-being.

2. Have them write down the essence of the problem, including listing the outcome if everything were to go wrong. This task will illustrate the extent of their worrying thoughts.

3. Review the nature of the underlying condition.

4. If patients are unable to decide what to do, help them to outline their options and tackle these systematically. If they are unable to decide whether the problem is theirs, help them to see if they can think of a similar situation and decide what would probably happen.

5. Try to help them to learn strategies for controlling their anxiety and reduce their panic response. Help them to develop a behaviourally simple and logical plan of action that is based on the step-by-step strategy. This involves identifying and challenging any irrational thoughts by asking the patient whether they are always or sometimes true, and whether there is any evidence to support these thoughts. If the answer is no, it helps to gradually increase the number of thoughts that the patient can manage to challenge. This can be done in a controlled manner, first by thinking of one or two thoughts, then by thinking of three or four thoughts, and so on until the patient can tolerate thinking about more thoughts. This process can be repeated several times until the patient is able to tolerate thinking about more thoughts.

6. Help them to develop a plan of action, and to put this plan into practice. This can be done by asking the patient to write down a list of steps they can take to cope with the situation, and then to discuss these steps with their doctor. This can help to ensure that the patient has a clear plan of action.

7. Help them to develop a plan of action, and to put this plan into practice. This can be done by asking the patient to write down a list of steps they can take to cope with the situation, and then to discuss these steps with their doctor. This can help to ensure that the patient has a clear plan of action.

8. Help them to develop a plan of action, and to put this plan into practice. This can be done by asking the patient to write down a list of steps they can take to cope with the situation, and then to discuss these steps with their doctor. This can help to ensure that the patient has a clear plan of action.
3. Ask them to make a list of a number of ways of solving the problem, choose the one that seems most practical, and have them write down the steps involved in effecting that solution. Coping with the problem should always be divided into steps. The patient will see some steps as being manageable, while other steps may require further thought and perhaps help from others. If necessary, make formal use of the structured problem-solving technique described below.

4. Talk about how they will manage to stay reasonably calm until the problem is solved. Many patients can be helped to remain calm by seeking comfort and support from sensible friends, by putting energy into physical tasks, or by relaxing while reading or listening to music. Alcohol is not a suitable means of relaxation for people with anxiety disorders. Very occasionally, diazepam 5–10 mg daily for 1–4 days may be of value for the patient who becomes distraught, agitated, or sleepless, while stress management programs are of value for people who are chronically under pressure because of their life circumstances or occupation.11

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**2 Generalised anxiety disorder**

**Features**
- Months of excessive anxiety and worry
- The worry is out of proportion to the event, pervasive and excessive, difficult to control
- Accompanied by muscle tension, hyperarousal and symptoms of the “flight or fight” response

**Psychological treatment in primary care**
- Education about nature of disorder
- Progressive muscle relaxation
- Structured problem solving
- Graded exposure to difficult situations
- Specialist referral to a cognitive behavioural program for non-responders

**A typical presentation**

A 25-year-old woman presented with worries about her health, her career and her relationships. She said that she had always worried easily, but over the past several months she had felt more tense and agitated. The current increase in anxiety began following a dispute at work with a colleague who she believed had taken advantage of her, but since then she had been unable to assert herself with this colleague. She frequently worried about the quality of her work and worried that making a mistake would ultimately cause her to lose her job. Over this time she had developed a pattern of waking frequently during the night and being unable to get back to sleep for two to three hours while thinking about all her worries. She had also come to see her general practitioner for various somatic complaints over the years, which she worried were signs of a serious physical illness.

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**3 Panic disorder and agoraphobia**

**Features**
- Sudden attacks of fear or anxiety in situations of little danger
- Symptoms of the “flight or fight” response, complicated by hyperventilation and worsened by the fear of collapse or death
- Avoidance, for fear of panic, of situations from which escape is not possible or help is not available, typically public transport, travelling alone, crowded or lonely places

**Psychological treatment in primary care**
- Education about nature of disorder
- Hyperventilation control
- Graded exposure to feared situations
- Specialist referral to a cognitive behavioural program for non-responders

**A typical presentation**

A 30-year-old woman asked her general practitioner to investigate her heart. She reported that several months ago, while attending a postnatal exercise class following the birth of her first child, she noticed a dramatic increase in her heart rate. She also noticed that her breathing became difficult, there was tingling in her fingers and around her mouth, her muscles became stiff, and she felt pains in her chest. Fearing she was having a heart attack, she fled the class and sought help at the local emergency department, where an ECG showed no abnormality. Since then, she had experienced similar symptoms on numerous occasions, always seeking medical advice for reassurance. She could travel alone, provided she carried her mobile phone in case she needed to call for emergency medical help. Even so, she avoided crowded banks, shopping centres, and movies in case medical help would not be able to help her in time should she experience another “heart attack”.

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**The classic anxiety disorders**

**Generalised anxiety disorder** (months of irrational worry accompanied by somatic symptoms of anxiety — see Box 2) is the commonest anxiety disorder. It is an acute-on-chronic disorder that arises in a person who is by nature a worrier, the habitual oversensitivity and overconcern finally getting out of control. Cognitive behaviour therapy, with the goal of bringing the worry process under control, is the most efficacious treatment.6,7 Benzodiazepines reduce the anxiety and worry symptoms but too often lead to dependence. Buspirone is equally effective but seldom used, and low dose sedative tricyclic antidepressants are also of use. However, as this disorder is often an extension of a worrying nature, the cognitive behaviour therapies that can teach new ways of coping are more appropriate.12 General practitioners can educate their patients about the flight or fight
response and teach them how to decrease their physical symp-
toms using techniques such as progressive muscle relaxation.
Teaching structured problem solving (described later) is also
helpful in allowing people to focus the worry constructively.

Panic disorder/agoraphobia (sudden brief attacks of inca-
 pacitating fear that commonly lead to avoidance of precipitating
situations—see Box 3) is also common, especially in young
women, who present to doctors because they feel that the
physical symptoms reflect cardiac or neurological conditions.

Tricyclic antidepressants, monoamine oxidase inhibitors,
high potency benzodiazepines like alprazolam and the SSRIs
have all been shown to reduce panic frequency, anxiety, and
phobic avoidance.6 Introducing the antidepressant drugs is
often difficult because these patients are sensitive to side
effects, and the dependency-producing potential of the high
potency anxiolytics is a real cause for concern. Most im-
portantly, these drugs relieve symptoms but do not cure the
disorder.

Many patients will find that hyperventilation control
(described below), if sufficiently mastered, will be effective in
controlling panic attacks. Once the panic attacks are con-
trolled, the doctor will then have to encourage patients to enter
previously feared situations using the principles of graded
exposure, also described below. Cognitive behaviour therapy
programs for panic disorder/agoraphobia are more effective
than medication6 and the benefits have been shown to be
stable for five years after treatment has concluded.

Social phobia (avoidance of social interactions, for fear of
scrutiny by others—see Box 4) begins in adolescence and
affects both sexes equally. It can be totally incapacitating and
lead to a miserable hermit-like existence when the condition
shades into avoidant personality disorder. The diagnosis is
best made, not from the situations avoided or from the anx-
xiety in those situations, but from the key fear that others will
think badly of them.

Again, cognitive behavioural programs are the treatments of
choice.6,12 Most such programs are conducted in specialised

4 Social phobia

Features

► Excessive and unreasonable fears of being in the
  centre of attention in case of negative evaluation
  because of looking anxious or doing something
  embarrassing

► Situations that could lead to scrutiny or evaluation
  (social functions, being in a crowd, speaking to
  others) are avoided or endured with intense anxiety

Psychological treatment in primary care

► Education about nature of the disorder
► Specialist referral to a cognitive behavioural program
  is recommended

A typical presentation

A 35-year-old man presented with anxiety at his
workplace. Since a recent promotion, he had been
having difficulty attending meetings where he might have
to present information to his peers. He found the
symptoms of pounding heart, trembling, sweating, and
blushing so unpleasant that he had excused himself from
many meetings and begun avoiding as many as
possible. He was seeking help because his avoidance
was beginning to be noticed by his superiors at work.
When asked about other situations that caused anxiety,
he said he had avoided many social activities since his
adolescence, particularly if there was a chance that he
might be the centre of attention. He did not get anxious
when at home with his wife or with close friends. He was
particularly worried about the possibility that he might do
or say something foolish or embarrassing at work or at
social gatherings, and worried that others would notice
him sweating or blushing and know that he was anxious.
He believed that they would evaluate him negatively
because of this.

5 Obsessive–compulsive disorder

Features

► Obsessions are thoughts, images or impulses that
  occur repeatedly, are intrusive and distressing and
  can’t be suppressed or neutralised

► Compulsions are repetitive behaviours used to
  control or neutralise the obsessions and prevent the
  harm and reduce the anxiety, but which are
  excessive and disabling

Psychological treatments in primary care

► Education about the nature of the disorder
► Advice to resist carrying out compulsions
► Specialist referral to a cognitive behavioural program
  for non-responders to medication

A typical presentation

A 40-year-old man presented with a long history of
checking behaviour that was significantly interfering with
his life. He checked on “dangerous” items repeatedly
before being able to leave his home because of recurring
thoughts that something terrible — like an appliance
staring a fire — might happen and that he may
inadvertently be responsible for harm befalling others.
He performed his checking in a ritualised manner,
ensuring that all electrical items were switched off and
unplugged, at times having to count to four as he stared
at each item. If interrupted during these behaviours or if
feeling under pressure, he had to restart his checking
rituals. Similarly, if the thought that some appliance might
have been left on occurred during his checking
behaviour, the time spent checking each item was
lengthened considerably. He reported that he was
consistently late in getting out of the house because of
his checking, and frequently had to leave work during the
day to go home and check items again. He had been
asked to resign from two previous jobs because of his
constant lateness and absences from work.
centres and doctors may have to encourage their patients to attend. It is important to realise that the benefits of such programs are considerable and long-lasting. Drugs from four groups (reversible inhibitors of monoamine oxidase [RIMAs], SSRIs, benzodiazepines and monoamine oxidase inhibitors [MAOIs]) have all been shown to relieve symptoms, but none lead to an enduring recovery from the disorder. Indeed, it is difficult to conceptualise that these anxiolytic and antidepressant drugs could do other than improve well-being, leaving the core issue in the disorder, “fear of scrutiny”, untouched.

**Obsessive-compulsive disorder** (fears of harm following intrusive, repugnant thoughts of contamination, violence or blasphemy, controlled by ceaseless washing or checking – see Box 5) is a rare, disabling condition that begins in adolescence and affects both sexes equally. The diagnosis, while often evident, is a rare, disabling condition that begins in adolescence and affects both sexes equally. The diagnosis, while often evident, is confirmed by the content of the recurrent obsession.

Treatment has concluded. About 50% of patients respond to SSRIs, with an average drop of 20%-40% in obsessions and compulsions. Again, the issue is over the relative merits of a simple but non-curative drug therapy with the limited goal of partial relief of symptoms versus a more complex psychological intervention with some chance of effecting a cure.

Specialist treatment with cognitive behaviour therapy that involves entering situations that evoke the thoughts but not carrying out the compulsions can provide superior results to medication, improvement that is usually maintained after treatment has concluded. Patients treated with medication should, as the intensity of the obsessions diminishes, be encouraged to resist carrying out the compulsions when entering situations that evoke the thoughts. Reassurance from the doctor which addresses the patient’s fears is contraindicated during treatment as the patient is encouraged to confront his or her fears and find them groundless.

**Post-traumatic stress disorder** (nightmares, flashbacks and emotional numbness that continue months or years after surviving a dreadful experience – see Box 6) often presents as depression, the traumatic experience being concealed. Three classes of drugs (MAOIs, tricyclic antidepressants and SSRIs) all reduce the intrusive thoughts, the anxiety and depression, and the sleep problems. These are real gains, but, while the symptoms respond to medication, the core inability to trust and feel safe again does not.

The core problem will respond only to careful and repeated exposure to the details and emotions surrounding the original experience and to the cues in the current environment that evoke the traumatic memories until they lose their power to disturb. This type of therapy should only be carried out by experienced specialist therapists, but anxiety management strategies such as hyperventilation control or progressive muscle relaxation and the drug treatment of comorbid conditions is within the range of all general practitioners.

**Psychological treatment of the major anxiety disorders in general practice**

The management of mental disorders is a guide published by the World Health Organization Collaborating Centre in Sydney that can be recommended to all general practitioners. It contains practical information about the recognition and treatment of people with mental disorders, patient information sheets about the common disorders and discussion of the three psychological techniques described below:

- hyperventilation control techniques to help patients lower the acute level of anxiety
- graded exposure to feared situations to eliminate the avoidance
- structured problem solving approach to facilitate crisis resolution.

Hyperventilation control and structured problem solving, along with other interviewing, prescribing and counselling skills, are also illustrated in the companion WHO-sponsored interactive CD-ROM Counselling and management skills in clinical practice.
The hyperventilation control technique\textsuperscript{6,17} has two key elements: regular monitoring of respiration rates by the patient and practice of the slow breathing technique to inhibit hyperventilation when anxious. The essence of the technique is simple — “Hold your breath for six seconds and then breathe in and out in a six second cycle” (Box 7), but, because it is to be used when stressed and anxious, it needs to be overlearned if patients are to be able to use it when they need it. Thus, general practitioners need to educate their patients about the rationale, and have them regularly practise counting the respiration rate and doing the slow breathing procedure, both at home and in the doctor's presence. Many patients will say that they already understand the technique, but if you have them rehearse during the consultation it is amazing how many think that deep breathing, which only worsens the hypocapnoea, is what is meant by slow breathing, which, of course, is aimed at reducing the hypocapnoea.

The structured problem solving approach is displayed in Box 8. At first glance it appears to be little more than applied common sense — carefully identify what troubles you, work out how to deal with it, do what has to be done, then review progress. But acting in a common sense fashion is exactly what people who are anxious have difficulty doing. The procedure was derived from problem solving techniques used in industry\textsuperscript{18,19} and is actually very sophisticated for, with the doctor's guidance, the patient learns to appraise situations accurately and then develop appropriate coping techniques. After one or two crises handled in this way patients seem to learn to carry out the technique for themselves. It is a most useful technique for general practice and doctors should photocopy figure 1, enlarge it to A4 size and use it routinely with patients who are anxious or distressed.

As Mitchell mentioned and others have shown,\textsuperscript{5,10,22} it is effective in the management of people with depression and of value in managing people who have attempted suicide.

The first step is to get the patient to specify the key threat or problem. Overwhelming and complex problems can usually be broken down into a series of component parts, best specified as a list of discrete goals, one of which is identified as the target problem. The target problem is ideally single, specific and potentially solvable.

Next, have the patient quickly list a number of ways that this problem could be solved or modified. Also list impractical solutions because they may contain the germ of a good idea. Then have the patient discuss the main advantages and disadvantages of each solution and agree on the preferred one. Now have the patient plan the steps required to put this solution into effect. The details should be written down, including names, addresses and phone numbers, as, when anxious, patients will all too often forget or confuse the rational steps they planned. Do not offer advice during the problem solving unless something the patient proposes to do is clearly impractical. It is their problem and by learning how to solve it they will be learning a process that they will continue to use independently of the doctor. Nevertheless, one must arrange to review the results, applauding the outcome. Real problems are complex, and even if the first attempt proves to be ineffective something of use will have been learned that can be used when the process is repeated.

<table>
<thead>
<tr>
<th>7 Slow breathing technique</th>
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</thead>
<tbody>
<tr>
<td><strong>Using the second hand on a watch or clock:</strong></td>
</tr>
<tr>
<td>➤ Hold your breath for six seconds</td>
</tr>
<tr>
<td>➤ Breathe in and out on a six-second cycle, saying the word “relax” as you breathe out</td>
</tr>
<tr>
<td>➤ After one minute, hold your breath again, then continue to breathe on a six-second cycle</td>
</tr>
<tr>
<td>➤ Repeat the sequence until anxiety has diminished</td>
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Structured problem solving with complex problems is an iterative procedure, a procedure infinitely more powerful than the usual advice from the doctor.

**Graded exposure**\textsuperscript{2} was one of the first of the powerful behavioural techniques. It is of value in assisting patients to overcome phobic situations by gradually re-exposing themselves to the feared situation in a manner that allows them to habituate themselves to it.

Confronting fears is common sense. However, few people with well established agoraphobia, social phobia or obsessive-compulsive disorder will be prepared to confront their principal fear and remain in the situation until the fear subsides. Hence the need for the graded approach.

The first step is a detailed analysis of the feared consequence. In agoraphobia the real fear is of the consequences of a panic attack, so, for example, express trains are feared because escape will be impossible and medical help unavailable should a panic attack eventuate. Conversely, in social phobia the real fear is of negative evaluation by others, and so an express train — where everyone becomes immersed in the view or their reading — is preferred to a commuter train, in which people get on and off all the time and are in a better position to notice others around them. The key question to ask about a fear is “In case what?” and most people will then usually describe the central fear. Once this central fear is understood, the doctor and patient can together generate a hierarchy of tasks — starting with one that can be done with minor discomfort and finishing with one that is almost unthinkable because of the anxiety it would provoke (Box 9).

Once the hierarchy is constructed the patient should confront the first situation repeatedly, remaining in the situation until the anxiety is halved, and repeat the task until there is little anticipatory anxiety associated with the prospect of entering the situation. By mastering the less anxiety-provoking situations, the person learns that fears that are confronted lose their power to frighten.

Benzodiazepines should not be used to make the task easier, for new learning will not occur; instead, dependence on benzodiazepines will only be reinforced. Likewise, dependence on “safety” behaviours or devices, like carrying a mobile phone to summon help, should be avoided simply because the person has to learn to face their central fear of panic and heart attack, or embarrassment and shame, unprotected by any external aids. The doctor will need to see the person regularly to check on progress, to encourage when motivation flags and to reward when tasks are achieved. Once a number of stages in the hierarchy have been surmounted, the rewards of increasing mas-
8 Structured problem solving

**Step 1: What is the problem/goal?**
Think about the problem/goal carefully, ask yourself questions. Then write down exactly what the problem/goal is.

**Step 2: List all possible solutions**
Put down all ideas, even bad ones. List the solutions without evaluation at this stage.

1. __________________________________________________________________________________________
2. __________________________________________________________________________________________
3. __________________________________________________________________________________________
4. __________________________________________________________________________________________
5. __________________________________________________________________________________________
6. __________________________________________________________________________________________

**Step 3: Assess each possible solution**
Quickly go down the list of possible solutions and assess the main advantages and disadvantages of each one.

**Step 4: Choose the “best” or most practical solution**
Choose the solution that can be carried out most easily to solve (or to begin to solve) the problem.

**Step 5: Plan how to carry out the best solution**
List the resources needed and the major pitfalls to overcome. Practise difficult steps, make notes of information needed.

**Step 6: Review progress and be pleased with any progress**
Focus on achievement first. Identify what has been achieved, then what still needs to be achieved. Go through steps 1 to 6 again in the light of what has been achieved or learned.

What has been achieved? __________________________________________________________________________________________________________________________________________________________

What still needs to be done? __________________________________________________________________________________________________________________________________________________________

9: Graded exposure

- Identify specific goals and break them into smaller, manageable steps
- Learn to master situations that cause mild anxiety
- Progressively master situations that are associated with greater anxiety
- Confront fears regularly and frequently
- Emphasise habituation to anxiety in each exposure session

**Example of a graded exposure hierarchy**

**Goal: To travel alone by train to the city and back**

1. Travelling one stop, quiet time of day (anxiety level 4/10)
2. Travelling two stops, quiet time of day
3. Travelling two stops, rush hour (anxiety level 6/10)
4. Travelling five stops, quiet time of day
5. Travelling five stops, rush hour (anxiety level 8/10)
6. Travelling all the way, quiet time of day
7. Travelling all the way, rush hour (anxiety level 10/10)

**Note:** Anxiety levels are those predicted by the patient before starting treatment

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**The good news**

The anxiety disorders are common and disabling mental disorders. They respond better to psychological techniques than they do to medication.6,7 These psychological techniques are more demanding of the doctor's skill and time than drug therapy, but they offer hope of a cure rather than temporary symptom relief. Few patients with anxiety disorders should stay sick. And that is good news.

**References**

Personality Disorders

1. DSM criteria – not provided


SPECIAL FEATURE: THE ETIOLOGY OF PERSONALITY DISORDERS: A REVIEW AND CONSIDERATION OF RESEARCH MODELS

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In addition to reviewing representative studies of genetic and environmental factors imputed in the etiology of the personality disorders (PDs), a number of models for conceptualizing and conducting etiological research are considered. In particular, it is proposed that research should initially concede a tripartite model (with separate temperament, personality, and disorder components). Such a model would allow identification of etiological factors having specificity to one or more components, and ones that are nonspecific in having relevance to all components.

Etiological issues are worthy of pursuing for two principal reasons. Firstly, because we know so little about the separate contribution of genetic and environmental factors to the personality disorders (PDs). This makes much of our teaching assumption-based at best, and restricts our capacity to define logical management strategies. Secondly, the number of PDs remains unclear. Etiological research has some capacity to assist taxonomic issues, so that an iterative process involving etiological and classificatory studies has many advantages.

Current measures of PD classes are ambitious, seeking to define "disorder" status as well as the underlying personality dimensions in a single-stage procedure, thereby confounding two potentially independent domains and being insufficiently weighted to the measurement of "disorder." Several examples can be provided from the DSM-IV (APA, 1994) descriptor sets. One descriptor of obsessive-compulsive PD (i.e., "shows perfectionism that interferes with task completion") begins by describing personality and ends with a focus on disordered function. A descriptor for avoidant PD (i.e., "avoids occupational activities that involve significant interpersonal contact, because of fears of criticism, disapproval, or rejection") conversely addresses dysfunction first, then personality style. Some PDs in the DSM-IV

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The views and assistance of Sandra Evans, Kerrie Eyers, Heather Brotchie, Robert Finlay-Jones, Yvonne Foy, Dusan Hadzi-Pavlovic, and an anonymous assessor of this manuscript are gratefully acknowledged.

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system (e.g., dependent PD) are defined with a focus on descriptors of personality, others (e.g., borderline PD) with a focus on disordered behavior and functioning.

In reality, an individual may be statistically "abnormal" on one or more personality dimensions, but not particularly disordered; conversely, there are those who express their disordered function widely, but lack dimensionally distinctive personality traits. Such confounding also limits our capacity to define the etiological contributions to the separate components (i.e., personality and disorder). The need to respect such concerns, and a lesser concern about distinguishing "temperament" and "personality," argues for the development of conceptual models, as well as a lexicon that allow interpretation of the current literature and the structuring of future research. At first pass, a tripartite model would appear to have utility for etiological pursuits, and is now detailed.

A TRIPARTITE MODEL ARTICULATED

TEMPERAMENT

As put by many (e.g., Berger, 1982), “temperament” can be conceptualized as referring to the genetically-driven, constitutional components (the "simple, non-motivational, non-cognitive, stylistic characteristics" enumerated by Rutter [1987]), which form a template at birth, ready for epigenetic development and environmental exposure. Temperament is the biogenetic hard wiring, significantly genetically driven, but with other prenatal biological influences conceded.

Measures of the early expression of temperament, however, show modest test-retest reliability over childhood and adolescence at best, and little clear overlap with adult expressions of “personality.” Both the nine-dimensional model (i.e., activity; rhythmicity; approach/withdrawal; adaptability; intensity; mood; persistence; distractability; and threshold) and the consolidated three-cluster model (easy, slow to warm up, and difficult) of childhood temperament (Thomas & Chess, 1984) show little resemblance to the “big five” model of adult personality—although links are more observable with the adult model of personality put by Buss and Plomin (1975), involving emotionality, activity, sociability, and impulsiveness. Those two caveats might encourage the view that, even if appropriately defined and measured, temperament and personality are differing constructs.

Differences, however, may be more apparent than real, for at least three reasons. First, a common sense one (ie how could personality spring from anything but the temperament bedrock?). Second, methodological reasons. Most temperament descriptors appear to focus more on specific aspects of behavioral style and on aspects manifested in infancy or early childhood. Third, epigenesis allows that continuity may be present despite varying expressions over development (especially when age influences surface expression considerably and so compromises test-retest estimates), and that continuity can be heterotypic. As an illustration, Rutter and Rutter (1993) have noted how a butterfly looks nothing like a caterpillar—and that “behaviors may change in
ETIOLOGY OF PD

form while still reflecting the same process.” Thus, lowish test-retest reliabilities for temperament measures are not necessarily a problem, particularly as the data sets almost invariably involve “surface markers” rather than the latent constructs which (if the definition of temperament is to be accepted) are likely to have greater continuity over time.

PERSONALITY

While manifested by a range of cognitive, intrapsychic, behavioral, and interpersonal characteristics, and requiring some constancy across time and situation, we allow some context-driven variation in expression. Vernon (1964) included habitual behaviors, organized traits, attitudes and roles, transactions between dispositions and situations, subjective and internal appraisal, and interpersonal relationships—with the last few emphasizing an interpersonal component.

As suggested, personality might merely reflect the evolution, maturation, or unfolding of temperament. It is more likely, however, that personality represents temperament enriched by a number of nonbehavioral components (e.g., motivational, cognitive, and intrapsychic), variably modified by a range of developmental influences. Such a model can be illustrated by considering the analogy of attachment to bonding. Bowlby (1969) has described the evolution of attachment in infancy and childhood, with initial attachment behaviors in the infant being instinctive (i.e., inherited, specific, and stereotyped), and with evolving repertoires that are progressively more interactive. Despite attachment behaviors being “hard wired,” they may be influenced by a number of variables—thus, being increased when a child is tired, hungry, in pain, or unwell, and increased by fear, anxiety and rejection by the attachment figure (see Rutter, 1980). As the infant matures, and as cerebral cortex-mediated functions come into play, there is cognitive interpretation of attachment figures, and the formation of selective bonds that are personal, social, and reciprocal, so that bonding reflects a more interactive and dynamic process. Just as attachment evolves into bonding, temperament may evolve into personality.

DISORDER

Two principal models underlie the psychological and psychiatric literature, although each includes elements of the other. First, there is definition primarily on the basis of the individual occupying some statistically deviant position on one or more dimensions defining temperament and/or personality. This would appear insufficient, as such a characteristic alone does not necessarily equate with impairment. Second, there are definitions more weighted to psychopathology, whether dimensionally or categorically defined, in that the individual and/or those around him must suffer as a consequence of the disorder. Thus, in addition to a dimensional component of temperament and personality, the second model emphasises a clinically focussed additional concept of disordered functioning. Suggested components include (a) impairment in social or occupational functioning or by subjective distress (APA, 1987); (b) “failure to attain the universal tasks of
identity, attachment, intimacy, or affiliation" (Livesley, Schroeder, Jackson, & Jang, 1994); and (c) as operationalized within DSM-IV (APA, 1994), being manifested in at least two of the following—cognition, affectivity, interpersonal functioning, and impulse control.

RELEVANCE TO ETIOLOGICAL STUDIES

While extremes in temperament and personality style might contribute to many PDs, they do not of themselves define PD, for those occupying one or more extreme dimensional positions may occupy a functional ecological niche and not be impaired or disordered. Again, it remains unclear whether formal PD status can be achieved on the basis of disorder alone and without any extreme dimensional temperament/personality component. Thus, as disorder may then be somewhat independent of dimensional extremes in temperament and personality, each may be contributed to by quite different etiological factors.

Let us assume that [A] = an obligatory inner core of temperament; that [B] = an obligatory mantle of transacted (individual with the world) characteristics; and that [C] = a crust of impaired or disordered functioning. Our recent and current DSM systems (defining PD status) involve combinations of [A+B+C], generally weighting [A+B] over [C]. The components need to be differentiated to allow the etiological contributions to each to be pursued, as well as any contribution made by one component to another (e.g., A, B, and A+B as predictors of C).

Since differences between temperament and personality may be slight or substantial, semantic or meaningful, and as their definition within any tripartite model is unlikely in the short term, it may be more useful to study the latent taxa, both in terms of their epigenesis across normal development and the extent to which genetic and environmental factors contribute to PD via such taxa. If personality is temperament plus overlay, we might expect a modification of genetic and other endogenous determinants, together with evidence of a greater contribution from the social world, as we move from studying temperament to personality, and perhaps with an even greater contribution from developmental factors as we move to studying determinants of the disorder component.

CAN WE MEASURE TEMPERAMENT AND PERSONALITY?

Representative (and quite different) measures developed for studying adults include: (a) the Eysenckian extraversion/neuroticism (EN) model, or his three-factor model with psychoticism added (i.e., tough-minded vs. tender-minded) (Eysenck, Eysenck, & Barrett, 1985) to generate an EPQ measure; (b) circumplex or interpersonal circles, dating back to Leary’s work, but developed by Wiggins (1982) and by Benjamin (1993), (c) the NEO (McCrae, 1991) or other “big five” measure, held to focus on “all the major sources of individual differences in personality” (Stallings et al., 1995); and (d) the TPQ, assessing novelty seeking, harm avoidance and
reward dependence—and with persistence subsequently added (Cloninger, Syrakic, & Przybeck, 1993).

From such contenders, others have sought to extract refined dimensions. For instance, Stallings et al. (1995), using a twin sample, undertook a joint factor analysis of the TPQ, EPQ, and KSP (Karolinska Scales of Personality), the last held to measure temperament dimensions. Four factors produced the best data description (and, reflect to my inspection: I = anxiety, harm avoidance, neuroticism; II = stimulus seeking and avoiding monotony; III = impulsiveness; IV = reward dependence). Secondly, short forms of the TPQ and the EPQ were completed by 2680 adult Australian twins pairs (Heath, Cloninger, & Martin, 1994). The authors concluded that the two systems were "not simply alternative descriptions of the same dimensions of personality, but rather each provided incomplete descriptions of the structure of hereditary personality differences." Rather than confirming overlap or consistency, the two scales appeared “instead to jointly assess five or six dimensions of genetic variability and at least six dimensions of environmental variability.”

Such analyses suggest that we neither know to what extent such measures assess temperament as against personality, nor their capacity to assess any overlap. As noted, definition of the key [A and B] components is more important in pursuing the extent to which they effect any etiological diathesis to disorder (or C status), rather than in modelling PD as merely a dimensional extreme on one or more of those components.

MEASURING THE “DISORDER” COMPONENT OF PERSONALITY DISORDER

Millon (1969) has conceptualized a disorder component that is, at least a priori, independent of personality and temperament dimensions. The model allows PD to be manifested across one or more differing parameters (i.e., an inflexible or defective response style; a personality style giving personal discomfort or decreased opportunities; the inability to function effectively and efficiently; the inability to adjust to the environment; vicious or self-defeating circles; and tenuous stability under stress). In relation to the last, while personality is defined as stable over situations and consistent over time, it may be that the disorder component of PD is more definable in stressful situations (Rutter, 1994). Millon (1986) further identified four functional domains (i.e., expressive acts, interpersonal conduct, cognitive style or attributions, and regulatory mechanisms such as defence styles) as well as four structural domains (mood/temperament; self-image; object representation; and morphological organization) for the expression of PD.

In addressing the need for a measure of “disorder,” there would appear to be distinct advantages to building on such an approach, perhaps by operationalizing such a matrix across intimate and family relationships, as well as across work and work relationships, weighted to their expression when the individual is stressed, and with a measure developed to ensure valid cut-off values. Such a measure should allow an individual to reach
PD status "at home and/or at work," so respecting a practical reality—that some PD subjects (e.g., the "successful sociopath") perform adequately (if not prosper) in some environments while creating problems (if not calamities) in other environments.

CAN ETIOLOGICAL STUDIES ASSIST DECISIONS AS TO WHETHER PD STATUS SHOULD BE DIMENSIONALLY OR CATEGORICALLY MODELLED?

Livesley et al. (1994) held that there were at least four models relevant for modelling PD. "(1) The distributions of the phenotypic features of personality disorder are continuous and show either bimodality or a point of rarity. (2) The traits composing personality disorders are continuously distributed, but there is a threshold effect; disability occurs only when a given trait magnitude is exceeded. (3) The individual personality disorder traits are continuous, but their structure differs in disordered and normal populations. (4) The latent structures, including the genetic substrates, underlying continuously distributed phenotypic manifestations are discontinuous" (p. 8).

Clarification might be assisted by identifying and testing several dimensional etiological factors (e.g., level of family discord or parent-child violence) against such models. A dose/response relationship between the relevant etiological factor and PD severity would support a dimensional model, while a discontinuous relationship (or trend break) would support a categorical model or the disjunctive PD components that are conceded by a tripartite model. In the debate over modelling the PDs dimensionally or categorically, there is a real risk in accepting only one of the two alternatives allowed by such a forced dichotomy. If a tripartite model has any validity, it is unlikely to be captured by a simple dimensional model or by a categorical model which has failed to identify whether the categories should be underpinned by severe expressions of personality traits and/or by disorder status.

IN PURSUITING THE ETIOLOGY OF THE PERSONALITY DISORDERS, WHAT SHOULD THE DEPENDENT VARIABLE(S) BE?

First, some categorical variables can be offered. Psychiatric research tends to assume the validity of class (e.g., DSM and ICD) systems. As subjects may meet PD diagnostic criteria on the basis of their personality traits and/or disorder status, this approach alone (even without considering limitations to valid class assignment) has numerous limitations. If categorical measures are to be used, there may be greater wisdom in defining and using the latent taxa underlying those surface marker classes as the dependent variables.

As there is clearly increasing support for dimensional approaches to assessing PD (Widiger, 1991), logic would dictate that etiological studies should also use dimensional measures. But which ones? Some might
require that the dimensional measure has the capacity to predict formal (i.e., DSM or ICD-defined) PD status. If we require such a capacity, are our choices narrowed? I will consider several representative and differing potential approaches.

TEMPERAMENT

Cloninger postulated that his TPQ measure had the capacity to generate eight PD classes (i.e., antisocial, histrionic, passive-aggressive, explosive, obsessional, schizoid, cyclothymic, and passive-dependent), but not paranoid or schizotypal. On testing, however, the TPQ poorly differentiated adapted individuals from those with personality disorder (Cloninger et al., 1993). Predictability was enhanced, however, by using the character subscales (i.e., self-directedness, cooperativeness, and self-transcendence) developed in the TCI (Temperament and Character Inventory). Thus, Svrakic, Whitehead, Przybeck, & Cloninger (1993) demonstrated that low self-directedness (in particular) and low cooperativeness scores predicted the number of PD symptoms and categorical PD diagnoses, with the chance of a DSM-III-R PD being diagnosed increasing from 22% to 94% as self-directedness scores decreased. Such analyses suggest that the links with PD status were driven more by scales that assess the disorder rather than any stem temperament component.

PERSONALITY STYLE

Widiger and Costa (1994) have considered the extent to which key dimensions of personality underlie the PDs and therefore allow their inter-relationship to be defined. Such attempts have generally failed to demonstrate that personality measures can predict PD status with acceptable precision. There are likely to be a number of reasons, but one reality is that PDs are not necessarily manifested by extreme positions on any dimensional personality measure but rather by oscillations (e.g., the borderline PD individual who moves from extreme love to extreme hate).

SEVERITY OF PERSONALITY DISORDER

Assuming that the PDs are underpinned by dimensions, Livesley (1986, 1987) generated items for the 11 DSM-III PD diagnoses and then derived three factors (i.e., interpersonal and intrapsychic dysfunction; impulsivity and deviant socialization; and compulsive behavior). Such approaches are not necessarily limited by the concern articulated earlier—that DSM-type systems amalgamate descriptors of personality and of disorder—as labeling of the identified factors in the Livesley studies are clearly weighted more to disordered function (i.e., dysfunction, deviant) than stem personality dimensions.

INSTRUMENTS ASSESSING AN AMALGAM OF PERSONALITY AND PERSONALITY DISORDER

Measures such as the SNAP (Clark, 1991) derive scores for both personality trait and personality disorder dimensions. Data from the SNAP manual
(Clark, 1991) demonstrated (Table 30) correlations between SNAP scale scores and Axis II diagnoses, but the overall classification rate of SNAP scores in assigning presence/absence of DSM PD status and PD sub-type has not been reported.

CIRCUMPLEX MODELS

Here the focus might be on the extent to which scores on key dimensions or axes, such as warmth and control (see Millon, 1996, pp. 58-59), predict Axis II status. Kiesler (1986), for instance, has proposed that DSM-III descriptors of most PDs have strong relationships with the taxonomic structure of interpersonal measures. However, overall classification rates of PD status and type have not been reported.

SEVERITY OF DISORDER MODELS

An obvious way of focusing on disordered functioning would be, as suggested earlier, to operationalize the criteria sets summarized in DSM-IV, or those parameters articulated by Millon (1986), and create dimensional scales. I am not aware of any such approach. We might anticipate that, as for the TCI self-directness and cooperativeness scores, there could be quite high prediction of PD status (because of the capacity of those scales to define disordered aspects of PD functioning), but limited prediction of individual PD sub-types.

CONCLUSIONS

The historical approach underpinning etiological research has been to accept categorical dependent variables (e.g., meeting of DSM Axis II criteria), despite numerous methodological limitations to deriving valid diagnoses. Psychiatrists, by and large, are currently reluctant to move to dimensional models, unless the dimensional measure used has the capacity to predict both (a) presence or absence of a PD, and (b) particular PD classes. Such objectives pose, in my view, an unnecessarily stringent quest (when we recognise the difficulty in reliably establishing PD diagnoses) and are not worthy of being pursued. They are no easier than our capacity to define meaningful categorical sub-types (e.g., males vs. females) in a school class by use of dimensional measures (e.g., IQ, age, height, and weight), and are made more difficult by a range of other definitional and methodological issues. If, however, we replaced the current DSM categorical basis of classification with a dimensional system, and subjected it to some deconstruction to generate dimensions assessing (a) temperament, (b) personality sub-type, and (c) extent of disorder, it would then be easier to pursue productive etiological research, particularly in identifying any differential, as well as universal relevance, of risk factors to the deconstructed dimensions.

EMPIRICAL ETIOLOGICAL STUDIES

Compared to the tripartite model, most of the empirical studies claim to have assessed either temperament, personality, or formalized personality
disorder classes. The extent to which such studies of temperament and personality correspond to the definitions advanced earlier in this article remains, however, problematic. The terms have been ascribed quite varying meanings in the absence of an accepted lexicon. Furthermore, as 'disorder' has rarely been conceptualized and operationalized, there are few studies examining etiological influences on disorder per se. The current overview will consider representative studies.

**STUDIES ASSESSING GENETIC INFLUENCES OR HEREDITABILITY**

**TEMPERAMENT**

Two representative studies assessing young children will be noted. Torgersen (1982) assessed temperament dimensions (as identified by Thomas and Chess) in a longitudinal study of 34 MZ and 16 DZ twins. At two months, hereditability was demonstrated for regularity, threshold, intensity, and mood. At nine months, all but destructibility and persistence were significant. At six years of age, the latter two were significant, but mood and regularity were not. The extent to which the temperament dimensions varied in their hereditability over time is capable of being explained by a number of factors, including measurement problems, epigenesis of temperament, and temperament patterns not being firmly established prior to four years of age (see Watkins, 1994).

Temperament dimensions isolated in two longitudinal studies (i.e., the New York study involving 141 children followed by Thomas, Chess, and Birch (1968) and the Buss and Plomin (1975) study of 91 pairs of children) were merged into a parental rating questionnaire called the Colorado Childhood Temperament Inventory (CCTI). Intraclass correlations (see Plomin and Rowe, 1977) indicated strong genetic influences for five identified traits (i.e., sociability, emotionality, activity, attention span-persistence, and soothability) but not for the sixth (i.e., reaction to food).

**PERSONALITY**

Several representative studies (using differing measures of personality) will be noted. Tellegen et al. (1988) administered the MPQ (11 primary scales and three higher-order scales) to MZ and DZ adult twins either reared together or apart. Genetic variances were 0.40 for positive emotionality (akin to extraversion), 0.55 for negative emotionality (akin to neuroticism), and 0.58 for constraint (corresponds to conscientiousness), allowing the authors to conclude that about 50% of measured personality diversity can be attributed to genetic diversity. Intraclass correlations were similar for MZ twins reared together and apart.

McCartney, Harris, and Bernieri (1990) undertook a meta-analysis of eight personality traits examined in studies of adult MZ and DZ twins. Hereditability was evident for most of the traits examined, being most distinct for anxiety, emotionality, sociability, activity-impulsivity, and dominance, and somewhat less evident for task orientation, aggression, and masculinity-femininity.
Cloninger (1990) overviewed a number of large-scale twin studies, estimating hereditability for neuroticism and extraversion (and psychoticism) at approximately 50%, while agreeableness and conscientiousness showed little or no genetic transmission (with environmental experiences appearing relatively more important). Extraversion appeared to have two components (impulsivity and sociability), each with hereditabilities of about 60%, but only partially sharing genetic antecedents.

As summarized by McGuffin and Thapar (1992), for traits assessed by questionnaires (e.g., extraversion, neuroticism), hereditabilities of 35-50% are reported. The seeming invariability of moderate hereditability estimates using self-report methodologies (whether completed by the child or by the parent) concerned Plomin (1982), who established that ubiquitous hereditability was no longer evident when objective observations were used. Noting that the most direct estimate of genetic influence is the correlation for identical twins reared apart, "because the correlation itself directly estimates hereditability," Plomin et al. (1988) examined ratings of early childhood by separate groups of elderly twins (MZ and DZ, reared together and reared apart). The correlations for identical twins reared apart were significant (0.33–0.41) for cohesion, achievement, active, cultural, and conflict, while low hereditability was suggested for control and organization. Thus, the authors concluded that there is a genetic influence on perceptions of childhood environment. Such artefactual influences must be conceded in estimating the hereditability of temperament and personality, and argue for the prioritizing of behavioral as against self-report measures, and for independent reporters or observers.

In addition to twin studies, genetic influences can be estimated from adoption studies. According to Carey and DiLalla (1994), conclusions correspond to those derived from twin studies of personality traits (with genetic factors dominant and common environment having only a small effect).

RELEVANCE TO ETIOLOGICAL STUDIES

If the tripartite model put earlier is valid, we might expect higher heritabilities to be demonstrated for dimensions of temperament than for dimensions of personality. Impressionistically, and as suggested by data in the preceding paragraphs, heritabilities appear to be of the same magnitude. This may be a false impression, and reflect failure to define and distinguish the integral and separate dimensions of temperament and personality. If a true impression, it may indicate that temperament is synonymous with personality, and that semantic differentiation of the two is sterile. Alternately, that personality, as the epigenetic burgeoning of temperament, is as capable of being influenced by genetic determinants and environmental modifiers as is temperament. It was put earlier that temperament can be conceptualized as the "hard wiring" but that does not mean that temperament is necessarily immutable. We now recognize that early social deprivation may affect neuronal development. Thus, Martin et al. (1991) examined rhesus monkeys reared without social contact in the first year of life, and described a range of abnormal behaviors and "psychosocial abnormalities."
At post-mortem, there was altered chemoarchitecture of some basal ganglia regions, and the authors concluded that “the postnatal maturation of neurotransmitter phenotypes in some structures is influenced by social environment” (p. 334). It is thus conceivable that hard-wired temperament may be as susceptible to developmental factors as is personality.

THE DISORDER COMPONENT OF PERSONALITY DISORDER

Livesley et al. (1993) stated that they assessed the hereditability of the “basic dimensions of personality disorder” in a sample of 175 twin pairs (90 MZ and 85 DZ). Their measure (the Dimensional Assessment of Personality Pathology—DAPP) is a 290 item, self-report measure assessing 18 dimensions. The labeling of many of the dimensions (e.g., conduct problems, intimacy problems) suggests a greater weighting to disorder rather than merely to the dimensional extreme of many personality traits. Genetic influences were suggested for all bar conduct disorders, with 12 of the 18 dimensions assessed having hereditabilities ranging from 40–60%, quantitatively akin to data reported generally for personality trait studies.

PERSONALITY DISORDER CLASSES

Any genetic contribution is usually pursued by three principal research paradigms—family studies (FSs), twin studies (TSs), and adoption studies (ASs). Dahl (1993) has provided a useful summary of work published through the early nineties (although several additional studies have since been published), illustrating the paucity of such strategies being applied to formally classified PD classes. Thus, only five of the PDs (i.e., schizotypal, borderline, histrionic, avoidant and dependent) have been examined using a FS strategy, two (schizotypal and borderline) by a TS, and two (antisocial and histrionic) by an AS strategy. The flavor of such research can thus be readily summarized.

In relation to FSs, Reich (1989) suggested an increased risk in relatives of probands for dependent PD. While no study has been undertaken for obsessive-compulsive PD, related analyses indicate the possibility of an obsessional spectrum being over-represented (Nigg and Goldsmith, 1994). Tarnopolsky and Berelowitz (1987) interpreted several studies of borderline personality as indicating an over-representation of borderline personalities in their relatives. Ruegg and Frances (1995) reviewed ten recent studies, which suggested that, in those with a borderline PD, the relatives had a seven-fold increase in the prevalence of antisocial PD (as against a two-fold increase in substance abuse and depression), speculating both sharing of the same inborn temperament as well as environmental factors. Rutter (1987) noted several FSs showing a relatively strong association of schizotypal PD with schizophrenia, and of paranoid PD being somewhat more strongly associated with delusional disorders than with schizophrenia. In a more recent and comprehensive review, Nigg and Goldsmith (1994) held that there was consistent evidence that schizotypal and paranoid PD are both more likely in relatives of schizophrenic probands. McGuffin and Thapar (1992) therefore invoked a severity-liability (or continuum) model for considering the relationship between schizophrenia and schizotypal PD.
Turning to TSs. McGuffin and Thapar (1992). Dahl (1993). and Nigg and Goldsmith (1994) have all provided useful reviews. Among representative studies. Torgersen (1984) provided data to suggest that schizotypal PD breeds true. with schizotypal disorders in 33% of MZ twins of schizotypal patients. compared to a 4% rate in DZ twins. While recognizing that criminality and sociopathy do not necessarily equate. McGuffin and Gottesman (1984) pooled seven twin studies of adult criminality and five studies of juvenile delinquency— concordance for adult criminality was 51% in MZ and 22% in DZ twins. but there was little difference in the juvenile delinquents (87% vs 72%). Torgersen (1984) interpreted a small set data to suggest that while borderline PD may be familial. genetic transmission was not clear.

Finally. there are the ASs. Rutter (1987) has suggested that schizoid PD shows stronger relationships with childhood autism (and Asperger's syndrome) than to schizophrenia. Grove et al. (1990) used the Diagnostic Interview Schedule in a study of 31 MZ twins reared apart and quantified hereditability of adult antisocial PD at 19%. Dahl (1993) considered two adoption studies of adults with antisocial PD. Conclusions were in line with ones put by McGuffin and Thapar (1992). who reviewed adoption and cross-fostering studies for delinquency and antisocial PD. and held that there was consistent evidence of a genetic contribution (more to petty than to violent crime). and that family background contributed—but only when there is a genetic predisposition.

**HEREDITABILITY MEANS WHAT?**

Any demonstration of hereditability offers little information on mechanisms. As noted by Nigg and Goldsmith (1994). high hereditability does not mean that "genes control behavior deterministically," as there may be numerous opportunities for environmental modification of gene activity. Thus. Rutter et al. (1990) suggested that. in addition to direct effects. possibilities include influences on temperament. vulnerability to stress. and response patterns to environmental stimuli. as well as the capacity to shape the environment. Of the three childhood temperament clusters (ie easy. slow to warm up. difficult). the last presents a high risk to the development of behavioral problems—but this is generally held to be a consequence of poor goodness of fit. with aspects of the sociocultural environment being difficult for the child to deal with and adapt to (Kashani. Ezpeleta. Dandoy. Dol. & Reid. 1991).

There have been strikingly few biological studies seeking to define possible contributing or mediating biological variables. In an heuristic model. Cloninger (1986) has proposed three mediating monoaminergic transmission systems as underpinning key dimensions of temperament (i.e., serotonergic for harm avoidance. noradrenergic for reward dependence. and dopaminergic for novelty seeking). Turning to narrow biological studies. the few reports are difficult to interpret. as positive findings are rarely replicated. while co-morbidity of PD and Axis I disorders in many of the studies makes interpretation difficult. These points are evident in considering the
review by Weston and Siever (1993), who reviewed the biological correlates of PDs by focussing on data in relation to the three DSM-III-R clusters.

As an example, however, of both focussed biological research and of a finding that has been replicated, Reist, Haier, De Met, and Chicz-De Met (1990) studied platelet MAO activity in 28 males with adjustment disorder and a co-morbid PD (usually borderline PD). Borderline subjects had lower MAO levels compared to controls, but did not differ when compared against other PDs, while platelet MAO activity had an inverse relationship with scores on a sensation seeking scale, suggesting that MAO activity may be a marker of impulsiveness—an important personality component of many PDs.

STUDIES ASSESSING ENVIRONMENTAL INFLUENCES

GENERAL INTRODUCTION

Genetic analyses (especially those using twin and adoptee paradigms) may provide information on the contribution of both shared and nonshared environmental factors. Examples of shared environmental factors include childhood poverty and social class, while one child’s exposure to a head injury, or differential parental care, might be an example of nonshared environmental factors.

Shared environmental variance can be estimated from the correlation between genetically unrelated children adopted together, and such influences have been most clearly demonstrated (according to Rutter & Rutter, 1993) for delinquency. Those authors noted a study by McGuffin and Gottesman, with concordance being 87% for MZ and 72% for DZ twins, suggesting a slight genetic component, but with the high DZ concordance pointing to a major shared influence (be it parental criminality, family discord, ineffective supervision, etc.).

Caveats have been expressed about interpretation of these analytic strategies. Plomin and Bergeman (1991) hold that there is evidence of genetic influences on many so-called environmental measures, including social class, social support, and—importantly for our considerations, life events—with heritability estimates of 43% for controllable and 18% for uncontrollable events. Thus, they claimed that many measures labeled and analyzed as environmental are, in fact, genetically influenced.

Plomin (1994) also articulated another important research finding: “the salient environmental influences are not shared by children growing up in the same family” (p. 823). He argued that environmental influences on behavioral development (or individual differences between family members) are largely of the non-shared environment. If valid, variables shared by children (e.g., parental divorce) would not contribute to individual differences, and the empirical research finds little evidence of shared environment contributing to personality and temperament dimensions (apart from conservatism). Instead, nonshared, nonfamilial factors tend to account for a considerable percentage of the variance. But does the interpretation hold for the disorder component of personality disorder? Reference will again be made to the Livesley et al. (1993) study which rated 175 twins on a measure
of personality pathology, and which identified a number of dimensions weighted more to disorder. Apart from ones assessing conduct problems and submissiveness (where there was a significant shared environmental contribution), nonshared environmental influences were dominant for most of the 16 remaining dimensions.

WHAT ENVIRONMENTAL FACTORS SHOULD BE CONSIDERED?

Fonagy, Steele, Steele, Higgitt, and Target (1994) have listed a large number of variables associated with childhood resilience, including certain parenting variables (e.g., competent parenting; at least one warm parent; nonexposure to parental separation); higher social class; better education; high IQ; age and sex (i.e., prepubertal for females and postpubertal for males); absence of organic deficits or, if any trauma, a younger age when exposed; social support in adulthood; easy temperament; good problem-solving abilities; locus of control; high self-esteem; interpersonal awareness; empathy; superior coping; capacity to plan; and sense of humor. Excluding likely temperament and personality variables, we are left with a relatively short list of likely converse factors for overview.

SOCIODEMOGRAPHIC FACTORS

There have been remarkably few studies considering their relevance, so only a brief comment is made here. In DSM-IV (APA, 1994), onset in childhood is noted for two PDs (i.e., antisocial and avoidant) and conceded in four others (i.e., paranoid, schizoid, schizotypal, and borderline); a male preponderance is described for paranoid, schizoid, antisocial, narcissistic and obsessive-compulsive, and a female preponderance for borderline and (possibly) dependent PD, as against a nearly equal sex ratio for avoidant and histrionic PD; and a lower social class weighting for antisocial PD.

Variable ages of onset and remission, as well as variable courses, might indicate differing etiological factors, such as the maturation or lessening of certain at risk underpinning temperament and personality dimensions. Tyrer, Casey, and Ferguson (1991) have suggested that two groups can be discerned - PD classes that show little variation with increasing age (i.e., obsessive-compulsive, paranoid, schizoid, schizotypal, and anxious) and ones tending to improve over time (i.e antisocial; borderline; histrionic; dependent; and narcissistic), with the two groups labeled mature and immature respectively. Stone (1993) reviewed a number of studies, and suggests that outcome is generally favorable for borderline PD after 10-30 years; that there are two broad outcomes for those with sociopathy (improvement more likely in those showing adolescent delinquency of a milder type but being less likely in those with childhood and severe adolescent expressions); that schizoid and schizotypal subjects tended to remain isolated and lead marginal lives; and that outcome data on the remaining PDs were lacking. If, however, any PD ameliorates with age, there is a need to identify whether it is a reflection of maturity, a reduced need to express certain personality characteristics, accommodation to the environment, or explainable by other factors.
ETIOLOGY OF PD

ORGANICITY

All forms of neurological insult (e.g., head injury, post-encephalitic damage) are held to increase the chance and severity of PDs, but most of the focus has been on child conduct disorder and adult sociopathic PD. Attention deficit disorder (with or without hyperactivity), as well as non-specific EEG changes, are over-represented in sociopathic PD. The evidence (see Watkins, 1994) appears to suggest that, when conduct disorder (CD) and hyperactivity are comorbid, the hyperactivity precedes and predisposes to CD, and it is only the CD that leads to later antisocial PD.

PARENTAL FACTORS

Clearly, parenting (more than any other formative socializing experience) has been implicated in a number of etiological frameworks for the PDs. Analytic theories, neoanalytic theories, or even family systemic theories will not be reviewed, as none have been empirically tested at a meaningful level. Nevertheless, some consensus is evident in any thick description of the PD literature. For instance, parental factors have been implicated strongly and consistently for certain PDs (e.g., borderline, antisocial, obsessive, and dependent) and less so for paranoid and schizoid PD. Second, whether formulated by analysts, attachment theorists, or family therapists, parents who infantilize their child, foster its dependency, or otherwise counteract the child’s separation-individuation process are implicated in the genesis of dependent PD. However, even if such parents had all such attributes and characteristics, it would not mean of necessity that they are causal factors, when a dependency gene might influence both such parenting and dependency in the child.

In relation to obsessive-compulsive PD, a theme of parental control is evident, with analysts imputing its origins in the parents being overly rigid and punitive in toilet training during the child’s anal stage. A similar thematic view was put by Millon (1981)—exposure to overcontrolling parents who punished disapproved of behaviors and autonomy. Such a causal postulate may have validity, or a higher-order genetic variable may instead induce both controlling parenting and orderliness and perfectionism in the child. In support of the latter, Adams (1973), for one, established that parents of children with obsessional personality style were more obsessional.

Anomalous parenting may be assessed in a variety of ways, ranging from measuring disturbances of fundamental parental characteristics through exposure to particular instances such as death, separation, violence, or incest. The first strategy has been addressed in a number of studies by use of the Parental Bonding Instrument, or PBI (a self-report measure of care vs. indifference/rejection and overprotection vs encouragement of independence), and with established reliability and validity (see Parker, 1983). Table 1 lists published controlled studies using the PBI. In essence, the following has been reported: (a) lower parental care—but no suggestion of overprotection—for those with DSM-III-defined avoidant PD and patients with schizotypal PD; (b) distinctly lower parental (and especially paternal) care, and somewhat greater protec-


TABLE 1. Published Controlled Studies Reporting PBI Data for Those with a Personality Disorder

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<td></td>
<td></td>
<td>Care</td>
<td>Protection</td>
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<td>18</td>
<td>Pt</td>
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<tr>
<td>Paris &amp; Frank, 1989</td>
<td></td>
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<td>25.2</td>
<td>14.4</td>
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<tr>
<td>Zweig-Frank &amp; Paris, 1991</td>
<td>62</td>
<td>Pt</td>
<td>20.4</td>
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<tr>
<td>Byrne et al., 1990</td>
<td>15</td>
<td>Pt</td>
<td>16.3</td>
<td>18.8</td>
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<tr>
<td>Torgersen &amp; Alnaes, 1992</td>
<td>41</td>
<td>Pt</td>
<td>17.1</td>
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<td></td>
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<td>Cont1</td>
<td>24.7</td>
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Schizotypal

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<td>Torgersen &amp; Alnaes, 1992</td>
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<tr>
<td>DSM-III-R clusters</td>
<td>7</td>
<td>Cluster A</td>
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<tr>
<td>Paris et al., 1991</td>
<td>60</td>
<td>Cluster B</td>
<td>20.7</td>
<td>18.1</td>
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<tr>
<td></td>
<td>42</td>
<td>Cluster C</td>
<td>23.9</td>
<td>16.5</td>
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<td></td>
<td>54</td>
<td>Cont</td>
<td>24.8</td>
<td>13.9</td>
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Avoidant

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<td></td>
<td>Parental care = 16.5</td>
<td>Protection = 15.10</td>
<td></td>
</tr>
<tr>
<td>Stravynski et al., 1989</td>
<td>15</td>
<td>Parental care = 23.7</td>
<td>Protection = 14.4</td>
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Note. N Controls were 1 patients without PD; 2 US normative data; 3 US control sample. ITALICs, significant difference.

The possibility of specificity (of anomalous parenting to certain PDDs) is important. In another research pursuit of depressive disorders, PBI-defined affectionless control was demonstrated to be specific to the nonmelancholic depressive disorders, with melancholic depressed subjects (both unipolar and bipolar) returning PBI scores akin to control samples (Parker, 1992).

Many (e.g., Rutter, 1987) have emphasized the importance of lowered self-esteem and compromised self-efficacy to PD development. In our PBI research (see Parker, 1983), we have demonstrated links suggestive of low parental care disposing to low self-esteem, together with parental overprotection delaying or inhibiting the child's separation-individuation process and restricting its social competence. As a PBI-type methodology has...
ETIOLOGY OF PD

potential to allow mediating mechanisms to be pursued, it would be useful (for those PDs where a parental contribution appears to be causal) to determine the extent to which PBI scores relate to the stem personality of the PD or, instead, to the disorder component.

PARENTAL SEPARATION

Such an event is often noted in the background of those with PDs (especially sociopathic PD), but its relevance and specificity as a risk factor has not been quantified. We can speculate that, depending on the nature of the separation (i.e., trivial vs. profound; temporary vs. permanent), it may be substantive or, at times, an epiphenomenon. While frequently imputed as relevant to the etiology of antisocial PD, it may be only an expression of a more likely ongoing risk variable—dysfunctional parenting as defined in the previous section. The message of confounding by higher-order variables needs to be respected in relation to the etiology of PD.

SEXUAL ABUSE

Ruegg and Frances (1995) reviewed recent research examining the prevalence of sexual abuse in those with PD. Rates were high, most frequently associated with borderline PD, but studies were uncontrolled and possibly influenced by studying those in treatment. The authors noted that associations may not be causal (i.e., genetically-driven impulsivity might influence the perpetrator’s behavior and the child’s PD).

A SYSTEMS PERSPECTIVE

To assist theorizing, we often consider expressions of anomalous parenting as unidirectional causal factors. It must, however, be rare for parenting to be unidirectional, being influenced to varying degrees by characteristics of the child, so that iterative processes must be conceded. That reality may be addressed by adopting family system theories, or related models. As reviewed by Wachtel (1994), the family communications and behaviors compose many circular loops or recursive patterns, with psychological difficulties being understood in terms of how each party punctuates what is actually a seamless flow. The capacity of statistical analytic strategies to partition out any causal contribution of parent, child, and parent–child factors to the etiology of PD in such bidirectional interchanges is clearly limited.

While we can concede the issue of iterative and/or cascading contributing events, empirical delineation is absent in studies pursuing the etiology of PDs. Rutter and Rutter (1993) have well described the theoretical complexities—how the child’s temperament might lead directly to disorder, or indirectly, such as by eliciting negative interactions from others or predisposing to stress situations; or by providing increased susceptibility to psychosocial adversity. Thus, they noted that “it is exceedingly uncommon for early and late experiences to be independent of one another,” as “people’s behaviour shapes their environment” and
"early experiences tend to determine later experiences," so that adversities may persist although environmental stresses change, leading to "substantial continuity in advantage/disadvantage" (p. 33).

CULTURE

Cultural factors can provide an ecological niche for the expression and accentuation of certain PDs. Thus, differential frequencies of those with an antisocial PD, histrionic PD, or schizoid PD might readily be anticipated in those attending a Hells Angels barbecue, an Academy Awards presentation, and a Dungeons and Dragons convention, but any such specificity would presumably reflect assortative mating rather than an etiological cause. Such cultural "endorsement" might well enhance the expression of the PD.

Can, however, cultural influences cause PDs? As posed by an assessor of this article, "are there particular historical eras or economic circumstances that help produce a greater number of PDs at a particular time?" Thus, would the survivors of the great depression not be more frugal, parsimonious, conscientious, and respectful to an employer? Again, would not membership of a particular religious group that emphasised ritual, penance, and activities such as self-flagellation, dispose to an obsessive-compulsive style and a compulsive PD? Millon (1996) has emphasised how cultural forces act as abstract formative influences on a social group. He recognized that commentators have brought slogans such as "the age of anxiety" and "the lonely crowd" to characterise conditions of American cultural life, and then noted some components of the culture contributing to such definable expressions of personality pathology.

However, in the absence of any clear and direct link demonstrated between culture and PD status, we are left with doing no more than conceding its relevance as having causal potential but, more importantly, serving as a context (Millon, 1996) within which varying PDs may be less readily or more readily expressed.

Thus, we need a social systems perspective—recognizing that the developing child is influenced by parents, family, social context, broader society, cultural, and political influences, etc.—with complex inter-relationships. The capacity of statistical analyses to address such complexities is limited, and the task must be simplified. Perhaps, rather than attempting to define or partition the contributing components, it might be more useful to define and examine "thick risk" disjunctive variables (e.g., the extent to which there is environmental fit between the child and the parents), as well as between the child and other signal variables (e.g., peers, school, and cultural influences).

Such an approach may provide salient information on the defining experiences that promote disjunction between temperament and an individual’s failure to find an ecological niche. Conversely, if an individual’s temperament both shapes the environment and drives any continuity of disadvantage, then an appropriate measure of that temperament will provide a thick risk variable for examination (and partialling out) in multivariate analyses. Thus, such research might identify those PDs that
are (a) seemingly weighted to temperament, (b) a product of temperament and disjunctive life experiences, and (c) shaped largely by developmental stressors and independent of temperament. In addition, such an approach might allow identification of those contexts producing problematic outcomes for those with particular temperament and personality dimensions.

HOW SHOULD WE CONCEPTUALIZE ETIOLOGICAL VARIABLES AS EXERTING THEIR EFFECT?

A number of possible models (and as outlined by Junker and Pilkonis, 1993) should be conceded for etiological studies, including: (a) a common cause model—that different PDs are determined by the same underlying process, be it genetic or developmentally focussed; (b) a spectrum model—meaning either that that two or more PDs, or Axis I and PD disorders, are related (i.e., in sharing etiological risk factors), but differ in their overt manifestations; (c) a pathoplasty or exacerbation model—certain PDs are distinct, but when coterminal, additive effects or interactions between their distinct features influence presentation but not the risk of either (i.e each can affect the other's manifestations); (d) a predisposition model—one PD or Axis I disorder precedes and increases the risk of another PD; and (e) a complication or scar model—a PD develops as a consequence of an initial PD or Axis I disorder.

PURSUING THE ETIOLOGY OF THE PD'S: QUO VADIS?

INTRODUCTION

In the absence of firm definitions and discrimination of the PDs as valid and distinct classes, together with the absence of reliable measures, research paradigms are clearly restricted. Several possible approaches will be noted.

[A] A “Top Down” Approach. Here, currently accepted PD classes might be regarded as the criterion measure. There may be some wisdom in having clinicians nominate patients judged as being representative of pure PD classes for interviews with researchers. The clinicians and researchers would independently rate those subjects on (a) sets of PD descriptors, (b) the extent to which they met broad PD class diagnoses (to allow for co-morbidity), (c) their interpersonal style, and (d) operationalized measures of disorder. The patients would complete equivalent self-report measures, as well as measures of personality and temperament. Research psychiatrists might undertake a clinical interview (blind to diagnostic data) and prepare a developmental formulation, while parents would be interviewed to (a) obtain ratings on the early expression of the child’s temperament and personality, as well as data on exposure to relevant risk variables, and (b) return corroborative witness data on the subject’s adult expression of personality, temperament, and personality disorder.

Analyses would (a) examine relevant inter-rater reliability issues, (b)
assess the extent to which criterion diagnoses were predicted by measures of temperament, personality, disorder and (subsequently defined) latent taxa of PD classes, (c) consider whether a criterion diagnosis of PD may be validly reached in a one-stage procedure (as current), or would require a two-stage procedure involving assessment of both personality style and extent of disorder, (d) identify the relevance of a range of developmental stressors, and their specificity to both particular PD classes and to personality style vs. disorder, and (e) seek to refine the descriptors of the criterion diagnostic classes.

[B] A “Bottom Up” Approach. Here the data base derived in Study A would be reanalysed, but with the criterion measure no longer being a DSM-type class diagnosis. Instead, analyses would seek to derive the substantive latent taxa (which drive the surface markers that currently define PD classes). The developmental data derived in the study would then be examined then as predictors of the latent taxa.

[C] A Cohort Study. A large number of studies have been undertaken where children have had detailed assessment of defined temperament dimensions. The suggestion is to restudy such children in adulthood, and examine the longitudinal predictability of the data collected in childhood—of adult temperament and personality, as well as of PD status. Developmental data would be derived, to identify those environments that might modify any diathesis to adult PD status, and to pursue the independence or inter-relationship of temperament and environmental factors. The objective here is to overcome a limitation of the tripartite model—that, while it may be useful to determine specific influences on separate temperament, personality, and disorder components, it remains conceptually unsatisfying if the different strands cannot be synthesised and integrated. There is then a need to follow the pathways from temperament to disorder, and identify in which contexts certain personality dimensions become problematic. Such cohorts may provide an efficient vehicle, subject to assessment being frequent and regular over time.

Such a study would respect the tripartite model articulated earlier—for reasons previously stated and for one additional one. Much etiological theorizing has probably been based on select groups of those with PDs—those with the greatest social dysfunction, or those in receipt of services and psychiatric attention. Studies, for instance, of antisocial PD, have generally been undertaken on such select groups, risking a weighting to those who have failed rather than succeeded with their antisocial PD, and perhaps then excessively prioritized certain etiological factors such as gross parental neglect. The etiology of “successful sociopathy” may relate more to factors contributing to plausibility, glibness, inflated self-appraisal, and lack of remorse. Thus, such a study would seek to define etiological factors contributing to the stem disorder (here antisocial personality) and, secondarily, factors contributing to the disorder and thence to its success or failed expression in the social milieu.

[D] Spectrum Disorder Studies. Millon (1986) has suggested that Axis I symptom disorders “may best be conceived as the upshot of a dynamic interaction between Axis II (personality) and Axis IV (stressors).” If valid, it allows etiological research to be pursued via dimensions that might
underlie some assumed continuum across Axes I and II. A number of models have been articulated. For instance, Vize and Tyrer (1994) suggested that “three dimensional spectra of mental state and personality can be identified fairly clearly: schizophrenias with eccentric personalities; substance misuse with flamboyant personalities and neurotic disorders with anxious and dependent personalities.” Again, Siever and Davis (1991) have proposed that four psychobiological processes (i.e., cognitive organization, affective regulation, impulse control, and anxiety modulation) are core substrates underpinning Axis I and II disorders. The authors held that validation of such a dimensional model could occur by pursuing such dimensions of psychopathology (e.g., by family history, biological correlates, treatment response, and clinical course), and offered several hypotheses. Thus, (a) for cognitive/perceptual organization, tests of higher cortical organization and abnormalities in dopaminergic function; (b) for impulsivity/aggression, pursuit of reduced serotonergic activity and symptom response to serotonergic drugs; (c) for affective instability, abnormalities in cholinergic and catecholaminergic function and symptom response to cholinergic and adrenergic challenges; and (d) for anxiety/inhibition, no clear biological candidates, but GABAergic and noradrenergic systems considered for exploration.

Pursuing the Nature of Treatment Response. While Klein (1985) has warned that migraine is not necessarily due to an insufficiency of aspirin, clinical psychiatry does favor a paradigm that organically driven conditions tend to have a preferential response to physical treatments, while environmentally driven conditions are more susceptible to non-physical treatments. Thus, the efficacy of methylphenidate for many with attention deficit disorder was persuasive evidence for an organic etiology.

The demonstration of any effective drug therapy for any PD might also argue for an organic etiology, although more parsimonious explanations would surface (e.g., that the condition was not a true Axis II disorder, as has been held, say, for avoidant PD being a synonym for social phobia—or in considering the overlap between borderline PD and affective disorder (Gunderson & Elliott, 1985). What do we know about the efficacy of physical treatments for the PDs? DeBattista and Glick (1995) noted that the pharmacotherapy of PDs has been slow but steady in recent years. They found some suggestion that the SSRIs may assist the impulsiveness, self-mutilation and overt aggressiveness observed in some PDs, that those with a schizotypal PD may obtain some benefit with antipsychotic and antidepressant drugs, and they noted an important negative study from the Western Psychiatric Institute which failed to find support for the use of haloperidol or phenelzine in those with a borderline PD.

Thus, progressive moves to respect spectrum conditions and to favor dimensional models for conceptualizing and measuring PDs suggest that there might be some wisdom in not merely restricting etiological research to pure Axis II PD groups. If a risk factor (e.g., a genetic influence) is identified, there would be a need to pursue the sequencing (i.e., is the PD causing Axis I disorder: is the Axis I disorder causing the PD: or do higher-order variables, such as genetic factors, have direct causal effects on PD and disorder - causal pleiotropism).
CONCLUSIONS

It is clear that our understanding of etiological influences on the PDs is limited, and in contrast with other major psychiatric disorders. Progress is likely to benefit from approaches which improve on current class-based approaches to conceptualizing and measuring the PDs. In this article, an alternate tripartite model for conceptualizing the PDs is explored, while several strategies for pursuing etiological research are described.

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**CALL FOR PAPERS**

For Special Sections of *Psychotherapy Research*

In an effort to explore timely issues in the field, the editors of the journal *Psychotherapy Research* have issued a call for papers dealing with the following topics:

**Research on Psychotherapy with Serious Persistent Mental Illness.** In general, psychotherapy research has ignored client populations recovering from psychotic episodes or coping with bipolar or other severely debilitating psychological difficulties (including severe forms of personality disorder). This special section is an attempt to redress this limitation. (Deadline for submission: December 31, 1998.)

**Research on Time and Psychotherapy.** Pressure from governments or insurance companies to contain mental health care costs has generated strong interest in time-related variables. For this special section, we are interested in research on long-term treatments, or conversely, very brief treatments, as well as dose-effect relationships, growth curve analysis, survival analysis etc. (Deadline for submission: June 30, 1999.)

All manuscripts should be submitted in quadruplicate (with Fax and E-mail information). Manuscripts from outside North America should be sent to: Bernhard Strauss, Klinikum der Friedrich-Schiller-Universität, Institut für Medizinische Psychologie, Stoystrasse 2, D 07740 Jena, Germany. North American manuscripts should be sent to: Robert Elliott, Department of Psychology, University of Toledo, Toledo, OH 43606, USA.
Measuring disordered personality functioning: to love and to work reprised


Objective: Current limitations to diagnosing and measuring the personality disorders encouraged a set of studies seeking to provide an alternate approach to modeling and measuring disordered personality function.

Method: A large set of self-reported descriptors of disordered personality function were factor analyzed in a sample of patients with clinician-diagnosed personality dysfunction, generating 11 lower-order and two higher-order constructs. Subjects and non-clinical controls also completed a measure of personality styles underpinning formalized personality disorder groupings. Properties of the refined self-report (SR) measure were assessed in an independent sample of patients with a clinically diagnosed personality disorder.

Results: Limitations in ‘cooperativeness’ and ‘coping’ formed the higher-order constructs defining disordered personality function, with these constructs relevant to all personality styles. Analyses of SR, corroborative witness (CW) and clinician-rated data in an independent sample supported measuring disordered personality function by our derived 20-item SR measure, and exposed limitations to clinician-based assessment.

Conclusion: Study findings build to a multi-axial strategy for measuring personality disorder, involving separate dimensional assessment of both disordered personality function and of personality style.

Introduction

‘Perhaps the word diagnosis can be eventually dispensed with in favor simply of assessment of functioning’ (1).

Individuals satisfying diagnostic criteria for one personality disorder (PD) commonly satisfy criteria for several other PDs (2, 3). This can reflect true comorbidity or inability of clinicians and classification systems to distinguish accurately between distinct PDs. Current diagnostic descriptors for PD assess both disordered function (DF) and personality style, with descriptors of DF generally broad and non-specific, risking further confounding. Thus, if several PDs include a particular aspect of functioning, such as impulsivity, then its presence will build to comorbidity even if the associated-personality ‘styles’ are truly distinct.

Difficulties in diagnosing and measuring the PDs validly have encouraged an argument that personality style and DF should be measured independently (4, 5). This would clarify whether most or all PD personality styles are associated non-specifically with DF, or whether each personality style shapes specific disordered functioning.

Another argument for measuring DF as an independent construct is to assist definition of PD status. DSM-IV and ICD-10 criteria for personality disorders emphasize personality traits. However, such traits may be intrinsically maladaptive, or only maladaptive above a certain imprecise threshold or in specific contexts.

Aims of the study

To define PD primarily on the basis of DF, and then assess personality style as a secondary
Measuring disordered personality functioning

Material and methods

Measures

Disordered function (DF). From our detailed literature review (6) we identified 17 constructs historically defining DF such as ‘instability under stress’, ‘impulsivity’ and ‘inflexibility’. A total of 141 descriptors were written for the constructs [in a self-report (SR) format], and reviewed by experienced clinicians in terms of content, relevance and ambiguity. We administered the descriptors to two non-clinical samples, totaling 146 subjects, to reduce the item set to a more practical number. A provisional factor analysis identified ‘non-cooperativeness’ and ‘disagreeableness’ constructs as synonymous, ‘maladaptability’ and ‘lack of humor’ had only one loading item each, while the construct ‘failure to form and maintain relationships’ did not generate a discrete factor. Items for the last three constructs were deleted together with items quantified as synonymous with other items (correlations of more than 0.70). A reduced set of 82 items generated our current study’s DF measure. Each item allowed four responses, ‘definitely false’, ‘mostly false’, ‘mostly true’ and ‘definitely true’, scored dimensionally (0, 1, 2, or 3 respectively).

Personality style (STYLE). A previous study (7) developed descriptors of 15 formalized PDs, including all those listed in DSM-IV and in ICD-10 (paranoid, schizoid, schizotypal, narcissistic, antisocial, borderline, histrionic, avoidant, dependent, obsessive and depressive), some previously listed in diagnostic manuals (passive-aggressive, self-defeating, sadistic) and an ‘anxious’ PD category. Analyses of large SR and observer-rated databases generated the 142-item STYLE questionnaire measuring personality style scores for each of the 15 putative PD categories. Each item allowed four responses, ‘definitely false’, ‘mostly false’, ‘mostly true’ and ‘definitely true’, scored dimensionally (0, 1, 2, or 3 respectively).

Global judgment of disordered personality function (GLOBAL JUDGEMENT). This measure requested clinicians to ‘rate the extent to which the individual’s personality style and function are such that they and/or others around them suffer as a consequence’. Options allowed ‘no’, ‘slight’, ‘moderate’ and ‘distinct’ limitations to function.
Parker et al.

Functional domains (DOMAIN). This measure required clinicians to rate the subject’s function across five ‘domains’ (work, and intimate, peer, family and work relationships), with four-point scales providing options of ‘functional’, and ‘possibly’, ‘probably’ or ‘definitely’ dysfunctional. Summed scores generated a total DOMAIN score.

Clinician-rated personality disorder style (PD VIGNETTE). This measure required clinicians to rate the extent to which the patient’s personality style corresponded to succinct descriptors of each of the 15 PD categories (i.e. ‘not at all’, ‘to some degree’, ‘reasonably well’ and ‘completely’).

Samples

Clinical sample. We approached some 50 psychiatrists to recruit suitable patients. Eligibility criteria were: (i) clinician-judged presence of either a PD or significant personality traits affecting function; (ii) age 18–65 years; (iii) not currently having a psychotic disorder, mania, severe depression, significant cognitive impairment or a distinct drug or alcohol problem; and (iv) competence in English. The psychiatrists completed GLOBAL JUDGMENT, DOMAIN and PD VIGNETTE measures for eligible subjects and invited them to complete an anonymous SR questionnaire (for their return by pre-paid mail) collecting demographic information as well as DF and STYLE measure data.

We recruited 249 subjects referred by 33 psychiatrists. The study strategy, respecting patient anonymity and choice in returning questionnaires, disallowed formal determination of the response rate.

Control sample. Research staff were asked to select friends and close colleagues judged over time as not showing any evidence of a PD. Invitees were requested to complete the STYLE and DF measures for return by pre-paid mail. To protect anonymity we did not record any identifying details, and did not determine the response rate. This sample consisted of 67 subjects.

Measure validation sample. This next study was designed and initiated after completing data analyses of the clinical and control samples. Hospital patients meeting the same criteria as for the clinical sample were requested to: (i) complete the DF measure; (ii) have a relative or friend complete a corresponding observer-rated DF measure ‘best describing’...how X...usually or generally feels or behaves, as they have been over the years, and not just recently’ (returned directly to the research team); and (iii) be interviewed by two clinical research staff.

The two raters, a psychiatrist (SK) and a psychologist (AO), administered a semistructured interview based on the Adult Personality Functioning Assessment measure (8), assessing lifetime functioning across six domains: work, intimate relationships, relationships with family and friends, work relationships, non-intimate social contacts, and coping and negotiating styles when stressed. Using six-point dimensional scales with structured anchor points, each rater independently assessed the extent to which the patient evidenced ‘cooperativeness’ and ‘effectiveness’ across each domain, with summed scores generating total interviewer-rated ‘non-cooperativeness’ and ‘non-coping’ scores respectively. Thirty-five subjects (and 26 corroborative witnesses) were recruited.

Results

Refining the constructs to develop a model of disordered functioning

Clinical sample members had a mean age of 39.1 years (SD 11.6 years), a mean of 15.0 years (SD 3.6 years) of education, and a slight female preponderance (52%). Only 27% were married, 18% divorced or separated and 0.4% widowed. Just over one-half were in full-time or part-time work, 13% were unemployed, 18% pensioners, 7% undertaking home duties and 6% students.

STYLE scores for respondents were converted to percentage scores for each putative PD category to allow standardized prevalence estimates. A score of 100% for a PD would represent all respondents scoring 3 (i.e. ‘definitely true’) for every item relevant to that PD. Percentage scores were: depressive (54%), anxious (53%), obsessive (48%), avoidant (41%), borderline (40%), schizotypal (38%), self-defeating (37%), schizoid (37%), paranoid (36%), dependent (35%), histrionic (34%), negativistic (29%), antisocial (28%), narcissistic (26%), and sadistic (20%). Males scored higher ($P < 0.05$) on the narcissistic scale and females scored higher on the borderline, anxious and depressive scales. There were no sex differences for the remaining STYLE scores.

We compared relevant SR STYLE and psychiatrist-rated PD VIGNETTE scores. Pearson correlation coefficients were: borderline (0.46), depressive (0.36), paranoid (0.35), avoidant (0.32), anxious (0.31), schizoid (0.28), passive-aggressive (0.27), narcissistic (0.27), dependent (0.24), histrionic (0.23), sadistic (0.20), antisocial (0.20), self-defeating (0.19), schizotypal (0.18) and obsessive (0.07). A
mean coefficient of 0.26 across the 15 PD categories suggested only slight overall agreement between subjects and psychiatrists in rating PD styles.

Initial factor analyses of the 82 DF items identified 15 factors, although not all of these were well supported (with some factors having less than four items, and some items loading <0.03). Deleting these factors and items resulted in a set of 67 items and 11 factors. Factor analysis of scale scores generated from the first-order factors identified two second-order factors. These solutions provided the basis for a confirmatory factor analysis (CFA) in this sample – an a priori pattern for 11 first-order and two second-order factors. The CFA was carried out in LISREL and the solution gave a moderately good fit to the data. Based on Hoyle (9), the RMSEA statistic (0.74) was slightly better than a marginal fit; the GFI statistic (0.65) was poor; while the CFI statistic (0.96) was very good. Given that we had a large number of items in a relatively small sample, we interpreted reasonable support for the proposed factor model.

Table 1 reports factor loadings and associations between item scores for each of the lower-order DF constructs and clinician-rated DOMAIN and GLOBAL JUDGMENT scores, with associations ranging from non-existent to slight, suggesting that severity of lower-order DF constructs did not correlate with clinical judgments of PD severity.

As for the STYLE measure, DF percentage scores were calculated for the 11 constructs to allow standardized prevalence estimates of: ‘instability under stress’ (58%), ‘self-defeating’ (50%), ‘pessimism’ (49%), ‘ineffectiveness’ (45%), ‘low self-direction’ (44%), ‘impulsivity’ (42%), ‘inflexibility’ (42%), ‘disagreeableness’ (35%), ‘not learning from experience’ (33%), ‘non-empathic’ (29%) and ‘uncaring to others’ (27%). The only sex difference (P < 0.05) was for males to score lower on the ‘non-empathic’ scale.

Based on the regressions of the second-order factors on the items from the CFA we took the 10 items with the highest weights for non-cooperativeness and summed item scores to form a non-cooperativeness scale. We similarly generated a 10-item non-coping scale. Contributing items (listed in the Appendix) came from seven of the 11 first-order factors. The two scales correlated r = 0.97 and 0.98 respectively with the factor scores for the second-order factors, and with each other (r = 0.56 and 0.64 for scales and factors respectively). There was no sex difference for non-cooperativeness (males = 11.6, females = 11.2, t = 0.54, d.f. = 307, P = 0.59) but there was for non-cooperativeness (males = 7.9, females = 6.0, t = 3.66, d.f. = 307, P < 0.001).

There was no difference in returning positive GLOBAL scores for either non-coping (F = 2.27; d.f. = 2, 97; P = 0.11) or non-cooperativeness (F = 0.41; d.f. = 2, 97; P = 0.67). The correlations between DOMAIN and the two scales were quite low (r = 0.18 and r = 0.16). These two results indicate negligible agreement between self-reported and clinician-rated levels of disordered functioning.

We next examined if DF (as measured by scales for lower-order and higher-order DF constructs) was associated specifically or non-specifically with differing PD styles as measured by the 15 STYLE scores. Table 2 shows relevant correlation coefficients. Lower-order DF scale scores were associated with STYLE scores, supporting a model whereby dysfunction was non-specifically associated with the separate PD styles. The non-specific

<table>
<thead>
<tr>
<th>Lower-order DF construct</th>
<th>Loadings on higher-order factors</th>
<th>Correlation between DF Scale and clinician-rated measure of PD severity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Factor 1 (non-coping)</td>
<td>Factor 2 (non-cooperativeness)</td>
</tr>
<tr>
<td>Disagreeableness</td>
<td>–</td>
<td>0.96</td>
</tr>
<tr>
<td>Inflexibility</td>
<td>0.73</td>
<td>–</td>
</tr>
<tr>
<td>Uncaring to others</td>
<td>–</td>
<td>0.88</td>
</tr>
<tr>
<td>Non-empathic</td>
<td>–</td>
<td>0.90</td>
</tr>
<tr>
<td>Ineffectiveness</td>
<td>0.94</td>
<td>–</td>
</tr>
<tr>
<td>Self-defeating</td>
<td>0.96</td>
<td>–</td>
</tr>
<tr>
<td>Failure to learn from experience</td>
<td>0.88</td>
<td>–</td>
</tr>
<tr>
<td>Impulsivity</td>
<td>0.62</td>
<td>–</td>
</tr>
<tr>
<td>Pessimism</td>
<td>0.91</td>
<td>–</td>
</tr>
<tr>
<td>Instability under stress</td>
<td>0.77</td>
<td>–</td>
</tr>
<tr>
<td>Lacking self-direction</td>
<td>0.99</td>
<td>–</td>
</tr>
</tbody>
</table>

Pearson correlation significant at *0.05 level and **0.01.
associations might reflect either the interdependence of the DF constructs or our STYLE descriptors truly being non-specifically linked to DF. Scores for the two higher-order DF constructs also linked non-specifically with STYLE scores (Table 2), with associations almost invariably stronger with ‘non-coping’ than with ‘non-cooperativeness’ scores.

We then contrasted data from samples with and without a personality disorder. On all STYLE measures and lower-order DF scales, patients returned significantly higher scores (\( P < 0.001 \)) than the non-clinical controls. On the 10-item DF scales, the patients returned significantly higher ‘non-coping’ (13.1 vs. 5.1, \( t = 10.9, \text{d.f.} = 314, P < 0.001 \)) and ‘non-cooperativeness’ (7.6 vs. 4.2, \( t = 5.8, \text{d.f.} = 314, P < 0.001 \)) scores. Receiver operating characteristic (ROC) analyses showed ‘non-coping’ to have an area under curve (AUC) of 0.87 and an optimal cutoff [minimizing the sensitivity (Se) and specificity (Sp) difference] of nine or more, at which point Se = 0.78 and Sp = 0.81. For ‘non-cooperativeness’ the AUC was 0.73 at the optimal cutoff of 6 or more (Se = 0.67 and Sp = 0.66). Some 60% of patients were positive on both cutoffs, compared with 15% of controls.

We next examined a central question: in modeling the PDs, should descriptors of personality style and/or of DF be prioritized? This question was approached by first determining the relative capacities of the 15 personality STYLE scores and the two 10-item DF scale scores to distinguish patients and controls, using discriminant analysis.

In one analysis, only the two higher-order DF scales were entered. The standardized weights (0.95 and 0.10) indicated that scores on the ‘non-coping’ scale discriminated more than ‘non-cooperativeness’ scale scores. The sensitivity and specificity in predicting true ‘cases’ and true ‘non-cases’ was relatively high (76 and 82% respectively, canonical \( R = 0.60 \)).

In a second analysis, the set of 15 STYLE scores only was entered using a stepwise procedure, with only two STYLE scores being retained in the final equation (anxious and depressive, with weights of 0.48 and 0.64 respectively). The sensitivity and specificity in predicting true ‘cases’ and true ‘non-cases’ was again high (81 and 91% respectively, canonical \( R = 0.60 \)). The prominence of anxious and depressive STYLE scores raised the possibility that groups might have been distinguished more by mood disturbance than by PD status. We therefore repeated the analyses, after removing ‘anxious’ and ‘depressive’ scores.

Table 2. Correlations of self-rated PD STYLE scores with self-report DF scores and with clinician-rated judgments

<table>
<thead>
<tr>
<th>PD-VIGNETTE</th>
<th>Lower-order DF scores</th>
<th>Clinician-rated PD VIGNETTE</th>
<th>Clinician-rated DOMAIN score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paranoid</td>
<td>0.63</td>
<td>0.43</td>
<td>0.38</td>
</tr>
<tr>
<td>Schizoid</td>
<td>0.59</td>
<td>0.35</td>
<td>0.28</td>
</tr>
<tr>
<td>Narcissistic</td>
<td>0.56</td>
<td>0.28</td>
<td>0.23</td>
</tr>
<tr>
<td>Antisocial</td>
<td>0.55</td>
<td>0.35</td>
<td>0.28</td>
</tr>
<tr>
<td>Borderline</td>
<td>0.57</td>
<td>0.35</td>
<td>0.28</td>
</tr>
<tr>
<td>Histrionic</td>
<td>0.54</td>
<td>0.35</td>
<td>0.28</td>
</tr>
<tr>
<td>Avoidant</td>
<td>0.48</td>
<td>0.35</td>
<td>0.28</td>
</tr>
<tr>
<td>Dependent</td>
<td>0.44</td>
<td>0.35</td>
<td>0.28</td>
</tr>
<tr>
<td>Obsessive</td>
<td>0.43</td>
<td>0.35</td>
<td>0.28</td>
</tr>
<tr>
<td>Anxious</td>
<td>0.49</td>
<td>0.35</td>
<td>0.28</td>
</tr>
<tr>
<td>Depressive</td>
<td>0.56</td>
<td>0.35</td>
<td>0.28</td>
</tr>
<tr>
<td>Sadistic</td>
<td>0.54</td>
<td>0.35</td>
<td>0.28</td>
</tr>
<tr>
<td>Self-defeating</td>
<td>0.57</td>
<td>0.35</td>
<td>0.28</td>
</tr>
<tr>
<td>Negativistic</td>
<td>0.59</td>
<td>0.35</td>
<td>0.28</td>
</tr>
</tbody>
</table>
from the predictor set of STYLE variables. Three of the 13 STYLE scores were retained: borderline, sadistic, and self-defeating (standardized weights of 0.52, −0.26 and 0.68 respectively) and with reduced sensitivity and specificity (77 and 87%, canonical $R = 0.56$).

When the DF scores for ‘non-coping’ and ‘non-cooperativeness’ were entered first and then the 13 STYLE scores were entered stepwise, only ‘self-defeating’ was added to the equation (with standardized weights of 0.47, 0.06 and 0.59 respectively). The resulting sensitivity (92%) and specificity (65%) gave no advantage.

As an additional test of capacity to distinguish samples, a series of ROC curves was calculated for the STYLE scale and two higher-order DF scales, and for composite scales where the best composite was calculated as described by Su and Liu (10). Table 3 reports sensitivity, specificity and the non-parametric AUC, indicating how well each predictor performed as a diagnostic test. Analyses of the higher-order DF scales alone showed that the ‘non-coping’ scale was superior to that of ‘non-cooperativeness’ scale, and that combining the scales did not produce any advantage over use of the ‘non-coping’ scale alone. An analysis of individual STYLE scores showed that all were highly effective in differentiating patients from controls (in particular, borderline, anxious, depressive and self-defeating styles). The performance of the combined STYLE scales was slightly superior to the combined DF scales (with respective AUCs of 0.94 and 0.89).

We next sought to validate the DF measure in the independent sample of 35 subjects (mean age = 35.1 years; 63% female) with personality dysfunction. Of these, 26 had a nominated family member (for example, parent, spouse, de facto partner) or close friend complete an observer-rated DF questionnaire. The mean ‘non-coping’ DF scale scores of the sample was higher than that generated in the development sample by the patients (16.4 vs. 13.1), while ‘non-cooperativeness’ scores were comparable (8.4 vs. 7.6).

For validation, we first examined agreement between patient’s SR and CW rated scores on the DF scales. ‘Non-coping’ scale scores were similar (SR = 17.0; CW = 15.9, $t = 0.95$, d.f. = 24, ns) and moderately correlated ($r = 0.61$, $P < 0.001$), as were ‘non-cooperativeness’ scale scores (SR = 8.8; CW = 8.8, $t = 0.08$, d.f. = 24, ns; $r = 0.41$, $P = .04$). Secondly, correlation between interviewers in rating lifetime ‘non-coping’ and ‘non-cooperativeness’ was high (Pearson correlations of 0.70 and 0.77 respectively, $P < 0.001$). However, when we examined the extent to which those interviewer-based ratings correlated with SR-rated and CW-rated DF scores, agreement was poor. The correlation of mean interviewer-judged rating of ‘non-coping’ with the relevant SR scale was 0.34 (ns) and 0.36 (ns) with the CW scale. The correlation of the mean interviewer-judged rating of ‘non-cooperativeness’ with the relevant SR was 0.33 (ns) and 0.25 (ns) with the relevant CW scale.

In summary, we found stronger agreement for the SR measure with CW reports than with interviewer ratings. This supports the validity of the SR strategy.

**Table 3. Capacity of two disordered function (DF) scale scores and 11 style scores to differentiate clinical and control samples, using receiver operating characteristic (ROC) analyses.**

<table>
<thead>
<tr>
<th>Analysis</th>
<th>Test</th>
<th>Se</th>
<th>Sp</th>
<th>AUC</th>
<th>AUC 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>DF scales – individual</td>
<td>Non-coping</td>
<td>0.83</td>
<td>0.77</td>
<td>0.89</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-cooperativeness</td>
<td>0.67</td>
<td>0.66</td>
<td>0.74</td>
</tr>
<tr>
<td>II</td>
<td>DF scales – combined</td>
<td>0.80</td>
<td>0.80</td>
<td>0.89</td>
<td>0.82–0.93</td>
</tr>
<tr>
<td>III</td>
<td>PS scales – individual</td>
<td>Paranoid</td>
<td>0.80</td>
<td>0.79</td>
<td>0.85</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Schizoid</td>
<td>0.77</td>
<td>0.75</td>
<td>0.85</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Schizotypal</td>
<td>0.76</td>
<td>0.82</td>
<td>0.94</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Narcissistic</td>
<td>0.72</td>
<td>0.66</td>
<td>0.78</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Antisocial</td>
<td>0.77</td>
<td>0.80</td>
<td>0.84</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Borderline</td>
<td>0.85</td>
<td>0.84</td>
<td>0.89</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Histrionic</td>
<td>0.69</td>
<td>0.71</td>
<td>0.77</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Avoidant</td>
<td>0.82</td>
<td>0.77</td>
<td>0.87</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dependent</td>
<td>0.76</td>
<td>0.80</td>
<td>0.850</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Obsessive/compulsive</td>
<td>0.67</td>
<td>0.77</td>
<td>0.80</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anxious</td>
<td>0.83</td>
<td>0.79</td>
<td>0.90</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Depressive</td>
<td>0.96</td>
<td>0.62</td>
<td>0.91</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sadistic</td>
<td>0.65</td>
<td>0.71</td>
<td>0.72</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Self-defeating</td>
<td>0.82</td>
<td>0.80</td>
<td>0.91</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Negativistic</td>
<td>0.71</td>
<td>0.71</td>
<td>0.82</td>
</tr>
<tr>
<td>IV</td>
<td>PS scales – combined</td>
<td>0.86</td>
<td>0.86</td>
<td>0.94</td>
<td>0.85–0.98</td>
</tr>
</tbody>
</table>

Se, sensitivity; Sp, specificity; AUC, area under curve for ROC.

**Discussion**

Disordered functioning – conceptually broad, empirically parsimonious

We commenced assuming that disordered personality function is likely to be expressed in multiple ways. A detailed literature review (6) identified 17 constructs, and we expected that subsequent refinement would still result in a sizeable number. However, results suggested a more parsimonious model of DF. For example, when the constructs were reduced to 11, each was moderately to highly associated with each other. This led to a secondary analysis defining just two higher-order constructs – ‘non-coping’ and ‘non-cooperativeness’, with scores on the two 10-
item derived scales being moderately associated ($r = 0.45$). Interdependence of the two constructs may reflect methodological issues, with associations between them being dependent on other variables (e.g. severity) or response biases (e.g. social desirability).

Alternatively, disordered personality function might be expressed – and therefore measured – very simply as failure to cooperate and failure to cope. There is historical support for such a model – with Freud (11) defining full maturity as requiring only two markers: ‘Lieben und arbeiten’ (to love and to work, p. 238). ‘To love’ was assumed to refer to the capacity to make and maintain intimate relationships, and assumes cooperation. ‘To work’ referred not merely to being employable but involved in ‘work-productiveness’.

Our strategy for refining the constructs of disordered personality function relied on factor analysis, a strategy that has been criticized as imposing a structure rather than discovering one (12), and its limitations need to be recognized here. In fitting a CFA we examined how well a previous finding ‘carried across’ differing samples. We considered that a two-factor higher-order solution provided both the ‘best fit’ of the data as well as the potential for greater flexibility in future applied research. However, the need to retain ‘non-cooperativeness’ can be challenged, in that ‘non-coping’ was superior to ‘non-cooperativeness’ in every relevant analysis. In the discriminant analyses, it was not only superior to ‘non-cooperativeness’ but made its contribution redundant.

However, until the model has been tested further, we suggest however that ‘non-cooperativeness’ be retained. Discarding it now could narrow definition of personality dysfunction to a coping domain and ignore a (cooperative) construct central to many definitions of PD. For example, Livesley et al. (13) defined PD as a tripartite failure of (i) the self-system, with failure to establish stable and integrated representation of self and others; (ii) adaptive function in interpersonal relationships; and (iii) prosocial and cooperative relationships, while Rutter (14) held that ‘a pervasive persistent abnormality in maintaining social relationships’ underpinned many categorized PDs.

Several authors (15, 16) have also considered disordered personality function to be manifested according to a coping paradigm. Millon (17) argued for three principal features differentiating normal and pathological behaviour (functional inflexibility, self-defeating circles and tenuous stability under stress) and later an additional set of five parameters (causing personal discomfort, reduction of opportunities, ineffective function, non-adjustment to the environment, and causing discomfort to others). Our analyses included all such constructs and, while demonstrating their utility as markers, defined them as lower-order constructs contributing principally to the higher-order construct of ‘non-coping’. Livesley et al. (13) emphasized inflexibility and inadequate performance in ‘the universal life tasks of identity, attachment, intimacy and affiliation’, also noting that their definition did not differ greatly ‘from Freud’s definition of mental health as the ability to work and to love’.

Despite starting with a very wide set of indicative constructs, our final two-construct model is strikingly similar to that emerging from Cloninger’s studies in which character scales of low ‘self-directedness’ and low ‘cooperativeness’ were proposed as the core features of PD (18). Our CFA empirically established the dominating contribution of the higher-order constructs, with, for example, scores on the lower-order construct ‘lacking self-direction’ loading 0.99 on the higher-order ‘non-coping’ construct, and the lower-order construct ‘disagreeableness’ loading 0.96 on the higher-order ‘non-cooperativeness’ construct.

Specificity of disordered functioning
If such a parsimonious model of DF is valid, scores on the DF measure should be associated with all, rather than merely some, PD categories. We did find some instances of specificity: for example, narcissistic and obsessive/compulsive personality STYLE scores were somewhat less strongly associated with ‘non-coping’ and ‘non-cooperativeness’ scores than other STYLE scores. Their less clear relationship with DF is supported by the observation (19) that narcissistic and obsessive PDs are the ‘two disorders that often allow patients to succeed in the Western cultures’ (p. 268). However, the overall pattern described in Table 2 supports a non-specificity model. This could reflect the nature of disordered personality function, in that all PDs are evidenced by limitations in coping and cooperativeness. It is also possible that the non-specific pattern may reflect a
higher-order factor, such as severity of disordered personality.

Validity of self-rating

Although our patient sample was derived from psychiatrists recruiting patients judged clinically as having distinct limitations in personality function, we found little agreement between SR and clinician-rated data. The correlation between psychiatrists’ judgments of personality styles (PD VIGNETTE) and patient-generated STYLE scores was only 0.26, and patient-generated DF and style scores correlated poorly with psychiatrist-rated DOMAIN scores. Despite high inter-rater agreement between two professionals in our validation study assessing ‘non-coping’ and ‘non-cooperativeness’, we found weak agreement between interviewers’ scores and both subjects’ SR and CW scores. However, SR scores were correlated with CW scores on the DF measure, supporting the SR strategy. Such results suggest that external raters, whether trained interviewers or treating psychiatrists, have limited capacity to assess DF – hardly surprising when external raters are observing individuals in a socially constrained context rather than in vivo.

Diagnostic classification

In considering how study results link with the formal diagnosis and classification of PDs, we should note key historical issues, with Livesley (5) describing three classificatory phases. First is the pre-DSM-III phase, dominated by clinical descriptions within phenomenological and psychoanalytic fields. Empirical research in the 1970s led to the second (DSM-III) phase, with PDs placed on a separate axis and having diagnostic criteria, encouraging further empirical analyses. Livesley argued that the field is entering a third, post-DSM-III/IV phase, reflecting limitations to the DSM model. Although the alternative ICD-10 classification differs in some details, its PD conceptualization is similar to DSM-IV.

The DSM-IV model is categorical and includes 10 formalized PDs organized into three higher-order clusters, a categorical classification consistent with traditional medical approaches seeking to identify specific diseases and syndromes. However, the multiple problems presented by a categorical system are well recognized (20). The necessity to judge disorders as either ‘present’ or ‘absent’ results in arbitrary distinctions between ‘normal’ and ‘abnormal’ personality. In clinical application, diagnoses of DSM PD categories have low reliability and validity. Structured and semistructured interviews have been developed because of such limitations to clinical assessment, but no scale has emerged as the ‘gold standard’. It has long been unclear whether subject or informant report is the more valid, with choices influenced by the subject’s insight, how well the informant knows the subject, the nature of the trait being measured and the purpose of the assessment (e.g. clinical assessment, research).

We earlier noted that current PD definition reflects an amalgam of descriptors of personality style and of DF. By examining the capacity of separate measures of these components to discriminate subjects judged a priori as possessing or not possessing significant personality problems, we sought to determine which construct was the superior discriminator. After stripping the ‘ depressive’ and ‘anxious’ PD STYLE predictors from initial analyses, to overcome distinctions being more driven by mood state, both STYLE and DF parameters had almost equivalent capacity to discriminate between patients and controls. However, the STYLE descriptors were developed from relevant DSM and ICD descriptor sets, so many were ‘contaminated’ by descriptors of personality function. If we had alternatively stripped STYLE descriptors of their ‘function’ components, our study would have been so much at variance with the existing DSM model as to make comparative judgments impossible. Nevertheless, as PD dysfunction was predicted strongly by both style and functioning components, the use of both parameters is supported.

The results encourage a research model testing the utility of a two-tiered model of PD which first emphasizes the likelihood of disordered personality function (e.g. ‘definite’, ‘probable’, ‘possible’, ‘absent’), and secondarily provides descriptors of personality style. This is consistent with a proposal by Livesley (5) that PD be diagnosed by DF alone, and noted within axis I of the DSM system, while personality styles would be positioned on axis II. In contrast to the current categorical diagnostic models, both tiers would ideally be dimensionally based, consistent with the view (21) – and empirical findings (13) – that PDs are maladaptive or extreme expressions of common personality traits rather than being qualitatively different from normal personality function.

The secondary component in the proposed two-tier model could emphasize either ‘normal’ or ‘abnormal’ personality styles. As an example of the former, the five-factor model of personality (22) proposes dimensions of neuroticism, extroversion, openness to experience, agreeableness
Parker et al.

...and conscientiousness. Emphasizing ‘normal’ personality dimensions would allow a bridge between the two disparate dimensional and categorical ‘worlds’, and between psychological and psychiatric research. The alternative second-tier option would be to derive prototypic phenotypes of distinct personality styles as observed by clinicians. Prototypes could respect those captured in current and past DSM and ICD systems or other categorical models (19), with an individual’s adherence to the prototype also being dimensionally measured. A third ‘mixed model’ is another option, and might assist model refinement. The classificatory accuracy and clinical utility of the two-tiered model could then be compared with more traditional PD assessment strategies.

Conclusions

Our review and the studies reported in this paper advance a new model for measuring and researching the PDs, and which might have classificatory utility. The model is dimensional and two-tiered, separating descriptors of DF from descriptors of personality style. The model also allows for disjunctions between tiers, in that some individuals may have extreme personality ‘styles’ but function well, and vice versa. For example, current descriptors of antisocial personality disorder emphasize the characteristics of the ‘failed sociopath’, but in reality descriptors that include personality style as well as level of function can be associated with either ‘success’ or ‘failure’.

The two-tiered model overcomes limitations of current models attempting to capture separate domains of style and function within a single field, and has the potential to provide more meaningful information to clinicians and to research endeavors.

Acknowledgement

This study was funded by an NHMRC Program Grant (222308) and an Infrastructure Grant from the NSW Department of Health. The authors thank the many psychiatrists who assisted with study recruitment.

References

Appendix

Ten items loading on each of the higher-order scales, and the lower-order scales from which each was derived.

<table>
<thead>
<tr>
<th>Lower order scale</th>
<th>Non-cooperativeness</th>
<th>Non-coping</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agreeableness</td>
<td>Even when I have to, I am unable to get along with family or people at work</td>
<td>I seem to fail more often than I succeed in life</td>
</tr>
<tr>
<td></td>
<td>I am generally described as a nice person*</td>
<td>My personality often causes me to lose out</td>
</tr>
<tr>
<td></td>
<td>I can be somewhat difficult in dealing with others</td>
<td>I know I cope poorly with things</td>
</tr>
<tr>
<td></td>
<td>In general, I will listen to and understand the other person’s point of view*</td>
<td>When things go wrong I am generally able to bounce back*</td>
</tr>
<tr>
<td></td>
<td>Friends see me as cooperative and agreeable*</td>
<td>I feel confident in my ability to size up and deal with any situation*</td>
</tr>
<tr>
<td></td>
<td>People at work see me as cooperative and agreeable*</td>
<td>I learn from the mistakes I make*</td>
</tr>
<tr>
<td></td>
<td>I tend to be very understanding of other people’s feelings and problems*</td>
<td>I am really resourceful in tackling problems*</td>
</tr>
<tr>
<td></td>
<td>I am generally ready and willing to lend an ear*</td>
<td>Others see me as a reliable person*</td>
</tr>
<tr>
<td></td>
<td>People see me as good-hearted*</td>
<td>I feel I have little control over where my life is headed</td>
</tr>
<tr>
<td></td>
<td>People who know me well would describe me as a caring person*</td>
<td>I feel like I am going around in circles in life</td>
</tr>
</tbody>
</table>

*Reverse scoring.
**Organic/Old Age Disorders**

**Neuropsychiatry**


**Old Age**


Neuropsychiatric Disorders

Perminder Sachdev
Professor of Neuropsychiatry,
University of New South Wales, and
Neuropsychiatric Institute,
The Prince of Wales Hospital,
Sydney
Why is thought being a secretion of the brain more wonderful than gravity a property of matter?
Attributed to Charles Darwin

What is neuropsychiatry?
Neuropsychiatry may be regarded as the application of the neurological paradigm to psychiatric syndromes. It brings together the descriptive, nosological and therapeutic strengths of psychiatry, the empirical foundations of neurology and the assessment skills of neuropsychology to deal with these disorders. Its sister discipline within neurology is behavioural neurology. Several general statements can be made about neuropsychiatry:
• All types of behavioural disturbances that occur in psychiatric disorders can also occur in conjunction with neurological disorders.
• That psychiatric and neurological disorders are responsible for similar syndromes does not mean that the pathogenetic mechanisms are the same, but it is likely that there is significantly shared pathophysiology.
• A thorough diagnostic assessment from a general medical viewpoint is an essential part of any psychiatric assessment.
• Neuropsychiatric syndromes should commonly be suspected in the elderly, and in patients with brain damage or substance abuse, or if the syndrome is atypical, and family history and a vulnerable personality are lacking.
• Psychiatric treatments, physical or behavioural-psychotherapeutic, rely on intervention in brain processes and are applicable to both neuropsychiatric and idiopathic psychiatric disorders.

Neuropsychiatric assessment
A neuropsychiatric assessment is no more or less than a good psychiatric assessment, differing only in the emphasis on medical-neurological factors. It comprises:
• A psychiatric and medical history
• A detailed mental state examination, including a cognitive examination
• A physical examination, especially neurological
• Neuropsychological assessment
• Laboratory investigations to evaluate general medical disorders and substance abuse, including electrophysiology (EEG, ERPs) and neuroimaging (CT, MRI, SPECT, PET) (see below).

History
An account from an informant is often vital. Physical symptoms and behavioural change are important, and these are documented chronologically. Pointers to brain disease (e.g. seizures, head injury, alcohol and drug use and cognitive impairment) are emphasised. Any personality change (e.g. impulsivity, aggressiveness or disinhibition) is also pertinent.
Mental state

The mental state examination has the same format as in general psychiatry, except for a more detailed cognitive assessment.

Cognitive state examination (CSE)

The essential brief cognitive assessment is possible at the bedside. For detailed assessment, referral to a clinical neuropsychologist is necessary. The CSE must include these domains or systems:

- Alertness and arousal
- Attention and concentration
- Orientation
- Memory
- Language
- Visual-spatial and constructive functions
- Frontal lobe and fronto-subcortical functions
- Other dominant (left) hemisphere functions: calculation, praxis, R–L orientation, finger gnosis
- Other non-dominant (right) hemisphere functions: dressing apraxia, neglect phenomena, agnosias
- Insight and judgement

Assessment of level of consciousness and attention is crucial as disturbance can influence performance in other tests.

Common bedside tests

For most purposes, a screening battery such as Folstein’s mini-mental state examination (MMSE) is a good starting point. It tests orientation, immediate and recent memory, concentration, arithmetic ability, language and praxis. Easy to administer in only 5–10 minutes, it has reasonable sensitivity but low specificity, and may be used for serial evaluations. The maximum score is 30; 27 or less indicates impairment; 25 or less is definitely abnormal. The MMSE may be normal in the presence of subtle impairment, which, if suspected, requires further evaluation.

It is useful to combine the MMSE with the clock drawing test, which requires the patient to draw a clock-face with hands indicating 10 past 10. This tests constructional abilities and, more importantly, planning and organisation (frontal lobe function).

Examination of the frontal lobes is central to many neuropsychiatric disorders and these tests are suggested:

- Behavioural observation: impulse control, delaying gratification, motivation, affective regulation, personal relationships
- Motor and expressive language
- Primitive reflexes: grasp, palmomental, snout, pout, glabellar tap
- Verbal fluency: semantic, e.g. saying as many words (not proper nouns and without repetition) as possible in 1 minute beginning with F or A or S; category e.g. naming as many objects in one category (e.g. animals) as possible in 1 minute
• Motor sequencing: Luria's hand sequences (e.g. *fist-ring test*, alternating repeatedly between making a fist and a ring with one hand and then the other; alternating between a fist, palm and cut movement with one hand and then the other)
• Reasoning and conceptualisation: similarities, differences, proverbs
• Planning and organisation: clock drawing test (as above, followed by copying a clock drawn by the examiner).

The tests are subject to several limitations and qualifications. They are usually influenced by various cognitive functions (e.g. a simple test like 'serial 7s' may be affected by impairment of attention, short-term memory and calculation ability). A battery of tests is therefore necessary to determine which particular function is disturbed. Failure on one test must be supported by others before dysfunction is established. All tests are designed to be administered in a standard manner; significant departure from this may render them invalid. Repetition of tests may lead to improved performance due to 'practice effect,' which may confound subsequent formal assessment by a neuropsychologist. Therefore, use only tests that are meaningful for a bedside assessment.

**Physical examination**

Neuropsychiatry has been called 'psychiatry with signs', and it is important to examine the patient for neurological and systemic disease. When definite signs of neurological disease are lacking, 'soft' or non-localising signs (e.g. high-level sensory integration, motor co-ordination, gait and posture, stereognosis) are sought. Their diagnostic significance however is uncertain.

**Laboratory investigations**

Electrodiagnostic techniques continue to play a role in diagnosis. The electroencephalogram (EEG) is important in evaluating epilepsy and delirium. It has a role in differentiating organic from non-organic disorders, though cautious interpretation is called for. Event-related potentials (ERPs) are useful to determine the integrity of sensory pathways. Sleep studies using EEG and other measures have many indications. The role of quantitative EEG and magnetoencephalography is still emerging.

Recent advances in *neuroimaging* have greatly affected neuropsychiatry. The techniques can be divided into structural and functional.

**Structural imaging**

The two major techniques are computerised tomography (CT) and magnetic resonance imaging (MRI). While CT is cheaper and more readily available, MRI offers many advantages. It provides excellent anatomical and spatial resolution, uses no ionising radiation, visualises the posterior fossa and pituitary regions without distortion due to bone, is more sensitive to white matter pathology, and can scan in any plane. A single scanning session can combine structural MRI with functional analysis of the brain using magnetic resonance spectroscopy (MRS), exogenous (gadolinium-tracking) or endogenous (arterial spin labelling) contrast based perfusion imaging, angiography and/or blood oxygen level dependent (BOLD) functional MRI (fMRI).
Functional imaging

These techniques are used primarily to provide information on the metabolism, blood flow, neurochemistry or activity of the brain. Positron emission tomography (PET) and single photon emission computed tomography (SPECT) are the most important nuclear medicine based techniques. Both rely on incorporation of a radioactive nuclide into a drug (a radiopharmaceutical) which is injected intravenously. Analysis of brain uptake and regional brain activity over time provides information about metabolism, blood flow, and so forth. SPECT scanning is cheaper and more readily available. However, its resolution is less than that of PET and its range somewhat limited.

Other functional techniques such as MRS and fMRI are increasingly popular. MRS is akin to chemical biopsy of the brain; BOLD fMRI permits study of minute changes in blood flow in relation to physiological function; and regional cerebral blood flow can be determined by MRI using either an external contrast agent such as gadolinium or by magnetically tagging the spin of blood cells.

Mental disorders due to a medical condition

Classification

Secondary or ‘organic’ mental disorders

These are characterised by mental symptoms judged to be the direct physiological consequence of a general medical condition. Previously categorised as ‘organic mental disorders’, DSM-IV now distinguishes those due to a general medical condition (secondary mental disorders) from those that are substance-induced or have no specified aetiology (primary mental disorders).

Primary disorders do have a basis in brain dysfunction but their aetiology is poorly understood and they are therefore ‘idiopathic’ (the term ‘secondary’ has replaced ‘organic’ to emphasise this point). They have been inappropriately referred to as ‘functional’, implying that psychological factors predominate. Since functional disturbance is part of all psychiatric disorders, this term is best avoided; indeed psychosocial factors may exacerbate disorders with a primary ‘organic’ cause. It is always necessary to consider biological, psychological and social factors, even when the primacy of one or other is clear.

Aetiology

Features which point to an organic contribution include: (a) cognitive dysfunction; (b) a general medical disorder known to be associated with neuropsychiatric syndromes; (c) an atypical psychiatric syndrome (e.g. late age of onset) with unusual clinical features; and (d) resistance to treatment. Diagnosis entails these questions:

- Does the patient suffer from delirium, psychosis, mood disorder, i.e. a syndromal diagnosis?
- Is a general medical condition present, or can a substance be implicated (as ascertained by history, physical examination and laboratory investigations)?
- Is the mental disturbance related to the medical condition or substance aetologically through a physiological mechanism? There are no infallible guidelines, but these considerations are helpful:
- Temporal association between onset, exacerbation or remission of the medical condition and the mental disorder (though there are many exceptions)
- Features atypical of the primary mental disorder
- Research evidence of a well-established link between the medical condition and specific mental disorder

• Can the disturbance be accounted for by another mental disorder? (ruling out a primary mental disorder such as depression or schizophrenia may be difficult)
• Does the disturbance occur exclusively during the course of a delirium?
• Could multiple causes be implicated?

Not only are there multiple causes of many neuropsychiatric syndromes, but those causes may produce a variety of syndromes (e.g. corticosteroids may, in different patients, be associated with depression, euphoria or frank mania, delirium, anxiety, psychosis, even dementia). The same applies to causes like alcohol, brain trauma and epilepsy. A number of variables determine the particular syndrome. Relevant variables in the person are age, gender, premorbid personality, past psychiatric illness, education, level of support and quality of social relationships. A second set of variables encompasses type of brain impairment, functional loss, brain regions involved, and degree of reversibility of the dysfunction.

Specific non-cognitive disorders
Psychotic disorder due to a general medical condition or substance-induced

Features are prominent hallucinations or delusions due to the medical condition or substance directly. While one feature usually predominates, often both are present.

Hallucinations

Their modality is determined by causal factors (e.g. hallucinogens usually cause visual hallucinations and alcohol auditory ones). People blind due to cataracts may visually hallucinate; in those deaf through otosclerosis, they are likely to be auditory. Auditory hallucinations typically occur in 'primary' psychoses whereas those in other modalities are often 'organic', the commonest being visual. Hallucinations vary from simple and unformed to complex. The degree of insight into the hallucinations varies. Some patients develop an elaborate secondary delusional system. Most people with hallucinations of an organic source in general hospitals are delirious. Hallucinations are common in the setting of drug abuse.

Hallucinogens (e.g. lysergic acid diethylamide (LSD), 3,4 dioxymethylenemethamphetamine (MDMA), psilocybin and mescaline) and prolonged use of alcohol are the common causes of hallucinosis. The former can lead to an acute hallucinosis through intoxication. In some patients who chronically abuse hallucinogens, episodic partial recurrences of prior hallucinogen-induced experiences (flashbacks) occur (termed 'Hallucinogen Persisting Perception Disorder' in DSM-IV). A few develop persistent hallucinatory psychoses. Alcoholic hallucinosis, consisting of vivid and persistent auditory hallucinations, often malicious, reproachful or threatening in nature, may follow cessation of, or reduction in, alcohol ingestion.

Visual hallucinations are associated with certain CNS disorders (e.g. epilepsy, migraine, brain–stem lesions) or eye diseases (optic neuritis, retinal detachment).
They may occur in the context of sleep or sensory deprivation, hypnotic states or hypnosis. The occurrence of vivid, formed and elaborate visual hallucinations in partially sighted elderly individuals has been referred to as the Charles Bonnet Syndrome. Visual hallucinations are also a common symptom in the late stages of Parkinson’s disease and in patients with Dementia with Lewy Bodies. Olfactory, gustatory and kinaesthetic hallucinations are rare and likely to be part of partial complex epilepsy or a psychiatric disorder.

Treatment depends on identifying the cause. Drug intoxication usually settles with time and in a safe environment, without drug treatment. Drugs with anticholinergic effects are best avoided since street drugs are often ‘cut’ with them. Antipsychotics may help as may correction of sensory impairment in the elderly (see Chapter 22).

Jim, a 55-year-old successful businessman, was admitted to the medical ward for investigation of gastrointestinal bleeding from a suspected peptic ulcer. Two days later he became fearful, refusing to co-operate. A psychiatric consultation revealed that he was hearing multiple voices that threatened to kill him. Jim believed these voices were coming from the paging system and that doctors and nurses were conspiring against him. He insisted on leaving hospital.

He was in clear consciousness and highly aroused autonomically. He had no past history or family record of psychiatric illness. He had used alcohol excessively (about 60–80 g/day) for over 2 decades but had reduced his intake in the last 5 years. He was being buffeted by marital and occupational tensions.

Alcoholic hallucinosis with secondary delusions was diagnosed, with the likelihood that abrupt quitting of alcohol use on admission had precipitated it. Treated with Risperidone (up to 4 mg/day), he began to improve after 3 days and recovered within 2 weeks. He was maintained on Risperidone for a further 3 months. He also undertook an alcohol dependence programme. His peptic ulcer was successfully treated.

Delusions

Described in several neurological disorders, delusions must occur in clear consciousness and without significant cognitive impairment to be categorised as secondary psychosis. Although their nature varies and depends in part on the cause, delusions are most often persecutory. They are well organised or fleeting and changeable.

Causes are diverse, but drugs are most common (e.g. amphetamines, dopaminomimetics such as L-dopa, Bromocriptine and Pergolide, corticosteroids, cannabis, and phencyclidine). Amphetamine is the classical culprit; in large amounts over a brief period it can produce psychosis even in normal volunteers. Psychosis may be preceded by irritability, restlessness and perceptual sensitivity. Delusions are usually of persecution and reference but more fluid than in schizophrenia. Associated visual and auditory hallucinations are common. Although the psychosis usually resolves when amphetamine intake stops, relief for acute symptoms may be needed.

The association of schizophrenia-like psychosis with chronic cannabis use has received much attention recently. The five longitudinal studies that have examined this
issue concluded that cannabis use in adolescence was associated with an increased risk of schizophrenia in adulthood, after accounting for potential confounding factors. There appears to be a dose-response relationship, and the effect is specific, with rates of depression not being associated with cannabis use. According to one review, cannabis use confers a two-fold increase in risk of schizophrenia or schizophreniform disorder.

CNS disorders such as Huntington’s disease, cerebrovascular disease, brain tumours and multiple sclerosis may produce delusions. Psychosis is over-represented in epilepsy, particularly chronic temporal lobe. Other causes include endocrine disorders (Cushing’s, hypo- or hypercalcaemia, hypopituitarism), connective tissue disease (systemic lupus, Sjogren’s syndrome, temporal arteritis, antiphospholipid syndrome), heavy metal toxicity and porphyria.

_Treatment_ requires identification of the offending agent or the underlying condition. Antipsychotics offer short-term relief of symptoms.

Matthew, a 30-year-old man, was brought to the hospital by police after aggressive behaviour toward his flatmates. He had become increasingly suspicious over 2 weeks, muttering something about the Russian Mafia. He had not ventured out of his room for 2 days. When his friends tried to force him to eat, he became violent and accused them of collusion with the Mafia.

Matthew was dishevelled, under-weight, hostile and threatening. He was vigilant and easily startled by the sound of passing cars. Admitted involuntarily, he was treated with a combination of a benzodiazepine and an antipsychotic. As his arousal settled, mental status examination revealed persecutory and referential ideas. Matthew believed that drug Mafiosi wanted to coerce him into their activities by using video, and were communicating through television programmes. He heard commands, which he was struggling to evade.

He had had a brief admission in an intoxicated state. His occupational and social histories were both unstable. Drug use history was not immediately available, but Matthew had venepuncture marks on both arms. Other than poor attention and concentration, his cognitive status was normal. Tachycardia was the sole physical abnormality. Urine drug screen revealed stimulants. Later, it became known that he had used large amounts of ‘Speed’ for many months.

Matthew was diagnosed with schizophrenia-like psychosis secondary to stimulants, and treated with Haloperidol (up to 10 mg/day). His symptoms receded over one week. He was advised to continue the medication for 3 months and to enter a drug counselling programme.

**Mood disorder due to a general medical condition or substance-induced**

This resembles a primary manic or depressive episode (see Chapter 10). Organic factors should be investigated in any patient with depression of late onset, atypical presentation, association with medical illness or non-response to conventional treatment. Mania is less likely to be secondary. Criteria for an affective episode may not be fully satisfied but predominant symptom are indicated by these subtypes: with depressive features, with major depressive-like episode, with manic features, or with mixed features.
Depression may be difficult to diagnose in patients with neurological disorders since many of them produce symptoms resembling it (diminished pleasure and interest, weight loss, insomnia, agitation, retardation, fatigue, impaired concentration). Experiential depressive features are the most robust indicators of an actual syndrome. Patients with dementia may, however, be unable to describe their symptoms, and depression must then be inferred.

Secondary depression is commonly caused by toxic or metabolic factors, with prior history of depression increasing its risk substantially. Medications, especially antihypertensives, are a notable cause. In fact, no antihypertensive with a central effect is exempt from risk. Other drugs leading to depression are corticosteroids, hallucinogens, antipsychotics and amphetamines (on their withdrawal). Several drugs (e.g. corticosteroids, L-dopa, tricyclic and other antidepressants) can trigger manic episodes, especially in people with an underlying bipolar illness.

Endocrine disorders should always be considered. Hypothyroidism commonly produces depressive features, and may also cause cognitive decline and overt psychosis. A severely hyperthyroid patient may come across as manic. An elderly person with hyperthyroidism may appear apathetic and withdrawn, thus creating diagnostic confusion. Psychiatric disturbance, particularly depression, is common in Cushing’s syndrome. Long-term corticosteroids or ACTH may produce euphoria or, less frequently, mania, depression or delirium. Depression can be a correlate of cerebrovascular (see below) and basal ganglia conditions.

Treatment of secondary depression requires remediating the underlying physiological abnormality or discontinuing the offending medication. If depression persists antidepressants or ECT may be necessary.

Treatment of acute secondary mania may call for antipsychotics. With persistent mania or recurrent episodes, a mood stabilizer is usually effective.

Catatonic disorder due to a general medical condition

Catatonia due to the direct physiological effects of a general medical condition is typified by such features as motor immobility (posturing, waxy flexibility, stupor), excessive motor activity (catatonic excitement), extreme negativism, mutism, peculiar voluntary movements (e.g. mannerisms), echolalia and echopraxia.

Many medical conditions can cause catatonia, especially neurological (e.g. neoplasms, head trauma, cerebrovascular disease, encephalitis) and metabolic (e.g. hypercalcaemia, hepatic encephalopathy, homocystinuria, diabetic ketoacidosis). Prevalence and onset reflect those conditions. In the differential diagnosis, consider antipsychotic-induced movement disorders (including neuroleptic malignant syndrome), catatonic schizophrenia and mood disorder with catatonic features.

Anxiety disorder due to a general medical condition or substance-induced

The essential feature is significant anxiety due directly to the physiological effects of a general medical condition or substance. Symptoms include prominent generalised anxiety, panic attacks, social phobia or obsessions and compulsions. Diagnosis is not made if anxiety occurs during delirium only.

Generalised anxiety and panic are usually caused by endocrine disorders (e.g. hyper- and hypo-thyroidism, phaeochromocytoma, fasting hypoglycemia, and hypercortisolism) or psychoactive substances. A common cause is intoxication (e.g.
caffeine, cocaine or amphetamines) or withdrawal from substances that depress the CNS (e.g. alcohol and sedatives). Uncommon causes are brain tumours in the vicinity of the third ventricle, trauma, cerebrovascular disease, migraine, encephalitis, multiple sclerosis, Parkinson’s disease, Huntington’s disease, Wilson’s disease, and epilepsy involving the diencephalon. Other causal factors are pulmonary embolus, chronic obstructive pulmonary disease, aspirin tolerance, collagen-vascular disease and brucellosis. Anxiety is occasionally the only symptom of vitamin B₁₂ deficiency, demyelinating disease and heavy metal intoxication.

Obsessive-compulsive symptoms are associated with Tourette’s syndrome, Sydenham’s chorea, anoxic injury to the basal ganglia, post-encephalitic parkinsonism, neuroacanthocytosis and other basal ganglia disorders. Treatment is of the underlying condition, as well of the anxiety symptoms themselves. Conventional anxiolytics and β-receptor antagonists are helpful. Obsessive-compulsive symptoms are treated with behaviour therapy and SSRIs.

**Personality change due to a general medical condition**

Change in personality functioning may manifest as amotivation, impulsivity or disinhibition. To be causally linked, the general medical condition must predate onset of personality change, and there should be no clouding of consciousness, significant loss of intellectual abilities, obvious mood disturbance, or prominent delusions or hallucinations.

Common causes are focal lesions of the brain or endocrine disorders (hypothyroidism, hypo- and hyperadrenocorticism). Head trauma is also important, as is subarachnoid haemorrhage, especially with an anterior communicating aneurysm. Brain tumours occasionally induce the syndrome. Its occurrence with temporal lobe epilepsy has been extensively debated, and Geschwind’s syndrome (tendency to write copious notes, circumstantiality, stickiness in personal relationships, preoccupation with religious themes and altered sexual behaviour) is described.

The pathological process determines clinical features. A common pattern is emotional lability and impaired impulse control and social judgement. The patient may be belligerent, and show temper outbursts or sudden bouts of unprovoked crying. Euphoria may mimic hypomania although the patient usually does not report feeling joyful. There may be socially inappropriate behaviour (e.g. sexual indiscretion), with little concern for its repurcussions; inappropriate jocularity and facetiousness and, extremely, a coarse manner or antisocial activity. A second pattern is indifference and apathy: the patient shows no interest in customary pursuits and is unconcerned with immediate events. As both patterns may be produced by frontal lobe damage (the first with orbitomedial, the second with dorsolateral prefrontal lesions), they are labelled ‘frontal lobe syndromes’.

A third pattern, seen with temporal lobe epilepsy, is excessive use of words in writing and speech, preoccupation with religious themes and, occasionally, aggressiveness. Suspiciousness or paranoid ideas but not amounting to delusions is another picture encountered.
The clinical features depend principally on the nature and localization of the pathological process. DSM-IV includes these subtypes: labile, disinhibited, aggressive, apathetic, paranoid, or combined. These often coexist with mild cognitive dysfunction (e.g. inattention and slight memory impairment), with irritability and suspiciousness also often present.

Course and prognosis depend on cause. Personality change may be transient (e.g. following chronic intoxication) or persistent (secondary to structural brain damage). In brain tumour or Huntington's disease, dementia may ensue. Some patients require custodial care or supervision to prevent any adverse consequences of impulsivity and inappropriateness (e.g. social ostracism or legal difficulties). Treatment focuses on the underlying condition. Medication may be indicated: tricyclics or SSRIs for organic emotionality; SSRIs, lithium, carbamazepine or propranolol for aggressiveness (although efficacy has not been confirmed). Patients usually need counselling, including discussion about a change of job or early retirement. The family needs support and advice on minimising their relative's inappropriate conduct.

Dementia

General aspects of dementia, including diagnosis and treatment, are dealt with in Chapter 22. The commonest cause in Western societies, Alzheimer’s disease (AD), accounts for about 50% of cases. Some of the non-Alzheimer causes of dementia (see Table 17.2) are discussed below.

Vascular dementia (VaD)

Probable vascular dementia is diagnosed when dementia is causally related to cerebrovascular disease. There may be clinical or neuroradiological evidence of single or multiple infarcts, multiple lacunae, widespread white matter lesions, or their combination. VaD was considered the result of multiple infarctions and referred to as multi-infarct dementia. While this is the commonest mechanism, we now know that it may arise from non-infarction ischaemic events and haemorrhage.

Factors that predispose to cerebrovascular disease and stroke also elevate the likelihood of VaD. They include increasing age (>60), male sex, Asian and Black ethnicity, hypertension, coronary artery disease, diabetes mellitus, hypercholesterolaemia, tobacco smoking, atrial fibrillation, mitral valve prolapse syndrome and, possibly, genetic factors. The latter are most strongly associated with cerebral autosomal dominant arteriopathy and subcortical infarct and leukoencephalopathy (CADASIL).

In cerebrovascular disease, age and premorbid intellectual level may influence the development of dementia. A series of strokes may occur, causing neurological deficits; transient ischaemic attacks in the setting of hypertension can also contribute. Stepwise deterioration is usual but, in the presence of subcortical pathology, the course tends to be more insidious. Personality and memory may be well maintained in early stages, but frontal problems of executive function, organisation and planning will be evident. Focal deficits include visuospatial and language disorders, identified with non-dominant and dominant hemisphere pathology respectively.
Assessment is geared toward identifying the extent of disabilities and contributing factors. CT and/or MRI are central to diagnosis, while neuropsychological assessment assists in the differential.

Management of risk factors can significantly reduce the incidence or, if dementia is present, halt progress and even achieve improvement. Control of hypertension is a critical protective factor. Control of other risk factors (hyperlipidaemia, platelet aggregation, carotid disease) may have a stabilising effect. Antiplatelet agents (e.g. aspirin or ticlopidine) are often recommended; anticoagulants may be indicated (e.g. in atrial fibrillation). Use of specific drug therapy is limited, with some outcome data to support the use of an acetylcholinesterase inhibitor (e.g. donepezil or rivastigmine). Non-cholinergic drugs that have been tried include pentoxyfylline and calcium channel antagonists (e.g. nifedipine, verapamil), but with limited success.

Dementia with Lewy Bodies (DLB)

There has been an upsurge of interest in DLB. Lewy bodies are intracytoplasmic eosinophilic inclusions that were first described by Frederick Lewy in 1914 in cells of the substantia nigra and other brainstem nuclei in patients with Parkinson’s disease (PD) and were considered pathognomonic of this disease. In the 1960’s, similar lesions were described in the neocortex and linked to dementia. A major component of Lewy bodies is a protein called α-synuclein. DLB is now considered to be relatively common, with neocortical Lewy body pathology being reported in up to 25% of dementia autopsies.

Clinical presentation varies, and surprises at autopsy are common. A group of experts has set these criteria:

- Progressive cognitive decline (of attentional and visuospatial ability, memory, frontal executive functions)
- Fluctuating cognition; recurrent visual hallucinations; Parkinsonism
- Other features that support diagnosis (falls, syncope, transient loss of consciousness, antipsychotic sensitivity, delusions, other hallucinations)
- Not so likely with evidence of stroke, other physical disease or brain disorder

DLB is much more common in males. Cognitive decline interferes with social and occupational function, but in the early stages memory loss may be overshadowed by attentional and visuospatial deficits and problems with frontal organisational tasks. Duration of illness averages 6 years compared to 10 for Alzheimer’s.

Cognition fluctuates in most cases. Variations in attention and alertness are pronounced, recurrent visual hallucinations are typically well formed, and motor features of Parkinsonism may precede the dementia. Secondary delusional beliefs may manifest. A neuropsychiatric presentation may occur. Parkinsonian features include rigidity and bradykinesia, with shuffling gait and falls, but less tremor and asymmetry than in PD. These features occur within a short time of dementia, particularly in the elderly, and in some constitute the presenting picture.

There is no specific treatment for DLB. There is a limited role for trialling an acetylcholinesterase inhibitor such as donepezil. Associated psychotic symptoms are
managed with antipsychotics (used cautiously), and depression with either tricyclics or SSRIs. As these patients are acutely sensitive to antipsychotics, developing severe parkinsonism, clozapine, quetiapine or aripiprazole are preferred drugs if antipsychotics are to be used. Restlessness and wandering may respond to L-dopa or beta-blockers.

**Fronto-temporal dementia (Pick and non-Pick)**

Some dementia patients in their fifties do not have cognitive impairment typical of Alzheimer’s, but insidious onset of behavioural and personality changes instead. This suggests either a clinical depression or a frontal lobe lesion. Some cases at postmortem have Pick bodies (cytoplasmic basophilic inclusions), indicating fronto-temporal dementia (FTD), which has been subdivided into: Pick’s, non-Alzheimer non-Pick’s, striato-nigral degeneration, corticobasal ganglionic degeneration, hereditary dysphasic dementia, motor neuron disease with dementia and progressive subcortical gliosis.

Characteristic features are apathy, reduced initiative, lack of concern, socially inappropriate behaviour and disinhibition, poverty of ideas, poor planning and organisational skills, variable verbal fluency and memory impairment (visuospatial function remains intact). Hypochondriacal, obsessional and paranoid symptoms along with hyperphagia, restlessness and distractibility are also seen. There are two broad groupings, one with slowness and apathy, and the other with restlessness, overactivity, distractibility and disinhibition. Pathology predominantly affecting the dominant hemisphere will show aphasia as the first severe clinical abnormality.

Neurological signs are usually absent in early stages but frontal release signs become evident with progression, along with signs including Parkinsonism and rigidity, autonomic abnormalities, dyspraxia, dystonia, tremor, dysarthria and disordered eye movement. Onset is a few years earlier than for Alzheimer’s. Men and women are equally affected. A family history of dementia in one in two cases contrasts with only one in eight with Alzheimer’s. It has been estimated that frontal dementias accounts for 15% of presenile dementias.

A 45-year-old policeman was brought to a neuropsychiatry clinic by his wife because of change in personality and deterioration in work over 2 years. Formerly well regarded by his colleagues, he had become increasingly careless, absenting himself without notice and leaving his paperwork incomplete. He had twice turned up in casual clothes, becoming angry when reprimanded by his superior officer. He made sexist comments in the presence of female colleagues. He was finally suspended when he asked one of them for sexual favours.

His wife reported that he had become most careless at home and could not be relied upon even to drop the children at school. He did not shower for days and, if she did not intervene, would wear dirty clothes with little concern. He had sold his car, purchased a mere six months previously, and taken out a loan for a better car. He had become intolerant of the children and was irritated by any boisterous play. He had become sexually demanding of his wife and would embarrass her in front of the children. His suspension from work did not seem to affect him. He refused to accept that he had a problem and had resisted attending. Past history was unremarkable except that his father had had memory problems in his 50s and died from a myocardial infarction. No other neurological problems were reported.
He presented as untidily dressed, fatuous and detached. However, he later became restless, playing with his clothes and with the instruments on the examiner’s table. He was discursive, wandering off the point. There was no evidence of elation, pressure of speech or psychotic symptoms.

Neurologically, the only abnormalities were a right palmomental reflex and a positive glabellar tap. He had difficulty performing complex hand sequences and showed perseveration. Neuropsychological assessment pointed to problems in attention, concentration, frontal-executive functioning, and dysfunction in expressive language. His memory was poor, with recognition better than free recall. A CT scan showed moderate atrophy largely restricted to the fronto-temporal regions bilaterally, the left side affected more severely. A PET showed hypometabolism in the fronto-temporal cortices bilaterally, with the parietal and occipital cortices relatively normal.

With diagnosis of fronto-temporal dementia he retired on medical grounds. Two years later he had deteriorated, the CT scan showing extensive atrophy.

Subcortical dementias

This concept is not universally accepted but serves to identify a number of dementing disorders lacking the features of cortical deficits (e.g. aphasia, agnosia and apraxia) so clearly seen in Alzheimer’s disease. Characteristic are disordered memory, poor attention, slowed up information processing, poor verbal fluency, impaired organisational and planning performance and abnormal visuospatial skills. The principal conditions are progressive supranuclear palsy (PSP, Steele-Richardson-Olszewski syndrome), Huntington’s disease and Parkinson’s disease.

Progressive supranuclear palsy (PSP)

This progressive dementing syndrome has its onset in the 50s and 60s, more commonly in men, and presents with typical neurological features: axial rigidity with an erect posture and tell-tale food stains on the patient’s clothing, rather than the stooped, flexed posture of Parkinson’s disease. Initial reduction in down-gaze is then followed by impaired up-gaze, pseudobulbar palsy, mask-like facies, brisk jaw jerk and palatal and pharyngeal reflexes, dysphagia and dysarthria.

Huntington’s disease

This is a progressive degenerative disorder with alterations in behaviour, cognitive function and movement. An autosomal dominant, the responsible gene is located on the short arm of chromosome 4. We can identify at-risk people by determining the number of CAG trinucleotide repeats; less than 34 represents no risk, 34–40 an intermediate risk, and above 40 likely expectation of developing the disease.

Average age of onset is the early 40s, but the disease can develop throughout life. Anticipation occurs with successive generations showing an increase in the number of trinucleotide repeats, and an earlier age of onset associated with paternal transmission. Higher numbers of repeats are linked with an earlier age of onset and a shorter course, generally 10–12 years, but occasionally over 20.

Disordered mood may precede the onset by several years, often with irritability. When established, the disease may lead on to schizophrenia.

The movement abnormality is a progressive choreiform and choreoathetoid pattern of irregular involuntary movements affecting the proximal and peripheral limbs, the face and tongue; swallowing and speech can be significantly
affected, raising the risk of aspiration. In the latter stages, movement is often much reduced, and patients die from infection, progressive wasting and cardiac failure.

Dementia is subcortical in type, and memory often preserved in early stages, with difficulties in executive function, planning and organisational tasks. Dementia may be the presenting feature but abnormal movements become apparent within a few years.

Pathology is centred on the caudate nuclei but also involves the cerebral cortex. Atrophy is visible on neuroimaging.

There is at present no specific treatment, except for symptomatic measures. Several drugs to delay the onset and slow progression have been developed in animal models and are undergoing therapeutic trials.

**Parkinson’s disease (PD)**

Dementia occurs in one in five in those with PD, more so in the elderly with a later age of onset and where disease progresses rapidly. Subcortical dementia produces impairment of frontal executive function (planning and organisation), visuospatial function along with speed of information processing, verbal fluency and memory. In some cases associated deficits suggest cortical involvement with aphasia, agnosia and apraxia, and a link with Alzheimer’s disease exists. Overlap with Lewy Body dementia and Alzheimer’s is important.

**Alcohol**

Alcohol is a pivotal factor in 6% of dementias, and in about 10% of those presenting for treatment. The underlying neuropathology includes Wernicke’s encephalopathy, cerebral atrophy, thinning of the corpus callosum and cerebellar changes. Associated factors include thiamine deficiency, metabolic disorder (a low-grade hepatic encephalopathy), nutritional deficiencies and a history of head injury.

Cognitive impairment is global, except for language. Frontal lobe impairment with affective blunting and poor organisational and planning abilities are prominent. Abstinence leads to improvement, suggesting that alcoholic dementia may be, at least in part, reversible.

**Normal pressure hydrocephalus**

This disorder which may account for about 5% of dementing patients, is one of the few potentially reversible dementing syndromes. Patients usually present with progressive dementia, gait disorder and sphincteric disturbance (urinary incontinence). Dementia may develop over months, with prominent memory difficulties and slowing of mental processing, sometimes suggestive of depression. More widespread deficits become apparent and there may ultimately be a catatonic-like picture. Episodes of confusion may be superimposed on the dementia. The gait disorder is characterised by small zig-zag steps and a tendency to repeated falls and difficulty turning. Spasticity and extensor plantar reflexes may develop later. The underlying pathology includes former subarachnoid haemorrhage, traumatic brain injury and meningitis, but often no identifiable pathology is found.

CT scanning reveals ballooning of the anterior horns of the lateral ventricles, often with periventricular lucency. Lumbar puncture may assist diagnosis. CSF monitoring over 24 hours helps to identify patients who are suitable for shunting.
Treatment involves introduction of a ventriculo-peritoneal or ventriculo-atrial shunt into the right lateral ventricle; a favourable outcome occurs within a few weeks in 40% of cases.

Trauma, anoxia and infection

This group, about 3% of dementia patients, includes traumatic brain injury due to motor vehicle accidents, or survivors of anoxic episodes secondary to cardiac arrest, hypoglycaemic coma, drowning or asphyxiation; and those with acute encephalitis, neurosyphilis or HIV/AIDS.

Prion diseases

The transmissible spongiform encephalopathies or prion diseases include both human and animal forms. The field has gained significance with suggested transmissibility across species, and of humans developing a variant of Creutzfeldt Jakob disease from animals with bovine spongiform encephalopathy (BSE) ('Mad Cow disease'). Other human forms are Gerstmann Strausssler Sheinker disease and kuru. Animal forms include scrapie in sheep and goats, mink encephalopathy and BSE.

Creutzfeldt Jakob disease

Though most cases are sporadic, about 15% have a positive family history. Both sexes are affected and onset is generally in the 60s. An iatrogenic form has been identified, with transmission of abnormal protein through neurosurgical procedures, dural grafts, human growth hormone extracts and corneal transplants. The disease is rare – one case per million – but this may be a conservative estimate. Course is rapid, with 75% of patients dying within 12 months.

A rapidly progressive dementing picture may simulate a confusional state or there may be a prodrome with anergia, anxiety and depressive features. Psychotic symptoms are frequent. Neurological features include ataxia with motor weakness and rigidity, cortical blindness, myoclonic jerks, dysarthria and possible seizures. A number of subtypes depends on the predominant neurological features.

Of major concern is the suggested development in humans of a variant of Creutzfeldt Jakob disease in the animal form of BSE. A major outbreak of BSE occurred in the United Kingdom in 1986 and about 10 years later reports began of cases of Creutzfeldt Jakob disease with a much younger age of onset, prominent psychiatric symptoms and a protracted history.

Delirium

Delirium (from delirio; to rave) is one of the frequently encountered neuropsychiatric syndromes. Its prompt detection and management are vital (it is often undiagnosed or misdiagnosed as dementia or psychosis). Onset usually heralds physical illness and calls for immediate medical attention. Delirium is preferred to its many synonyms (acute confusional disorder, acute brain syndrome, toxic or metabolic encephalopathy, toxic psychosis) to represent transient global cognitive impairment of presumed organic aetiology.
Prevalence is 1% of the general population but is much higher in hospitalised patients, varying from 5–15% depending upon the nature of the medical or surgical ward; in elderly inpatients it may reach a third.

Children and the elderly are particularly vulnerable – children when suffering febrile illness and the elderly when subject to intercurrent infection or drug mismanagement. The presence of dementia raises its risk with 40% of such patients delirious on admission. Conversely, one in four confusional states is associated with dementia. Depression, acute psychological stress, sleep or sensory deprivation and bereavement increase the risk of delirium, as do brain damage, substance abuse, drug or alcohol dependence and hearing or visual impairment.

**Clinical features** include a typically acute onset, developing over hours to three days, depending on aetiology. Duration may be a day to three or four weeks, with resolution depending on speed of diagnosis and treatment of underlying pathology.

The hallmark is clouding of consciousness – reduced alertness and awareness, impaired arousal and difficulty with attention (manifest as distractibility). These features can be difficult to identify in milder forms, when the patient misunderstands the admission process and clinical interview. There is often a disordered sleep-wake cycle, the patient somnolent during the day and restless and agitated as night approaches. Psychomotor activity ranges from apathy and inactivity to restless picking at bedclothes and objects, and marked agitation with hyperactivity, even aggression. The clinical picture may fluctuate during the day, with features more prominent towards evening.

Cognition is impaired, with fragmentary and erratic performance, poor registration of information and faulty recall, and disorientation for time and place. Language functions may be impaired for naming and paraphasia can occur. Thinking is often disturbed with rambling, circumstantial, and repetitious irrelevant content. Poorly organised persecutory delusions may manifest but are usually transient.

Misperceptions, illusions and hallucinations are common, particularly visual. Affective responses vary: some patients are indifferent, others show considerable anxiety, agitation, fear, anger or depression. Rarely, a sense of elation dominates.

**Differential diagnosis** addresses the common dilemma of whether the patient has dementia or dementia complicated by secondary delirium. A detailed history with information from the family is critical. An acute psychotic disorder, schizophrenia, agitated depression (and occasionally mania) can be mistaken for delirium, or vice versa. Careful observation, examination and special investigations are then needed to clarify matters.

**Investigations**

All patients require investigation tailored to leads derived from the history and physical examination. The following are considered: full blood count and blood film, ESR, liver function tests, drug assays, urine test, blood culture, EEG, electrolytes, blood sugar, urinary drug screen, CSF examination, blood gases, CT scans and chest X-ray. Further specialised investigations may be necessary.
Treatment

It is important to treat delirium as an emergency, since the longer the delay the greater the chance of morbidity and death. The underlying cause is identifiable in 9 out of 10 cases. Common causes are listed in Table 17.4.

A general principle of management is that the primary cause be identified and treated. Treatment itself, particularly drug interactions, however, may compound the problem if not carefully planned and monitored.

General measures include attention to hydration, nutrition, ventilation, temperature control, skin care to prevent decubitus ulcers and physiotherapy. Appropriate environmental features are a well-lit room, dim light at night, a calendar and clock, a radio or television for sensory stimulation, familiar nurses and regular visits of family and friends. Reorientation, provision of basic information, careful observation and prevention of injury are all relevant.

Management of an agitated, restless or fearful patient is a challenge. If at all possible, restraint is minimised since it often agitates the patient further. Shackles should never be used, while tying the patient to the bed or to a chair is potentially hazardous. If necessary, a restraining jacket can be used. When intravenous or central lines, catheters or nasogastric tubes are in place, and forcible removal by the patient could cause injury, the hands may be loosely bandaged or back slabs applied to the arms.

Drug treatment of any agitation may be indicated. A high potency antipsychotic such as haloperidol is safe. Depending upon weight, age and physical condition, the initial dose is 0.5 – 5 mg i/m, repeated hourly if the agitation persists. When the patient is calmed, oral medication, usually in divided doses, is substituted. Other antipsychotics have been used in this scenario. Benzodiazepine also has its proponents. Excess sedation can, however, aggravate delirium. After the confusion has receded, medication is continued for about 3 – 5 days.

Regular review is essential, given the risk of intercurrent infection, dehydration or anaemia. When the delirium has resolved, the patient is reassured and supported to deal with fragmented, frightening recollections. In the elderly, review of the cognitive status is done to check for any underlying dementia.

A 70-year-old woman found wandering in the neighbourhood was brought to the hospital by the police. She could give her name, but did not know her address or where she was, and thought the year was 1949. She repeatedly asked, ‘Where is James?’ She was agitated, with rapid breathing. She became frightened when being put to bed and looked anxiously at the dimly lit wall behind the bed as if responding to visual illusions and/or hallucinations. A medical review revealed low grade fever, tachycardia, hypertension, tachypnoea and crepitations in the chest. Lobar pneumonia was later confirmed. She was also in pre-renal uraemia and the blood glucose was elevated. CT scan of the brain was normal but an EEG showed diffuse slow waves.

She was admitted to a medical ward with the diagnosis of delirium secondary to pneumonia, with dehydration on the background of hypertension and diabetes. With treatment, the sensorium gradually improved and she was fully oriented five
days later. She was amnesic for the first three days but showed no evidence of continuing cognitive deficits. She was soon able to return to independent living.

Specific medical condition

Neuropsychiatric aspects of cerebrovascular disease (CVD)

CVD follows ischaemic heart disease and cancer as the third leading cause of death in people aged 50 and over. Stroke patients often experience a catastrophic decline in physical, sensory or language ability as well as neuropsychiatric consequences – cognitive, mood, behavioural and personality change. Loss of independence and disability often result in grief and anxiety states, as well as posing significant stress for carers. Vascular dementia has been considered earlier. We offer an account here of non-cognitive psychiatric presentations.

Typical major depression occurs in about a quarter of patients in the first few months following a stroke. Those with cortical strokes closer to the frontal lobes, particularly left anterior, are particularly vulnerable, according to one group of investigators, but the evidence for this is not consistent and meta-analyses have not supported this anatomico-functional relationship. Its diagnosis and treatment are crucial as depression worsens the physical prognosis and prolongs disability. Treatment is standard although the patient is more sensitive to medication side-effects. While biological factors probably lead directly to post-stroke depression, the psychological and social impact on patient and family are also notable.

The high prevalence of depression in stroke patients, and the report of white matter disease and basal ganglia vascular abnormality in late-onset depression has prompted much discussion on a neurological subtype of depression which is secondary to vascular disease (so called, Vascular Depression). Their inter-relationship is complex, however, and a direct aetiological model may be too simplistic. Studies do not consider confounding factors into consideration, including the mediating role of physical ill-health in general. Moreover, depression is known to worsen vascular disease, reversing the direction of the relationship. There may also be common pathophysiological factors such as inflammation. The concept therefore awaits further appraisal.

Labile affect (or pathological laughing and crying) is common. Usually the patient cries inappropriately and precipitously without an emotional cue, and is embarrassed and distressed by it. Lability usually wanes over time. While it may be amplified by depression, lability should not be mistaken for it in the absence of other features. Some patients respond to a tricyclic (imipramine or amitriptyline, 25–75 mg/day) or an SSRI.

A syndrome of ‘apathy’ involving loss of interest and motivation is differentiated from depression by the patient denying sadness, and sleep and appetite remaining normal. The patient may also demonstrate unawareness of one side of their body or visual field (hemi-neglect), or indifference to their disability (anosognosia). These clinical pictures are more apt to arise with parietal lobe infarction, particularly right-sided. Personality change is common, taking various forms: apathy, impulsivity, aggression or coarseness. Inability to perceive or express emotion (aprosodia) can follow strokes of the right frontal and temporo-parietal
regions (corresponding to Broca’s and Wernicke’s language areas of the dominant hemisphere).

First occurrence of mania in the aftermath of a stroke is rare but may result from a subcortical lesion in the limbic area. Bipolar disorder may occur de novo, tending to affect those with a history of depression and/or a family history of bipolar disorder.

Psychosis after stroke and in the absence of cognitive impairment is unusual. However, as noted earlier, persecutory delusions and hallucinations can occur in VaD as part of the tendency to greater confusion in the evening (‘sun-downing phenomenon’), or as an aspect of delirium.

Human immuno-deficiency virus (HIV) infection and acquired immuno-deficiency syndrome (AIDS)

The diverse effects of HIV reflect the many mechanisms of its pathophysiology. HIV invades the brain tissue soon after infection and the later immunological compromise of AIDS can lead to secondary brain damage through infection, tumours or vascular complications. The patient, adjusting to a serious physical illness, also has to wrestle with the responses of relatives and suffer the effects of stigma. Predisposing personality disorder or substance abuse complicate the picture.

There is conflicting evidence on whether mild cognitive impairment is present soon after acute HIV sero-conversion, but in up to 70% of cases cognitive deficits occur in the late stages. HIV Associated Dementia (HAD) has a prevalence of 25–60% until the time of death and is an AIDS–defining condition. This subcortical dementia involves HIV infection of the deep white matter and basal ganglia, resulting in personality change as well as slowing and deterioration of cognitive function and movement. HAD is occasionally a presenting sign of HIV, in which case life expectancy is an average 6 months.

Delirium occurs frequently in AIDS due to the metabolic effects of secondary infections and neoplasms. Fluctuations in consciousness and disorganised or apathetic behaviour, with or without psychosis, indicate probable delirium.

Depression has a rate of 30% for the duration of the disease. The differential diagnosis includes cognitive slowing due to HAD, and malaise associated with systemic illness or an adjustment disorder. Suicide rates are increased 30 to 60 times. Mania is less likely to present de novo than as a consequence of delirium or as a side–effect of medication. Anxiety is common. Often it relates to uncertainty about the future and the possibility of infecting others. Patients can erroneously conclude that somatic symptoms relate to progression of the AIDS.

Psychotic episodes (as with mania) may indicate the chance association of a pre-existing psychotic illness but can be a result of delirium, subtle cognitive dysfunction or focal brain effects of secondary disease. Occasionally, a schizophreniform disorder in the absence of an organic cause may be due to direct HIV brain infection.

Treatment includes antiretroviral agents such as Zidovudine, which decrease the rate of neuropsychiatric disorder. Antiviral drugs, however, can produce serious neuropsychiatric complications as well, as with DDC (dideoxynosine and dideoxycytidine). Prompt identification and treatment of associated medical conditions is important. Low dose high potency antipsychotics are appropriate to
manage psychosis, mania and delirious agitation, but increased sensitivity to side-effects is problematic. Antidepressants are effective for depression; psychostimulants (e.g. Methylphenidate) can assist apathetic syndromes. Lithium may be appropriate for recurrent and resistant depression, but may worsen AIDS-related diarrhoea.

Psychological therapies help patient and carers to deal with anxiety, depression and grief, while support groups offer education and support.

**Traumatic brain injury (TBI)**

TBI is the most common cause of neurological presentation, after headache, in young adults. Neuropsychiatric problems are frequent in TBI and may be disproportionate to actual neurological deficit. They include cognitive impairment (dementia, amnestic syndrome, dysphasia), mood disorder (depression or mania), personality or behavioural change, anxiety disorder (generalised anxiety disorder, post-traumatic stress disorder, obsessive-compulsive disorder) or, rarely, psychosis. The psychiatric outcome of TBI has many determinants, e.g. type and extent of injury, duration of altered consciousness and period of post-traumatic amnesia. Outcome is further modulated by education, premorbid personality, culture, support, medical and rehabilitative care, and financial complications. The patient’s coping skills and locus of control are also pertinent. Furthermore, the TBI must be considered in the context of the stage of the life cycle of both patient and family.

Controversial is the validity of Persistent Post-Concussional Syndrome (PCS) as a diagnostic entity. 10–15% of patients with mild brain injury have persistent symptoms beyond a year, typified by somatic (headache, dizziness, fatigue, insomnia), cognitive (concentration, memory and executive dysfunction), perceptual (sensitivity to noise and light) and emotional (depression, anxiety, irritability) features. There are few objective signs and neuroimaging is usually normal. These patients often have a history of psychiatric problems and of extensive disruption because of the accident. Other risk factors are female sex, litigation, poor socio-economic status, previous injury and associated somatic response. Both physiological and psychological factors operate in PCS; the point at which physiogenesis becomes psychogenesis is hard to delineate, and may have iatrogenic sources.

**Epilepsy**

While epilepsy is compatible with good mental health in most people, psychiatric disturbance in this group greatly exceeds that in the general population. About a third have psychiatric problems (a half of those with temporal lobe epilepsy). A general practice survey found that 17% had marked social problems, and 20% could not work or were capable of restricted employment only. In a survey of all children on the Isle of Wight, the rate of psychiatric disorder in those with uncomplicated epilepsy was four times that of controls.

The range of disorder is wide including depression, anxiety, psychosis and personality disturbance. Depression is common in the inter-ictal period, but has also been linked to the peri-ictal period. Suicide rate is three times the general population and most commonly due to overdoses on anticonvulsants. Depression in epilepsy is usually treated with SSRIs and SNRIs. Tricyclics may be used provided the risk of overdose is kept in mind. Even though some anticonvulsants are used as mood
stabilisers, certain drugs such as vigabatrin, topiramate and phenobarbitone are sometimes associated with depression soon after initiation. Antidepressants lower the seizure threshold, with the risk being higher with tricyclics and MAOIs. ECT can be used for severe depression in these patients and does not worsen the epilepsy.

Anxiety disorder, in particular generalized anxiety, is common. Some patients with sudden unexpected seizures develop agoraphobic symptoms. Occasionally, panic attacks with dissociative symptoms may be confused with complex partial seizures.

The link with schizophrenia-like psychosis (SLP) is notable. Epileptic-psychoses are considered as ictal, post-ictal and inter-ictal. Post-ictal psychosis begins hours or days after a flurry of seizures and is usually brief. Other patients develop brief inter-ictal psychoses – a pattern whereby epilepsy and psychosis alternate. This may be accompanied by EEG changes that have been termed ‘Forced Normalization’, in which the EEG abnormalities seen in the inter-ictal state tend to decrease or disappear during the psychotic period. There is considerable evidence that patients with epilepsy are at greater risk of developing chronic SLP, and that schizophrenic patients have a higher prevalence of epilepsy. This psychosis is usually difficult to differentiate from primary schizophrenia, although symptoms are largely paranoid-hallucinatory, and associated with catatonia, affective blunting, and volitional features; negative symptoms are lacking. Patients with temporal lobe epilepsy are more at risk of SLP (mediobasal temporal lobe epilepsy in particular). Risk factors may include intractable epilepsy, early onset, secondary generalisation of seizures, use of certain anticonvulsant drugs, developmental brain abnormalities and temporal lobectomy. SLP is managed with antipsychotics, but their potential to lower the seizure threshold should be appreciated, with the atypical antipsychotics, particularly clozapine, having greater propensity toward this.

There is no specific personality associated with epilepsy, but irritability and aggressiveness are common. Some patients with resistant temporal lobe epilepsy have features of Geschwind’s syndrome (see p. ).

Further reading
A good description of the fronto-subcortical circuits from a clinician’s perspective.

An influential book on neuroscience.

This text is good on psychiatric aspects of most medical conditions, particularly uncommon ones.

The classic to consult for most neurological problems.

A multi-authored text with authoritative chapters on most aspects of neuropsychiatry; recommended as standard reference.

Fig 2: T2-weighted and proton density (right) images of an axial brain MRI scan from an elderly patient with a Major Depression with melancholic features. The widespread white matter hypertensities are possibly ischaemic in aetiology. The Depression was resistant to drug treatment and responded partially to electro-convulsive therapy.
Table 17.1 Secondary mental disorders (DSM-IV)

**Cognitive disorders**
- Delirium
- Dementia
- Amnestic Disorder
  - Amnestic Disorder due to a general medical condition
  - Substance-induced Persisting Amnestic Disorder
  - Amnestic Disorder NOS
- Cognitive Disorder NOS

**Non-cognitive disorders**
- Psychotic disorder due to a general medical condition or substance
  - With delusions
  - With hallucinations
- Mood disorder due to a general medical condition or substance
  - With depressive features
  - With major depressive-like episode
  - With manic features
  - With mixed features
- Catatonic disorder due to a general medical condition*
- Anxiety disorder due to a general medical condition or substance
  - With generalised anxiety
  - With panic attacks
  - With obsessive-compulsive features
  - With phobic symptoms
- Personality disorder due to a general medical condition*
- Sexual dysfunction due to a general medical condition or substance
- Sleep disorder due to a general medical condition or substance
- Mental Disorder NOS due to a general medical condition or substance

*These alone are not diagnosed as substance-induced.

Table 17.2 Causes of dementia

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Frequencies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s disease</td>
<td>50%</td>
</tr>
<tr>
<td>Vascular dementia</td>
<td>15%</td>
</tr>
<tr>
<td>Mixed Alzheimer’s / vascular</td>
<td>20%</td>
</tr>
<tr>
<td>Lewy Body dementia</td>
<td>17%</td>
</tr>
<tr>
<td>Frontal dementias (Pick / non-Pick)</td>
<td>15%</td>
</tr>
<tr>
<td>Subcortical dementias</td>
<td>4%</td>
</tr>
</tbody>
</table>
  - Progressive supranuclear palsy             |             |
  - Huntington’s disease                       |             |
  - Parkinson’s disease                        |             |
| Alcohol                                      | 6%          |
| Normal pressure hydrocephalus                | 5%          |
| Trauma, anoxia, infections                   | 3%          |
| Prion disease                                | 2%          |
Table 17.3 Diagnostic criteria for delirium DSM-IV

A. Disturbance of consciousness (reduced clarity in awareness of environment) with reduced ability to focus, sustain or shift attention

B. Change in cognition (memory deficit, disorientation, language disturbance) or development of a perceptual disturbance not better accounted for by pre-existing, established or evolving dementia

C. Disturbance develops over hours to days and then fluctuates

D. Evidence from history, physical examination and laboratory findings that disturbance is caused by direct physiological consequences of a general medical condition. One of the following may be considered:
   - Direct physiological consequence of a general medical condition
   - Symptoms develop during substance intoxication
   - Medication is related to the disturbance
   - Symptoms develop during or shortly after a withdrawal syndrome
   - The delirium has more than one aetiology

Table 17.4 Causes of delirium

The mnemonic is DELIRIUM useful

**Drugs**
- Intoxication /withdrawal of alcohol, benzodiazipines, barbiturates, narcotics
- Anticholinergic drugs (atropine, benztropine, benzhexol); anticholinergic effects of antipsychotics and tricyclics
- Antihistamines
- Anticonvulsants (phenytoin, carbamazepine, clonazepam, valproate, vigabatrin)
- Antiparkinson drugs (amantadine, L-dopa, bromocriptine)
- Cardiac drugs (beta-blockers, digoxin, theophylline, diuretics, hypotensives)
- Anti-inflammatory drugs (non-steroidal drugs, steroids)
- Sympathomimetics (ephedrine, amphetamines)
- Antibiotics
- Antineoplastic drugs
- Other (e.g. Chlorpropamide, Cimetidine, Ranitidine, Lithium, Metrizamide)

**Endocrine**
- Diabetes; thyroid, parathyroid or adrenal dysfunction

**Epilepsy**
- Ictal or post-ictal

**Lung disease**
- Pneumonia, chronic obstructive airways disease, sleep apnoea

**Infection**
- Encephalitis, meningitis, syphilis, HIV, septicaemia

**Injury**
- Concussion, subdural, extradural haemorrhage, burns, general and cardiac surgery, fractured neck of femur

**Intracranial**
- Tumour, raised intracranial pressure
Renal
• Acute and chronic end-stage failure

Intestinal
• Carcinoma, obstruction, ileus

Unstable circulation
• Arrhythmias, congestive cardiac failure, myocardial infarction, hypertensive encephalopathy, hypoperfusion, blood loss, shock

Metabolic
• Hyponatraemia, hypokalaemia, acidosis/alkalosis, hepatic failure, dehydration

Table 17.5: The neuropsychiatric complications of HIV-AIDS
• Direct HIV brain infection: encephalitis, meningitis, white matter disease, neuronal cell death
• Secondary brain infection: viral, mycotic, protozoan, bacterial
• Neoplastic infiltration: e.g. lymphoma
• Vascular: embolism and thrombosis secondary to vasculitis and endocarditis
• Metabolic effects of systemic disease: hypoxia, ‘toxaemia’
• Iatrogenic: e.g. drug side-effects of nucleosides and neurotropics

Fig 1: T2-weighted (left) and proton density (right) images of an axial brain MRI scan from an elderly patient with a late-onset depression. The widespread white matter hypertensities are possibly ischaemic in aetiology.
Quality Dementia Care: No time like the present: the importance of a timely dementia diagnosis
Quality Dementia Care Series:
No time like the present: the importance of a timely dementia diagnosis

Dr Jill Phillips (University of Newcastle)
Professor Dimity Pond (University of Newcastle)
Dr Allan Shell (University of New South Wales)

September 2010
Foreword

The first step in addressing the health care needs of individuals with dementia is to provide an accurate and timely diagnosis. Unfortunately a timely diagnosis is not the experience of many people with dementia, their families and carers, and improving the capacity of the current primary care system to do better is a high priority for consumers.

Market research commissioned by Alzheimer’s Australia indicates that over 90% of Australians say they would be likely to visit their GP if concerned about their memory. For that reason, an important part of the advocacy of Alzheimer’s Australia in recent years has been to urge the Federal government and stakeholders in primary care to take action to better support GPs in the diagnosis, assessment and ongoing management of dementia. While individuals will vary in the choices they make, most people concerned about their memories and their families take the view that early intervention is necessary if they are going to be able to properly plan their finances, lives and care for the future.

To have effective primary health care for dementia, attention needs to be focused on adequate incentives and training for GPs and practice nurses to secure an appropriate response to those presenting with cognitive impairment, be that independently or through referral from family or other health care providers. Primary care should be able to address the chronic disease elements of dementia such as counselling, access to community base support, medical management of emergent disorders and legal and driving issues.

Alzheimer’s Australia is working to ensure that these issues are properly addressed between stakeholders and the Federal government. Meanwhile, this publication is a step towards assisting GPs in achieving the important goal of timely diagnosis.

Alzheimer’s Australia is grateful to Pfizer Australia for providing an unconditional grant that made the writing and publishing of this publication possible. With further support from Pfizer in 2011, Alzheimer’s Australia hopes to promote a series of GP training workshops that will include a major focus on the diagnosis and care of people with dementia.

Lastly, I should like to thank the authors of this report for writing this publication.

Glenn Rees
Alzheimer’s Australia
Introduction

The purpose of this paper is to outline the steps involved in the diagnosis of dementia and to promote timely diagnosis. This paper is aimed at general practitioners, practice nurses, other health professionals, policy makers, academics and those people concerned about their memory or that of a family member or friend. This paper updates and extends the Alzheimer’s Australia paper, Early Diagnosis of Dementia (March, 2007).
What is dementia?

Dementia is an umbrella term for a range of conditions (see ‘Types of dementia’). It is characterised by loss of memory, and impairments in thinking and problem-solving capabilities. Features may include impairment in language, memory, perception, and cognitive skills. These may result in loss of intellect, personality, rationality, social skills and normal emotional reactions [1-3]. Dementia results from degeneration of nerve pathways and the conditions associated with it are typically progressive [1,3].

Types of dementia

There are many different causes and, thus, types of dementia. The most common is Alzheimer’s disease, which is associated with distinctive changes in the brain tissue in the form of ‘tangles’ and ‘plaques’. While Alzheimer’s disease can develop in younger people, it is most common after the age of 65 years. It accounts for at least 50 per cent of cases [1].

Vascular dementia is thought to be the second most common form of dementia and is associated with problems of blood circulation in the brain. It may account for up to 20 per cent of cases [1].

Mixed dementia, which contains elements of both vascular dementia and Alzheimer’s disease, is also common. Elements of Alzheimer’s disease and vascular dementia are often both present upon autopsy [2].

‘Dementia with Lewy bodies’ accounts for about 15 per cent of all dementias [1] and is marked by fluctuating alertness and attention, hallucinations, falls and Parkinsonism or slowing of, and increased stiffness of, movement.

Frontotemporal dementia typically occurs between the ages of 45 and 65 years and can involve profound personality and behavioural changes and/or language impairment. It accounts for approximately 5 per cent of cases [1].

There are many other possible causes of dementia including alcohol-related dementia, Parkinson’s disease, Huntington’s disease and Creutzfeld-Jacob disease. An accurate diagnosis helps to maximise benefits from appropriate treatment, and enhance understanding about prognosis and symptoms.

A person complaining of memory loss or other cognitive changes, which are present on testing but not severe enough to have dementia, may be classified as having a Mild Cognitive Impairment (MCI). MCI is a relatively new concept and more research is needed to understand the relationship between MCI and later development of dementia. MCI does not always lead to dementia and can improve, even reverting to normal. Regular monitoring of memory and thinking skills is recommended in individuals with this diagnosis.

Common symptoms

The symptoms of dementia are not always obvious to the person or their family and friends. The early symptoms can include memory problems, difficulties in word finding and thinking processes, changes in personality or behaviour, a lack of initiative, and changes in day to day function at home, at work or in taking care of oneself. Symptoms will differ according to the type of dementia. As the condition progresses, symptoms may become more obvious and could include:

- increased memory loss
- decreased ability to perform routine tasks
- impaired judgement and ability to understand concepts or follow a plot
- learning and concentration difficulties
- altered sleeping patterns
- eating disturbances
- disorientation to time and space, and getting lost in familiar places
- focal neurological signs
- muscle rigidity [1].

How common is dementia?

In Australia, the number of new cases of dementia (incidence) is estimated to increase from 75,000 in 2010 to 385,000 in 2050 [1]. The total number of people with dementia in Australia (prevalence) is projected to increase from 257,000 in 2010 to over one million in 2050 [4]. Dementia prevalence is strongly age-related and, with an ageing population, it is estimated that the number of cases of dementia will more than double to around 565,000 over the next 20 years [4].

According to the Australian Bureau of Statistics (ABS) [5], dementia and Alzheimer’s disease was the third leading cause of death in 2008, having risen from sixth in 2003, and from seventh in 1999. The ABS reports the number of deaths due to dementia and Alzheimer’s disease to have increased 138 per cent from 3,427 in 1999 to 8,171 in 2008.

This paper does not include detailed information about all of the warning signs and it is recommended that consumers consult other sources for this information. Visit the Alzheimer’s Australia website at www.alzheimers.org.au for comprehensive information about dementia and care and available education and training.
### Risk factors associated with dementia

Many risk factors have been associated with dementia. Some are firmly established, while others remain unconfirmed and are the subject of ongoing research [1]. Drawing on a comprehensive literature review, Woodward et al. [6] presented the following risk factors for Alzheimer’s disease:

<table>
<thead>
<tr>
<th>WELL ESTABLISHED</th>
<th>LIKELY</th>
<th>LESS LIKELY</th>
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<tbody>
<tr>
<td>Old age</td>
<td>Hypothyroidism</td>
<td>Depression</td>
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<tr>
<td>Genetic factors:</td>
<td>Vascular risk factors:</td>
<td>Elevated homocysteine (a by-product of chemical reactions in the body)</td>
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<td>- Down Syndrome</td>
<td>- Smoking currently</td>
<td>- Fatty diet</td>
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<td>- Apolipoprotein E status</td>
<td>- High blood pressure</td>
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<tr>
<td>- Genetic mutations (rare)</td>
<td>- Diabetes (generally Type 2)</td>
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<td>- Family history of Alzheimer’s disease</td>
<td>- Atrial fibrillation</td>
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<tr>
<td>- Low birth weight for gestational age</td>
<td>- Obesity in mid life</td>
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The risk factors for vascular dementia include many of the above plus male gender, cardiac disease or major cardiac surgery, obesity, stroke, family history of vascular disease and elevated cholesterol. As yet, no amenable risk or protective factors for dementia with Lewy bodies and frontotemporal dementia have been identified [8].

### Protective factors associated with dementia

According to Woodward et al. [6], actions that may reduce the risk of developing dementia include control of the following factors:

- **Nutrition** – moderate to high intake of polyunsaturated and monounsaturated fats is associated with reduced risk for dementia. It is not yet clear whether B12 or folate supplementation reduces the risk, but it is prudent to check for and treat any deficiencies of these vitamins [6]. While there is no convincing evidence that antioxidants prevent dementia, it does not mean they are ineffective, rather there is currently a lack of evidence. Some studies suggest that omega-3 fatty acids (for example, fish oil) may reduce risk of dementia [7], as could moderate to high intake of polyunsaturated and monounsaturated fats [8]. A high intake of saturated fat is a risk factor for Alzheimer’s disease and vascular dementia [6].

- **Activity** – several observational studies have found an association between physical activity in mid to late life and a lower risk of cognitive decline and dementia. Physical activity has numerous health benefits and may reduce cardiovascular risk factors, improve blood flow to the brain and possibly stimulate nerve cell growth and survival [6]. Cumulative activities over a day, are found to be protective, and include those that involve socialising, mental activity and leisure such as: walking for pleasure or excursion; going to the cinema or to a restaurant, sporting venue, club, or church; community or volunteer work; visiting relatives or friends, and having visitors; going to classes; reading, watching TV, listening to the radio; knitting, music or other hobbies. Woodward et al. [6] also note that numerous studies suggest a high level of education is associated with a lower risk. It seems that complex mental activity throughout all stages of life may reduce the likelihood of developing dementia [11].

- **Vascular risk factors** – high blood pressure in mid-life has been identified as a risk factor. Treatment of high blood pressure appears to reduce the risk of cognitive decline and dementia. Diligent reduction of high blood pressure and regular monitoring are recommended throughout mid and later life [6]. High cholesterol and type 2 diabetes in mid-life are also risk factors for dementia [12,13]. Regular monitoring of blood cholesterol and blood sugar levels from mid-life and effective treatment of hypercholesterolaemia and diabetes are also recommended. Current smoking is a risk factor [14] and more research is needed to clarify to what extent smoking cessation reduces the risk of dementia.

Some doctors suggest that further research into the limitations of existing studies is needed before any recommendations on interventions to reduce dementia risk can be made [15]. However, others highlight there is good evidence that preventative strategies including physical, mental and social activity together with good nutrition and control of vascular risk factors may reduce the risk or delay the onset of dementia [16]. Encouraging people of all ages, and particularly those in their forties and fifties, to reduce their risk of dementia will enhance physical health as well as brain health and can do no harm. Further information about the risk factors for dementia, including helpful advice about what might be done to reduce risk, is available from Alzheimer’s Australia’s Mind your Mind<sup>®</sup> program:

1. Alcohol intake associated with a lower risk of dementia appears to range from 1 up to 4 standard drinks per day [9,10].
2. Information and resources on dementia risk reduction are available from the Alzheimer’s Australia website at www.alzheimers.org.au.
Treatment of dementia

There is currently no cure for dementia. However, better understanding of the changes that occur at the molecular and cellular levels has led to the development of drug treatments that can slow the worsening of the symptoms of Alzheimer’s disease [3,17,18]. In Australia, four drugs are approved for the treatment of Alzheimer’s disease. Donepezil, rivastigmine and galantamine are cholinesterase inhibitors that make more of the neurotransmitter acetylcholine available at brain synapses, and help to enhance memory function [19]. Memantine acts on the neurotransmitter glutamate and can relieve symptoms in middle and later stages of Alzheimer’s disease [20].

Medical comorbidities such as diabetes and hypertension must also be managed. Preventative primary care through vaccination, personal hygiene, restful sleep, hydration, nutrition, and dental care is important [16].

Psychosocial treatments and support are also beneficial. People with dementia need emotional and practical support. Their safety must be ensured, for example, by providing support for those who live alone, supervising their medication doses, and preventing wandering or driving if necessary. Care to assist them with functional losses in daily activities will eventually be required. Physical, mental and social activity and stimulation should be encouraged and maintained to avoid de-conditioning [16]. Dementia carers also require support, especially education about dementia and its progression. They need professional support in providing activities, overseeing medication, managing crises and handling problem behaviours, all of which require availability and input from dementia care professionals [16].

Diagnosing dementia

Obtaining a diagnosis of dementia can be a difficult, lengthy and intensive process. While circumstances differ from person to person, everyone has the right to:

- A thorough and prompt assessment by medical professionals
- Sensitive communication of a diagnosis with appropriate explanation of symptoms and prognosis
- Sufficient information to make choices about the future
- Maximal involvement in the decision-making process
- Ongoing maintenance and management
- Access to support and services.
What are the benefits of a timely diagnosis?

- Checking concerns about cognition – changes in memory and thinking ability can be very worrying. Symptoms similar to dementia can be caused by several different diseases and conditions, some of which are treatable and reversible, including infections, depression, medication side-effects or nutritional deficiencies. The sooner the cause of these symptoms is identified, the sooner treatment can begin. A medical review of any symptoms and identification of the cause of symptoms can bring relief.
- Planning and assistance – timely diagnosis enables persons with dementia and their families to receive help in understanding and adjusting to the diagnosis of dementia, and allows them to prepare for the future. This might include making legal and financial arrangements, changing living arrangements, and finding out about aids and services that will enhance quality of life for the person with dementia and their family and friends. Timely diagnosis can give the person an active role in decision-making and planning, while family members can educate themselves about the disease and learn effective ways of interacting with the person with dementia.
- Treatment – timely diagnosis allows for prompt access to medications and medical attention. There is evidence that the currently available medications for Alzheimer’s disease may be more beneficial if given early in the disease process. In some people, these medications can help to maintain daily function and quality of life as well as stabilise cognitive decline. However, they do not help everyone and they are not a cure.
- Health management – general practitioners need to remember the possibility of dementia in people with multisystem disease. Early stage dementia can be overlooked when the patient seeks consultation for other conditions. A diagnosis can also help in the management of other symptoms that may accompany the early stages of dementia, such as depression or irritability. Factors that might exacerbate cognitive problems can be checked for and treated. For example, vascular risk factors, poor nutrition, lack of stimulation and activity and some medications can contribute to cognitive impairment.
- Medication review – a person with dementia needs to have a medication review. Some medications, such as anticholinergics, can exacerbate dementia symptoms. Memory problems may interfere with a person remembering to take important medications such as those for diabetes, heart disease or high blood pressure. A Webster pack can help to simplify administration of medication.

Current practice in diagnosing dementia

In specialist practice, the diagnosis can be made at a syndromal level – that is, there is dementia – and at a disease, or aetiological level e.g. there is Alzheimer’s disease or Lewy body dementia. The accuracy of the diagnosis of dementia syndrome is very high, with doubt only occurring in a small percentage of cases usually at the borderline of normal, or MCI. The diagnosis of aetiology, even in specialist clinics, is about 90% accurate against post-mortem confirmation – though this is improving with new biomarkers [21]. However, in primary care diagnosis is more challenging. Findings from a variety of sources and tests must be pooled before a diagnosis can be made, and the process can be complex and time consuming. Even then, early in the course of the disease, uncertainty may still remain, and the diagnosis is often conveyed as “possible” or “probable”.

Practitioners involved in diagnosing dementia may include:
- The General Practitioner (GP) – is usually the first contact when concerns about thinking or memory arise. The GP takes a medical history, may carry out a brief test of memory and concentration, and organise further investigation.
- Specialist Geriatrician/ Memory Clinic – the GP may refer to this service in many cases.
- Clinical Nurse Specialist in Dementia – a nurse who undertakes assessments for the detection of dementia.
- The Practice Nurse (PN) – a nurse who works in a General Practice and whose role includes identification and assistance in the assessment processes (such as aged care health assessments), support for patients and carers, and networking with community services.
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If the person is still concerned about his or her thinking or memory after a GP consultation, a referral to a specialist for evaluation is appropriate. It is important to remember that the choice is up to the patient, who can ask the GP for a referral.

Specialists involved in diagnosing dementia may include:
- Geriatrician – a medical practitioner who specialises in the care of older people.
- Old Age Psychiatrist (Psychogeriatrician) – a psychiatrist who deals with mental health issues in people aged from 65 years, and people younger than 65 years with age-related mental health problems, such as younger onset dementia.
- Neurologist – a medical practitioner who specialises in the diagnosis and treatment of disorders of the nervous system, including the brain, spinal cord and nerves.
- Clinical neuropsychologist – a psychologist who specialises in the assessment, diagnosis and treatment of disorders associated with conditions affecting the brain.
- Psychiatrist – a medical practitioner who specialises in the diagnosis and treatment of mental disorders, emotional disturbances and thought disorders.

Such specialists have a detailed knowledge of the memory and behaviour changes associated with dementia. They may perform or arrange for in-depth assessments, brain scans and blood tests.

In Australia, a specialist medical practitioner must confirm the diagnosis of Alzheimer’s disease for a patient to be eligible for subsidised Alzheimer’s medications.

Other assessment options may include:
- Aged Care Assessment Teams (ACATs) - multidisciplinary teams often comprised of social workers, occupational therapists and physiotherapists, nurses and doctors. ACATs are usually based in hospitals or regional community health centres. ACATs assess the health needs of ageing individuals, put the individual in contact with relevant services, make recommendations about the level of care required, and approve eligibility for certain services.
- Memory clinics - incorporate a range of specialists in the diagnosis of dementia.
While they provide diagnostic services for all types of dementia, they may also offer specialist services for younger onset or rare forms of dementia. Memory clinics are known as Cognitive Dementia and Memory Services (CDAMS) in Victoria.

Web-based cognitive tests are becoming available. There are ethical and professional issues relating to the use of these tests. For example, are the instructions understood? How should scores be interpreted? Have the tests been validated? Useful information about dementia symptoms may be available on quality websites, but diagnosis remains a process that must be undertaken by a qualified health professional.

The process of diagnosis
The first step is to assess symptoms with a thorough medical history, physical examination, and evaluation of memory and thinking abilities. Other causes of dementia-like symptoms must be ruled out through laboratory tests and in some cases, brain scans. Conditions that can produce dementia-like symptoms include:

- Neurological problems such as stroke, brain tumour, head injury
- Mental disorders such as depression, delirium
- Abnormal function of liver, kidney, thyroid, hormonal system
- Nutritional deficiencies and anaemia
- Other causes such as poor eyesight or hearing, severe constipation, side effects of medication, diabetes or infections.

If these conditions are ruled out and symptoms meet the criteria for a dementia, a later step in the process is to determine the cause of the dementia. Common causes include Alzheimer’s disease, vascular dementia, dementia with Lewy bodies or frontotemporal dementia.

Medical History
A complete medical and family history is required along with details of current problems. Questions will be asked about forgetfulness, orientation, problem solving, coping with everyday life, mood, alcohol consumption and medication usage. It needs to be established when the change in function was first noticed, whether the change was sudden or gradual and whether the person’s difficulties are getting worse. Determining the onset and progression of symptoms can help to differentiate types of dementia. Descriptions of the person’s difficulties from family members, obtained if the person consents, are vital in the diagnosis process.

Medical Testing
Medical tests sometimes used in the diagnosis of dementia include blood, urine and genetic tests, and brain scans. Blood or urine tests can exclude other causes of dementia symptoms by testing for infections, vitamin and nutrient levels, as well as kidney, liver and thyroid function. Genetic testing is only performed in rare cases where there is a family history of younger onset dementia, and is currently not a common practice in the diagnosis of dementia. Although researchers have identified some more common genes, such as ApoE 4, that increase the risk of developing Alzheimer’s disease, these genes do not cause the disease. Currently available genetic tests for ApoE 4 do not reliably aid in predicting who will develop Alzheimer’s disease, so are not routinely conducted.

Investigations
Brain scans can be used to detect brain abnormalities such as tumour, stroke or brain haemorrhage, brain shrinkage (atrophy) and increased pressure of fluid in the brain. Routine brain scans, which include computerised tomography (CT) scans and magnetic resonance imaging (MRI), are relatively non-invasive procedures that produce an image of the brain. Brain scans do not always show abnormalities in people diagnosed with dementia, as sometimes there are no visible changes in the brain. Sometimes, brain scans can be used to help determine the type of dementia. A person with vascular dementia might show evidence of strokes or other vascular changes in the brain, whereas a person with Alzheimer’s disease might show evidence of brain shrinkage in certain regions.

Other types of brain scans are used in specialist or research settings. Positron Emission Tomography (PET) is a type of functional brain imaging; Single Photon Emission Computed Tomography (SPECT) is a brain scanning technique that can show changes in blood flow. Functional Magnetic Resonance Imaging (fMRI) provides information about brain function as well as structure and is typically used in research studies. It is likely that these will become more commonly used in diagnosis in the future.

Psychological Evaluation
Mood can influence cognition and the symptoms of depression can often be mistaken for dementia. Consequently, tests of mental wellbeing may be included in the diagnosis process. These may involve interviews or questionnaires to ask about the presence of symptoms of depression or anxiety.

Cognitive Evaluation And Screening Tests
Tests of cognitive functioning are obviously very important in the diagnosis process. These tests are used to determine the extent of any memory or thinking problems and can be used to track progression over time.

Initial dementia screening tests can be quite brief and simple, such as the person giving the date, copying a diagram, learning a short list of words, or naming common objects. Common brief assessments include the Mini-Mental State Examination (MMSE) and the Brief Cognitive Rating Scale. These screening tests may also include gaining information about the person from their carer or support person. Currently, the MMSE is used for determining whether someone is eligible for subsidised prescriptions of medications for treating Alzheimer’s disease. The Dementia Outcomes Measurement Suite (DOMS) provides more information about these and other tests [22].

4 CT scanning involves use of specialised x-rays to generate a 3-D image of brain structure and is useful to rule out other causes of symptoms. MRI uses a strong magnetic field and radio waves instead of x-rays to produce a 3-D image. MRI can be used to rule out other causes, find characteristic patterns of brain damage, and differentiate between types of dementia.

4 For further information see the podcast from Austin Health in which Professor Chris Rowe discusses PET and detection of cognitive decline in our aging population: www.austin.org.au/podcasts
Recently developed screening tests include:

- The General Practitioner Assessment of Cognition (GPCOG) – a quick, valid, and efficient test for dementia screening in primary care that can use informant information if necessary [23]. Brodaty and colleagues also note that the GPCOG scores appeared to be independent of the patient’s Geriatric Depression Scale (GDS) score, a popular test for diagnosis of depression in the elderly.

- The Rowland Universal Dementia Assessment Scale (RUDAS) – a simple screening tool that tests multiple cognitive domains and appears unaffected by gender, years of education and seems culturally fair. It was developed using culturally diverse study populations and advisory groups [24].

- The Kimberley Indigenous Cognitive Assessment - includes several subsections including a cognitive assessment section (KICA-Cog) and a briefer cognitive screen (KICA-Screen) that can be used in conjunction with carer input (KICA-Carer) [25]. It is a valid dementia test for older rural and remote dwelling Indigenous Australians that does not appear to be affected by educational level.

- The Montreal Cognitive Assessment (MoCA) – a brief cognitive screening test which also has a high sensitivity and specificity for detecting Mild Cognitive Impairment (MCI) [26].

- The MiniCOG – composed of a three item recall and clock drawing. It has been established as an effective routine screening test for use in primary care practice [27].

Brief screening tests can be followed up by more detailed neuropsychological tests (for example, the Cambridge Cognitive Examination – CAMCOG), which explore different areas of function such as memory, language, reasoning, calculation and ability to concentrate [28]. Some people perform well on brief screening tests but memory and thinking impairments may be found with more comprehensive testing. In other cases, people who have performed more poorly on brief tests may be found not to suffer from dementia on more detailed screening. The more detailed tests are able to distinguish between different patterns of decline and are therefore important in helping to identify the type of dementia affecting the person. There are also tests used in drug trials, such as the Alzheimer’s Disease Assessment Scale Cognitive test (ADAS-Cog), which are not used for screening.

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Experiencing a diagnosis of dementia

A diagnosis of dementia is an emotional time for both the person with the illness and for those caring for them. Bamford and colleagues [29] found that following disclosure, studies reported feelings of shock, anger and fear and negative effects on self-esteem and confidence, whilst non-disclosure (or a vague or possible diagnosis) was confusing, upsetting and difficult to understand. Positive impacts from disclosure, such as an end to uncertainty and confirmation of suspicions, were also reported.

1 For more information about different types of dementia, please see the Alzheimer’s Australia web page on Types of Dementia at www.alzheimers.org.au
Recent research conducted by Pond et al. [30] captured benefits and disadvantages of diagnosis for both the person with the dementia diagnosis and their carer or support person. An excerpt from this project revealing carers’ experiences is shown below:

An important benefit is early access to medication which may delay the progression of the disease:

“But I know there is medication that can be put into place if you catch it early” (10LR).

Early awareness of a person’s diagnosis was important for carers. This knowledge allowed them to prepare for the future management of caring for the person with significant memory problems:

“I would much rather know that there are medical issues or possible you know Alzheimer’s issues or dementia or something along that line so that we can get up to speed get things in place and be pro-active in dealing with it... for a whole range of reasons more to do with myself than anything else” (28TB).

The participant above indicated that timely and relevant information on issues such as support networks and dementia services was also important. However, like this participant, other carers reported difficulty accessing information. It was suggested that a central information point to direct people to the appropriate service was essential:

“I’m just sort of finding that there’s no kind of system that I could go to, an information session or get a briefing as to now that we’ve transitioned to this level and you’re sort of the primary support person, these are the sort of things that you might need to consider and these are the services that are available and here’s how you qualify for them. Like an information package” (28TB).

Not everyone appreciated the benefits of receiving a diagnosis. A carer perceived that she had to shield her husband from the negative connotations of dementia. Consequently, using the term “memory loss” was preferable to “Alzheimer’s”:

“He’s still got his wits about him in a lot of ways. And I just didn’t want to present him with the word Alzheimer’s. Because it has connotations that you know, ah are not particularly nice so I thought well we’ll just leave it that he’s got a memory loss problem and, and leave it at that” (27PM).

Findings from this project also provided insight into how carers perceived the experience of caring for the person with dementia [30]:

Carers reported providing more or less care, depending on the progression of the dementia. At a minimum, and where the carer believed the dementia to be mild or even non-existent, a carer might provide “emotional support” or care that was “very basic at this stage.” In this instance, a stepdaughter provided: daily phone contact, transport, financial/money management, medication management and some assistance with household cleaning.

Carers also emphasised the need for vigilance, particularly in relation to monitoring/surveillance of a person with dementia:

“Well, I won’t let him go anywhere on his own. We always go out together, come home together” (3.MT.W).

“I just feel I’ve got to watch him” (8.JL.W).

“I’ve got to keep any eye on things when she’s cooking the lunch; when she puts things on the griller; she’s got a habit of leaving the griller turned on and walking away. She turns the gas off at the main, but then next time she comes to turn the stove on the gas is still on and she can’t smell the gas and so, one of these days she’ll probably blow herself up” (82.W).

Despite a person experiencing early stage dementia, there were carers who were adversely affected by the person’s memory impairment. For example, fearful for what his wife may or may not do if he left her alone for a long period of time, the carer below restricted his leisure time:

“Well I’ve got to be around a bit. I do leave her and I, I’m confident. Like I go and have a game of golf occasionally and play a game of bowls and I can go down the street, go and see a mate. … she doesn’t mean to be molly coddled if that’s what you mean and I don’t need to be there twenty four hour service, but I wouldn’t leave her on her own for any length of time. I think she’d forget to take her medicine I’d reckon” (9.JF).

If constant visual contact was not possible, some carers developed strategies to address the problem. A daughter described the systems she had put into place to monitor her mother:

“Well actually, she lives in a little rumpus room about two metres away, across like a patio and then it’s the house, so she’s got a room like that. And we have a baby monitor, because we used to have a door bell and she used to forget to ring the door bell for help and so now we’ve got a baby monitor and we can hear every movement almost (chuckle)” (40.HS).

Local and overseas research suggests that there are significant delays from symptom onset to diagnosis of dementia. For instance, in Europe the average time to diagnosis after the caregiver first noticed symptoms was 20 months, with caregivers waiting, on average, 47 weeks before bringing the affected person to the attention of a physician [31]. In Australia, families first notice symptoms of dementia an average of 1.9 years before the first health professional consultation and there was an average of 3.1 years before a firm diagnosis was made, which was consistent with other overseas studies [32]. The consequence of this delay is a lost opportunity for earlier medical and social interventions for those suffering dementia and their families [32].

**Barriers to diagnosis**

Speechly et al. [32] identified two areas of delay in diagnosis: prior to, and following, the first consultation with a health professional. Some carers initially arranged support instead of seeking medical advice. The stigma associated with dementia and the misinterpretation of symptoms also contributed to the delay. Some carers perceived that their concerns raised in the initial consultation with a health professional were dismissed, symptoms not acted upon, or referral delayed. Recent research has explored this latter aspect [33].

Paterson and Pond [33] conducted an extensive literature review to identify the most frequently cited barriers to diagnosis and disclosure of dementia. These included:

- **GP limitations - difficulties in differentiating normal ageing from dementia; a perceived lack of need to determine a specific diagnosis; GP lack of confidence or training; and risk of misdiagnosis.**
Practical limitations – a paucity of specialist diagnostic services, especially in rural areas; lack of a recognised, quick-to-administer screening tool; limited consultation time; and the patient’s impaired ability, which hinders an accurate history and participation in self-care.

Negative attitude to dementia – the stigma associated with dementia; doubts about the efficacy of medications; a perception of the patient as unable to comprehend or cope with the diagnosis, and the risk of detriment to the doctor-patient relationship.

Their research also suggested that involving the practice and community nurses in the diagnostic process may overcome some of the barriers and improve detection rates.

The GPs’ perceptions of barriers to timely diagnosis of dementia have also been explored [34]. It seems that in the consultation context, patients and GPs often have competing health priorities, and that GPs are reactive and rely on patients to alert them to their issues [34]. This can be problematic for dementia sufferers who may lack insight into the problems they are experiencing. Often the carer or family member of the patient is the person who alerts the GP to possible dementia. Consequently, should someone live alone, the absence of this information from a carer or family member can hinder the diagnostic process. Also, diagnosis is particularly difficult in carers who develop cognitive impairment themselves, as consultations often focus on their caring responsibilities.

For some people, other barriers to diagnosis, especially to a timely diagnosis, include the belief that memory problems are a normal part of ageing, the perceived stigma attached to dementia, the lack of a cure, and fear about the future. Timely diagnosis and awareness about dementia are the first steps in designing management strategies. As more effective treatments become available in the future, timely diagnosis will become even more important.

Pond et al. continue to conduct research into barriers to the process of disclosure regarding dementia diagnosis. An excerpt from research in progress appears below:

“You’re not really seeing what’s going on.” (GP)
The GP consultation occurs in a time limited context which is not conducive for identifying dementia – “it’s really difficult to do on someone you’ve never met before in a 15 minute interview … if they’re in the early stages of it, you can’t possibly know” (GP#9) – “unless they behave erratically in the room?” (GP#17). Furthermore, other health concerns may cloud the issue: “if there is something like anxiety or depression coexisting…[it] makes it difficult for us…you do only see them in that…brief little time they come in with their…scripts” (GP#7).

“The patients mask it particularly well.”
People with early dementia may perceive their memory decline as part of the normal ageing process and not as a health issue. They may hold negative connotations of becoming old and forgetful and try to present themselves in the best possible way; they want to be seen to be coping. Nonetheless, “the hardest thing is if … the person’s got obvious memory loss and they either, or their family, have chosen to significantly ignore it …Then trying to bring it up…it makes it harder” (GP#8).

“Giving bad news… no one likes [to do it]”
Most GPs considered that patients were often “fearful of the diagnosis” (GP#17), “they don’t want to be told that. No. Memory problem, no. Alzheimer’s, please don’t tell me that” (GP#18). However, disclosure was largely deemed essential – “Patients rights come to the fore…to know … as with any condition” (GP#15). In the main, GPs favoured conveying the diagnosis to the patient with the patient’s family/carer(s) present.

“I find it better to do so with other family members there.”
When patients were accompanied by family/carer(s), often the family/carer(s) would be the focus for information so that they could understand the disease. In turn, this knowledge would help them support the patient. A diagnosis of dementia has “implications for the patient and the family” (GP#15) “and most… carers actually really…want to know what they can do about it” (GP#6). It was important for the carer to be informed and understand about dementia to help them cope with the consequences of the disease: “The best piece of advice… is to actually explain why they’re doing that and they’re not actually lying, that they don’t have that piece of information, so they’re filling it in with something else. Once the carer understands why they’re saying what they’re saying and doing what they’re doing…. that seems to help the carers more than anything” (GP#11). However, informing about dementia was further complicated by “the unpredictability of the decline” (GP#21). “Everyone can fluctuate. One day they’re quite good, another day they’re terrible” (GP#5).

Multiple factors interplayed when disclosing the diagnosis of dementia. Communicating the diagnosis sensitively suggested the underlying stigma attached to the ‘dementia’ label. The GPs appeared sensitive to the negative connotations the word ‘dementia’ implied and tended to couch their disclosure of the diagnosis in other phrases: “I don’t mind disclosing the diagnosis, because I don’t disclose it as dementia, I disclose it more as memory impairment” (GP#10); “I don’t think you necessarily need to use the words dementia or Alzheimer’s disease…. words like ‘memory loss’ or ‘memory not working as well as it used to’ are euphemisms that are quite useful” (GP#13), “Memory impairment or cognitive decline seems to be a bit safer…. There is some stigma, there’s also…. a lot of fear associated with dementia” (GP#21). Overall, disclosure was “about maybe confirming people’s fears, then trying to give them a constructive way to move on” (GP#19).
Solutions to the barriers

Professor Henry Brodaty [35, p.1-2] identified six reasons why early diagnosis does not occur and suggested possible solutions\(^g\). These are summarised in the table below:

<table>
<thead>
<tr>
<th>OBSTACLES TO A DIAGNOSIS OF DEMENTIA</th>
<th>POSSIBLE SOLUTIONS:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Personal unawareness.</td>
<td>Increase awareness about dementia and the significance of memory impairment.</td>
</tr>
<tr>
<td>Personal reluctance to seek help.</td>
<td>Raise public awareness. Reduce stigma in the community and dispel the myths surrounding dementia.</td>
</tr>
<tr>
<td>A belief that memory loss is an inevitable part of ageing – and training at least half of the people presenting to their GP with memory loss do not receive a diagnosis of dementia – it’s just ‘old age’.</td>
<td>More education for GPs.</td>
</tr>
<tr>
<td>Non referral for specialist opinion. Reasons may include: lack of awareness by GP, unavailability of specialist services, financial impediments.</td>
<td>May include: more education for GPs, improved relationships between GPs and specialists, better access to specialist services (e.g. telepsychiatry for rural or remote services), improved communications/links between health practitioners and organisations involved in the diagnosis and management of dementia.</td>
</tr>
<tr>
<td>Lack of management plans.</td>
<td>Education for health practitioners, for patients and families about the availability of management possibilities.</td>
</tr>
<tr>
<td>Medication not prescribed – GPs’ lack of awareness of antidementia medication, or a lack of conviction of their efficacy.</td>
<td>Education about the use of medication, advocacy for reimbursement for antidementia medication.</td>
</tr>
</tbody>
</table>

\(^g\)For a good review of the benefits of timely diagnosis, please see “Six reasons why timely diagnosis of dementia does not occur and ten reasons why it should” by Professor Henry Brodaty [35].

After the diagnosis

Timely diagnosis of dementia is the first step in understanding and managing the condition. Communicating a diagnosis of dementia can allow for planning to begin. Although many people with early stage dementia will initially feel “shattered” by the diagnosis, many also say they feel a sense of relief that the cause of their difficulties is identified, and knowing the diagnosis can increase their sense of independence and enable an active role in planning for their future. It can be difficult to take in information at the time of diagnosis, so scheduling another GP consultation time to talk about the diagnosis, possible benefits of medication and side effects, and referral to support services is important. Should the person with dementia have a support person, it will generally be helpful for them to be involved and attend these consultations. The carer can be an important source of information about changes in behaviour and other concerns, and can help the person with dementia to gather information.

It is important to encourage persons with dementia and their families and carers to consider using the information and support services offered by organisations such as Alzheimer’s Australia. Both parties will need ongoing sources of support as the condition progresses and behaviours change. The GP may be well placed to monitor the mental and physical health of the person and their family carers as the care burden increases. Similarly, the GP may encourage planning around ceasing driving and use of alternative transport as functional deficits and driving risk increases.

If the GP is concerned about any tension between privacy considerations and the care relationship, the Office of the Privacy Commissioner is available to provide advice.

Life doesn’t stop with a diagnosis. Quality of life can be maintained, as there are many available sources of support, which can help the person with dementia as well as their family and friends. Alzheimer’s Australia provides assistance and support.
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Visit the Alzheimer’s Australia website at www.alzheimers.org.au for comprehensive information about
• dementia and care
• information, education and training
• other services offered by member organisations

Or for information and advice contact the National Dementia Helpline on 1800 100 500
Alzheimer’s Australia Publications

Quality Dementia Care Series
1. Practice in Residential Aged Care Facilities, for all Staff
2. Practice for Managers in Residential Aged Care Facilities
3. Nurturing the Heart: creativity, art therapy and dementia
4. Understanding Younger Onset Dementia
5. Younger Onset Dementia, a practical guide
6. Understanding Dementia Care and Sexuality in Residential Facilities

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In Our Own Words, Younger Onset Dementia, February 2009
National Consumer Summit Younger Onset Dementia Communique, February 2009
Dementia: Facing the Epidemic. A vision for a world class dementia care system, September 2009

These documents and others available on www.alzheimers.org.au
Care of Patients with Dementia in General Practice

Guidelines

Royal Australian College of General Practitioners

NSW Health
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Funded by
the NSW Department of Health
under the NSW Action Plan on Dementia 1996-2001

Endorsed by
the Royal Australian College of General Practitioners
The Mind’s Decay

Some of God’s people have to go that way,
Who once were loving, witty, joyful, sage,
Showing the Spirit’s gifts in rich array,
Now aged babes;
Like ships adrift from reason’s anchorage,
No hand upon the rudder of the will,
Abandoned derelicts, yet floating still.

ELIHU
Foreword

The project to develop these guidelines was funded by the NSW Department of Health under the *NSW Action Plan on Dementia 1996-2001*. The purpose of the project was to provide general practitioners with a resource for the care of people with dementia that encourages early intervention, ongoing management throughout the course of the disease and partnership with carers and other service providers. The project was funded following advice from the General Practitioner Working Group on Dementia and aimed to address some of the issues raised in the Mid-Plan Report on the *National Action Plan for Dementia Care 1992-1996*.

The project aimed to bring together the Royal Australian College of General Practitioners (RACGP), representing practising doctors, and two university departments of general practice to address the important and so far somewhat neglected issue of quality care of patients with dementia in the community, including support for their families and carers. It did this by establishing these guidelines, and then testing them in general practice.

The objectives were:

1. To develop guidelines for the diagnosis and ongoing management of people with dementia in general practice in partnership with carers, families and relevant services.
2. To field test these guidelines in general practice.

An advisory committee of GPs and other relevant stakeholders such as geriatricians, psychogeriatricians, nurses, and carer consumer representatives was established to oversee the project, and met three times during the course of the project. A half-time project officer was employed to work under the guidance of the grant holders and the advisory committee.

The team conducted a review of the literature and current guidelines and liaised with practitioners in related disciplines. Draft guidelines specifically related to the usual working procedures of general practice were developed with input from the advisory committee and three focus groups of general practitioners. These were then field tested by 17 general practitioners who used them in their practices to audit their current management of 119 patients against the guidelines. They then provided feedback about the usefulness of the guidelines with these patients, and their comments were used in finalising the guidelines.

A further survey by the NSW Department of Health of eight GPs unconnected with the trial indicated satisfaction with the format of the guidelines.

Subsequently the guidelines have been updated from time to time when new information has come to hand.

The guidelines have been endorsed by the Royal Australian College of General Practitioners.
Advisory committee

An advisory committee was established to oversee and provide input to the project. As well as meeting formally three times during the course of the project members were able to provide input on an ad hoc basis throughout the project period.

Members

**A/Prof John Snowdon**
Psychogeriatrician
Central Sydney Area Health Service
Chair, Royal Australia and New Zealand College of Psychiatry, Faculty of Psychiatry of Old Age

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Alzheimer’s Association of NSW
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**Jill Elias**
Carer (Consumer)

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**Marilyn Goff**
Nurse Educator – Gerontology
NSW College of Nursing, Sydney

**Richard Fleming**
Dementia Services Development Centre, Sydney

General Practitioners

**Metropolitan:**

**Dr Bob Elliot**
Dr Robert Yeoh (also President, Alzheimer’s Association of NSW)

**Dr Ven Tan**

**Dr Bandu Heart**

**Rural:**

**Dr Carmen Ast** (Tamworth, NSW)

**Dr William Redmayne**
(Quirini and Murrurundi, NSW)
Using these guidelines

These guidelines are directed to the care of patients with dementia who are living at home or with family, rather than to those in facilities such as hostels or nursing homes, though they may apply to some of these.

The guidelines are arranged in 3 parts with increasingly detailed content in each part:

- Summary guidelines (Part 1)
- Full practical guidelines in a format suitable for general practice (Part 2)
- Background and supporting evidence (Part 3).

For convenient access, each part of these guidelines follows the same format, consisting of the following 3 sections:

- Patient presentation
- Assessment
- Management.

This means that the general practitioner can simply refer to the summary guidelines (Part 1) for brief information regarding assessment, for instance. If required, more detailed information can be found in the assessment section of the full guidelines (Part 2), and background and supporting evidence for this information can be found in the assessment section of Part 3.

It is recognised that in practice these aspects of care are not undertaken separately but rather, are part of an iterative process often taking place over a long period of time.

These are at present consensus guidelines, since the process of gathering formal evidence to support them is still to be done. They rely heavily upon previous guidelines issued in a number of countries, only one of which claims to be evidence based (see references).

The guidelines were updated in December 2002.
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Patient presentation

The general practitioner may become aware of the possibility of dementia in three ways:

**Presenting problems**
Patient or family presents with symptoms possibly relating to dementia:
- decline in memory
- decline in thinking, planning and organising
- reduced emotional control and changed social behaviour to the extent of interference with everyday activities.

**Early pointers**
GPs should be aware of case-finding by seeking early pointers to dementia when treating other conditions in older patients, such as:
- forgetting appointments, medication
- decline in grooming, self-care
- social withdrawal.

**Screening**
Should older people without symptoms be screened for dementia?

No, there is no evidence of benefit but practitioners should have a high level of suspicion and assess the patient if there are any possible indications.
When the issue of possible dementia has been raised, assessment is needed to confirm dementia, consider differential diagnosis, determine severity and extent of disability, evaluate any co-morbidity and assess family and social support and environment.

In many cases for patients over the age of 75 or indigenous people over 55 the Enhanced Primary Care health assessment item in the Medical Benefits Schedule can be used.

What to include

**History and functional assessment**
This should include:
- full clinical history
- interviews with patient and family, conducted together and separately
- ability to undertake daily activities (dressing, washing, managing finances, telephone).

**Physical examination**
A complete and thorough examination directed towards known and possible co-morbidity.

**Investigations**
Tests to exclude reversible causes.

**Cognitive assessment**
Use of one of the simple tests of cognitive ability such as MMSE and/or the clock drawing test.

**Home visit**
This is necessary to get the best history and assess the safety and quality of the environment.

What to determine

**Differential diagnosis**
Dementia must be distinguished from other conditions, particularly:
- normal ageing
- delirium
- depression
- drug effects.

Sub-types of dementia should be considered.

**Ability/disability**
Functional status must be assessed in terms of:
- activities of daily living (ADL)
- instrumental activities of daily living (IADL)
- personal safety
- communication ability
- nutrition, hygiene and medications
- driving
- legal capacity for decision making.

**Co-morbidity**
Exclude or manage optimally, conditions which may aggravate dementia, such as cardiac or renal failure, nutritional deficiencies and visual and hearing impairments.

**Family/social support and environment**
Assess carer and family stress and support, and any improvements needed to the home environment.
**Decisions to be made**

**Plan of action**

Assessment and management need at least several consultations over weeks or months, and probably a plan for some years, arranged with patient and family. The Enhanced Primary Care health assessment or care planning items in the Medical Benefits Schedule can be used.

**What, when and how to tell patient and family**

Patient, carer and family need to know what to expect, and the distress of the diagnosis needs to be handled sensitively.

**Referral**

Referral may be necessary if the diagnosis is uncertain or the problems cannot be handled in the general practice.
Management

When dementia has been diagnosed, severity determined, abilities and disabilities clarified and family/social support and environment assessed, management can continue.

**Areas for management**

**Dementia and disability**

Management of the dementia may require:

- behavioural strategies
- environmental change
- drugs, which may delay cognitive decline but do not influence underlying pathology.

**Co-morbidity – acute – chronic**

Regular review to ensure optimal control of co-morbidity should include:

- medication and compliance review
- consideration of extent of depression and anxiety
- nutrition and hydration
- prevention of constipation
- exclusion of silent infection, particularly urinary
- early detection of any physical illness and need for pain relief.

**Health promotion**

- diet – Meals-on-Wheels?
- exercise
- medications – need for domiciliary review? Webster pack?

**Prevention**

- immunisations – pneumococcal vaccine, flu vaccine
- falls prevention.

**Patient / family / social support**

- housing
- legal and financial matters
- driving and other risk activities
- regular checking of carer’s health
- full reassessment of the patient at least annually.

**Aspects of management**

A management plan should be drawn up with the patient and family, taking into consideration the following issues:

- Initial stage
- Long-term plan
- Follow-up.

This should include regular consultations as well as allowing for extra consultations when necessary.

- Referral?
Audit of care for persons with dementia

These questions are suggested as an audit for the general practice management of a person with dementia.

<table>
<thead>
<tr>
<th>Circle yes or no to each item:</th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Does this person have a definite diagnosis?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Have reversible causes of confusion been excluded?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Is co-morbidity managed optimally?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Have the person and their family been:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Consulted throughout the process?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>2. Told about available services?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>3. Told about sources of education?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>4. Given the contact phone number of their State branch of Alzheimer’s Australia?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Have medications been reviewed:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. At the time of diagnosis?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>2. Three-monthly?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>3. Six-monthly?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Has psychiatric co-morbidity been assessed?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Have measures such as driving, enduring power of attorney, enduring guardianship and will been discussed?</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Have arrangements been made for a three-monthly review of support needs of carers?</td>
<td>Y</td>
<td>N</td>
</tr>
</tbody>
</table>

(Adapted from NZ Guidelines, 6:41)
2.1 Patient presentation

The general practitioner may become aware of the possibility of dementia in three ways:

- presenting problems
- noting early pointers when treating other conditions, or screening.

**Presenting problems**

Patient or family presents with problems possible relating to dementia.

The common clinical symptoms contributing to diagnosis include:

- a decline in memory to an extent that it interferes with everyday activities
- a decline in thinking, planning and organising day to day things, to an extent that it interferes with everyday activities
- communication problems eg always repeating, asking same questions, not finishing sentences, always saying strange things
- decline in finding words or other aspects of language
- a decline in emotional control or motivation, or a change in social behaviour, as manifested by symptoms such as emotional lability, irritability, apathy and coarsening of social behaviour (eg in eating, dressing and interacting with others) (see pp29-30 for a typical presentation).

There may also be other presenting symptoms for less common forms of dementia.

**Early pointers**

Case-finding and warning signs

_Mrs A, aged 78, attending regularly for management of hypertension and arthritis was only recognised as having dementia when police and NRMA contacted relatives when she reported having lost her car keys 16 times. She explained that thieves were taking them, and also stealing her Weet-Bix._

It is important to be alert to cognitive impairment in elderly patients. GPs may note early pointers to dementia when treating other conditions (see p31).

Early pointers that general practitioners should look out for or may note when treating other conditions:

- giving up activities/stopping going out (asking for home visit rather than attending surgery if not acutely unwell)
- presenting with mid-life crisis type symptoms, not coping at work, wanting demotion
- recent instability of previously well controlled chronic condition eg hypertension, diabetes
- recent increase in presentations to surgery with vague complaints
- recent presentation with apparently classic complaint which fails to respond to usual therapy eg angina which does not respond to anti-anginal therapy, fracture with pain persisting unusually long after healing apparent on X-ray
- failure to attend for repeat prescriptions on time or turning up too frequently for repeats
- failure to attend a specialist referral (especially if recurrent)
- asking to go into care (or hospital) without apparent physical problems
- recurrent attendances to local hospital Emergency Department for vague or non-acute reasons.
Early diagnosis is important because much can be done for the patient at this stage to improve lifestyle and reduce risks, and for carer and family by providing information and support. However this must be done sensitively to avoid distress, because dementia is frequently seen as a hopeless condition, referred to by one spouse as a ‘funeral that never ends’, or as stigmatising. (See p29 for benefits of early diagnosis).

If a problem is suspected by the doctor, a simple way of opening discussion is to ask the patient ‘how is your memory?’. However, many patients with mild dementing illness will be either unaware or unwilling to admit to cognitive problems. Patients who do complain of memory loss are more likely to have depression than dementia; dementia is more likely to be the cause if others complain about the patient’s memory. However, people who complain of memory problems are at greater risk of later developing dementia. It may be helpful to try to ask about the patient’s memory at regular intervals of time in order to assess the possibility of dementia (Schofield 1997).

Screening?

Screening is different from case-finding as it refers to action to determine the presence of likely or possible disease in a person without problems or symptoms pointing to the possibility of dementia.

Should patients be screened for dementia? The short answer is no! (See p31).
When the issue of possible dementia has been raised, assessment is needed to confirm dementia, consider differential diagnosis, determine severity and extent of disability, evaluate any co-morbidity and assess family and social support and environment.

Full assessment may need several consultations over a period of time.

The most common cause of dementia is Alzheimer’s disease, which accounts for about 40% of the cases seen. Other dementias include vascular dementia (20%), Lewy-body dementia (20%), with the remaining 20% made up of many others including:

- frontal lobe dementia
- parkinson’s disease with dementia
- normal pressure hydrocephalus
- post traumatic
- toxic (particularly alcohol) or anoxic encephalopathy
- prion diseases eg creutzfeldt jakob disease
- down’s syndrome
- AIDS.

Enhanced Primary Care medical benefit items

The Enhanced Primary Care (EPC) Package introduced by the Federal Government in 1999 includes several elements providing benefits for the assessment and management of patients with early dementia:

- health assessments of people aged over 75 (55 for ATSI people)
- care planning for people with chronic conditions and multidisciplinary care needs
- case conferencing for people with chronic conditions and multidisciplinary care needs.

Health assessment includes activities of daily living, mood and cognition, social function, home situation, and carer health needs. Patient consent is essential. If there are difficulties with patient consent, the carer and the patient’s immediate family should be consulted.

Details are available in the Royal Australian College of General Practitioners (RACGP) document Standards and Guidelines for the Enhanced Primary Care Medicare Benefit Schedule Items available on the RACGP website www.racgp.org.au

Particularly useful may be the following appendices:

- Patient Information sheets
- Home Safety Checklist
- Health Assessment Proforma – practice record and patient summary.

For further information, see the Commonwealth Department of Health and Ageing’s website www.health.gov.au/epc

History and functional assessment

Mr B, aged 72, lives with his wife. He attends somewhat irregularly for his hypertension and peripheral vascular disease. He denies any memory loss or difficulties, but his wife says he forgets the names of their grandchildren, leaves lights and gas on, has lost interest in sex and is often cranky. She is now becoming afraid to leave him when she goes to bowls.

A full clinical history should be taken. This should include interviews with the patient and their family or carer conducted together and separately. Patients may be unaware of or refuse to admit they have symptoms; carers may be defensive or simply reluctant to upset the patient, or occasionally wanting to ‘dump’ the problem.

Asking carers to keep a diary of the patient’s behaviour, or giving them checklists to fill in, can help assess the patient’s decline and allow the progression of the condition to be monitored. However, GPs should be aware that the quality of the information about the patient will depend on how much time the carers and/or family members spend in the patient’s household. Useful instruments to assist in this are available (see p.33).
**Functional assessment**

It is important to assess the extent to which the patient’s problems with memory, cognition and communication are interfering with his or her ability to undertake daily activities (see p17). Health Assessment checklists may be useful (Appendices A1 and A2).

**Physical examination**

A complete and thorough clinical examination is necessary. This should be directed towards finding evidence for:

- specific conditions which may cause dementia eg stroke, Parkinson’s disease, cerebrovascular disease, hypothyroidism
- underlying chronic conditions which may aggravate dementia eg hypertension, cardiac failure, renal failure, diabetes, anaemia
- conditions which may cause delirium eg respiratory or renal infection.

It is important to assess specifically the patient’s level of consciousness as, if impaired, this may be an important pointer to delirium which may need to be treated as an emergency.

There is often considerable co-morbidity found in people with dementia, and they may benefit from a methodical examination in search of treatable conditions (see section p14).

**Investigations**

Although encountered rarely, potentially reversible causes of dementia are important to detect. This has led to the development of a list of tests which should be undertaken in any person with dementia, to ensure that reversible causes will not be overlooked.

- Hb, WBC, ESR
- renal function/electrolytes
- liver function
- thyroid function
- blood sugar
- serum calcium and phosphate
- urine – WBC, protein, sugar (culture if delirium)
- serum B12, folate levels
- CT scan without contrast
- CXR (if delirium)
- syphilis serology (if specific indications)
- HIV testing (if specific indications) (see p28).

**Cognitive assessment**

Suitable well-recognised tests of cognitive ability are the mini-mental state examination (MMSE) (Appendix B1) and the clock-drawing test. A shorter alternative to these is the Australian-developed GPCOG (Appendix B2). Limitations in the interpretation of these tests include:

- other issues that may impair performance such as the presence of dysphasia, sight impairment, deafness, poor educational level, cultural factors, an awareness of the fact that the patient is being tested and fear of testing
- factors that may overcome decreased cognition such as better intellect and education (see p35).

**Home visit**

*Mrs C, aged 75, lives alone. She has attended frequently for years with hypertension, chronic airways disease, NIDDM and osteoarthritis, all becoming less well controlled. On making a home visit, the GP finds that her medication is scattered around an untidy and dirty house, and there is little food. Neighbours help out, but say her son manages her affairs and never lets her handle money.*

Such a situation raises many issues and emphasises the importance of a home visit. One or more home visits by a general practitioner and/or other members of the team will be needed before assessment is complete. This will usually result in additional history prompted by the situation, better assessment of functioning, sometimes a better environment for cognitive testing, and appreciation of the safety and quality of the environment.
Differential diagnosis

Mr E, aged 82, had hypertension, COAD and epilepsy for years, all well controlled on medications, and seemed to cope well living alone. He phoned the ambulance at 6am to take him to hospital because of a fever, but no abnormality was found. He later refused to pay the bill because he said they had not found out what was wrong with him, and became more and more reclusive. The GP was concerned when he did in fact develop a recurrent low grade fever.

Several conditions can present with similar symptoms to those of dementia. These include:

- normal cognitive changes associated with ageing
- delirium
- depression
- drug-induced effects
- mild or moderate intellectual disability
- subnormal cognitive functioning because of a severely impoverished social environment and limited education.

Asking the patient ‘are you depressed?’ and ‘how’s your memory?’ at regular intervals of time may help to differentiate between depression and dementia.

Normal ageing

The perception of failing memory among the elderly is common, with about 25% of non-demented, healthy elderly complaining of memory impairment.

Several features of cognition characterise ‘normal’ ageing. There is a generalised decline in the speed of processing, yet accuracy of response is not affected. Verbal abilities remain stable over the lifespan. Most types of memory also remain stable over life, including immediate memory and long term or remote memory. New learning or recent memory is also relatively resistant to ageing, although not to the same degree.

<table>
<thead>
<tr>
<th>Description</th>
<th>Person with dementia</th>
<th>‘Normal’ older adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Forgets</td>
<td>Whole experience</td>
<td>Parts of an experience</td>
</tr>
<tr>
<td>Forgets words or names for things or objects</td>
<td>Progressively worsens</td>
<td>Occasional lapses of memory</td>
</tr>
<tr>
<td>Delays recall of names</td>
<td>Often</td>
<td>Rarely</td>
</tr>
<tr>
<td>Follows written or verbal directions</td>
<td>Gradually unable</td>
<td>Usually able</td>
</tr>
<tr>
<td>Ability to use notes, reminders, cues from the environment</td>
<td>Gradually unable</td>
<td>Usually able</td>
</tr>
<tr>
<td>Follows a story on TV, in a movie or in a book</td>
<td>Gradually loses ability</td>
<td>Usually able</td>
</tr>
<tr>
<td>Calculations</td>
<td>Gradually loses ability</td>
<td>May be slower than before</td>
</tr>
<tr>
<td>Self-care capacity (dressing, bathing, cooking etc)</td>
<td>Gradually unable</td>
<td>Usually able</td>
</tr>
</tbody>
</table>
The four ‘D’s – dementia, delirium, depression and drugs

The differential diagnosis should include the four ‘D’s of geriatric practice – dementia, delirium, depression and drugs. Remember that the patient’s age, level of education, cultural background and co-morbid illnesses may affect their assessment.

A comparison of the clinical features of delirium, dementia and depression

<table>
<thead>
<tr>
<th>Feature</th>
<th>Delirium</th>
<th>Dementia</th>
<th>Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>Acute/sub-acute depends on cause, often twilight</td>
<td>Chronic, generally insidious, depends on cause</td>
<td>Coincides with life changes, often abrupt</td>
</tr>
<tr>
<td>Course</td>
<td>Short, diurnal fluctuations in symptoms; worse at night in the dark and on awakening</td>
<td>Long, no diurnal effects, symptoms progressive yet relatively stable over time</td>
<td>Diurnal effects, typically worse in the morning; situational fluctuations but less than acute confusion</td>
</tr>
<tr>
<td>Progression</td>
<td>Abrupt</td>
<td>Slow but even</td>
<td>Variable, rapid-slow but uneven</td>
</tr>
<tr>
<td>Duration</td>
<td>Hours to less than 1 month, seldom longer</td>
<td>Months to years</td>
<td>At least 2 weeks, but can be several months to years</td>
</tr>
<tr>
<td>Awareness</td>
<td>Reduced</td>
<td>Clear</td>
<td>Clear</td>
</tr>
<tr>
<td>Alertness</td>
<td>Fluctuates; lethargic or hypervigilant</td>
<td>Generally normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Attention</td>
<td>Impaired, fluctuates</td>
<td>Generally normal</td>
<td>Minimal impairment but is distractible</td>
</tr>
<tr>
<td>Orientation</td>
<td>Fluctuates in severity, generally impaired</td>
<td>May be impaired</td>
<td>Selective disorientation</td>
</tr>
<tr>
<td>Memory</td>
<td>Recent and immediate impaired</td>
<td>Recent and remote impaired</td>
<td>Selective or patchy impairment ‘islands’ of intact memory</td>
</tr>
<tr>
<td>Thinking</td>
<td>Disorganised, distorted, fragmented, slow or accelerated incoherent</td>
<td>Difficulty with abstraction, thoughts impoverished, marked poor judgement, words difficult to find</td>
<td>Intact but with themes of hopelessness, helplessness or self-deprecation</td>
</tr>
<tr>
<td>Perception</td>
<td>Distorted; illusions, delusions and hallucinations, difficulty distinguishing between reality and misperceptions</td>
<td>Misperceptions often absent</td>
<td>Intact; delusions and hallucinations absent except in severe cases</td>
</tr>
<tr>
<td>Stability</td>
<td>Variable hour to hour</td>
<td>Fairly stable</td>
<td>Some variability</td>
</tr>
<tr>
<td>Emotions</td>
<td>Irritable, aggressive, fearful</td>
<td>Apathetic, labile, irritable</td>
<td>Flat, unresponsive or sad. May be irritable</td>
</tr>
<tr>
<td>Sleep</td>
<td>Nocturnal confusion</td>
<td>Often disturbed. Nocturnal wandering and confusion</td>
<td>Early morning awakening</td>
</tr>
<tr>
<td>Other features</td>
<td>Other physical disease may not be obvious</td>
<td></td>
<td>Past history of mood disorder</td>
</tr>
</tbody>
</table>

(Adapted from NZ Guideline 6:22 and LoGiudice 1999)
**Delirium**

It is essential that delirium be discounted early in the diagnostic process. The underlying physical disorder, together with decline in cognition, may constitute a medical emergency. Immediate evaluation of the underlying causes and initiation of possible treatment is imperative.

Delirium is a confused state precipitated by an underlying organic cause, although this may not always be obvious. Some clues to the diagnosis include:

- sudden change in mental state or behaviour (informant history is of utmost importance)
- recent change in medication
- evidence of infection
- visual hallucinations (which indicate delirium until proven otherwise)
- very old, physically ill, with known dementia
- recent surgery
- looks unwell, perplexed or anxious
- vision and hearing impairment.

Some of the causes include substance abuse, medication effects, infections, vascular changes, hypoxia, metabolic problems, surgery and trauma. Delirium is not always of short duration and of florid symptomatology; a sub-acute confusional state can last for months.

Patients with delirium may have dementia as well, and this needs to be assessed when the cause of the delirium has been treated.

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**Depression**

It can be difficult to differentiate between dementia and depression. Depression can manifest as dementia; conversely, dementia can present with depressive symptoms early in the illness.

Because patients with dementia may also be depressed and have poor insight or ability to express their mood changes, other clues must be sought. Features such as a past history of depression, recent onset in symptoms, poor appetite and loss of weight, depressed mood, thoughts of self-reproach, guilt or suicide, and delusions favour a diagnosis of depression (see p.36).

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**Drugs**

Many drugs can cause or aggravate cognitive impairment. Problems particularly arise when doses are changed or new drugs are added, and occasionally when drugs are stopped. It is therefore important that a full drug history be obtained.

The following drugs are those which are more commonly involved:

- antidepressants
- lithium
- minor tranquillisers
- neuroleptics
- alcohol and other recreational drugs
- analgesics (dextropropoxyphene, nefopam, opiates)
- anticholinergics
- anticonvulsants
- antidiabetics (if cause hypoglycemia)
- antihistamines
- beta-blockers
- corticosteroids
- ciprofloxacin
- digoxin
- dopamine agonists (eg levodopa, bromocryptine)
- H2-antagonists
- non-steroidal anti-inflammatories
- quinine
- theophylline.
Dementia sub-types
Dementia may be of many types. The more common are discussed below. Differentiation may be important for management in some cases eg recognition of early Parkinsonism with dementia may allow improvement with specific treatment, and it is important to avoid neuroleptic drugs with Lewy-body dementia.

Alzheimer’s disease
In addition to progressive memory impairment (especially recently acquired memories), language impairment is an important sign of Alzheimer’s disease. The ability to repeat phrases is usually preserved but naming (initially of uncommon words) is impaired. Other deficits occur with visual and spatial abilities such that there may be difficulties in recognising familiar faces or objects. Apraxias (or difficulty completing complex motor tasks eg miming how to hold a brush and brush one’s hair) may interfere with abilities to carry out activities of daily living. Impairment in arithmetic (acalculia) may interfere with managing accounts and/or a cheque book.

Non-cognitive symptoms might include decreased emotional expression, increased stubbornness, diminished initiative and greater suspiciousness. Delusions may occur in about 30% of patients.

Vascular dementia
Vascular dementia is the second most common cause of dementia after AD, with which it may coexist, accounting for 15–20% of cases. A rapid onset with focal deficits and significant somatic complaints may suggest vascular dementia, as may emotional lability, impaired judgement, early neuropsychiatric symptoms and gait disorders. There is relative preservation of personality and verbal memory.

There are several types of vascular dementia. Where it is caused by multiple small infarcts progression is normally stepped (whereas Alzheimer’s disease progresses gradually).

However sometimes vascular dementia can have a gradual onset and progression similar to Alzheimer’s disease eg when the cause is ischaemic rather than infarction. A computed tomography (CT) scan without contrast may help confirm or exclude a vascular aetiology (Grey Matters 7:9).

Dementia of the frontal lobe type
Dementia of the frontal lobe type describes the syndrome of disordered executive function (impairment of initiation, goal setting, and planning) and disinhibited behaviour with only mild abnormalities on cognitive testing. These people are prone to angry catastrophic reactions. The apathy may be difficult to distinguish from depression. One cause of this syndrome is Pick’s disease which is associated with focal atrophy of one or both frontal and/or temporal lobes.

Dementia with Parkinsonism
These two syndromes can often co-exist. Rigidity and postural instability develop in approximately 30% of people with Alzheimer’s disease. Similarly people with Parkinson’s disease can develop dementia due to coexistent Alzheimer’s disease, cerebrovascular disease or other causes.

Lewy-body dementia
This dementia is characterised by cognitive impairment which affects both memory and ability to carry out complex tasks and fluctuates within one day. This fluctuation can be confused with delirium. In addition, however, at least one of the following is seen:

- visual or auditory hallucinations
- extra-pyramidal features such as sensitivity to neuroleptics or a Parkinsonian appearance
- repeated unexplained falls
- transient clouding or loss of consciousness.

Alcohol dementia
Characteristically this presents with amnesic deficits. Other cognitive deficits may be seen which often include frontal lobe features.
Creutzfeldt-Jakob disease
This is a rare cause of progressive dementia caused by a proteinaceous agent (prion) which is potentially transmissible. It is usually of short duration (1-2 years) and the early stages may be characterised by irritability or unusual somatic sensations. Motor signs such as myoclonus, Parkinsonism and motor neurone dysfunction may be prominent. Visual impairment may occur. An electroencephalograph (EEG) can be diagnostic.

Hydrocephalus
Normal pressure hydrocephalus is characterised by the triad of gait disorder, urinary incontinence and cognitive decline. As each of these elements is common in elderly people, their occurrence together does not necessarily signify a diagnosis of normal pressure hydrocephalus. The condition is sometimes responsive to shunting, but the likelihood of cognitive improvement is highest when the dementia is of short duration.

Sub-cortical dementia syndrome
In this condition, unlike Alzheimer’s disease, there is relative preservation of language, calculation and tasks requiring coordinated motor function. This syndrome may be seen in conditions such as Parkinson’s disease, Huntington’s disease, progressive supra-nuclear palsy, Wilson’s disease and other disorders affecting predominantly the basal ganglia and/or thalamus. (see p.39).

Ability/disability

Assessment of functional status
Assessment of the patient’s ability to manage personal care, such as bathing, dressing and feeding, and other activities of daily living such as using the telephone, shopping and banking, are essential parts of the evaluation of dementia. If the patient is having trouble undertaking such activities – particularly against a background of memory or cognitive problems – then a dementing illness may be suspected.

The use of recognised simple instruments may make such assessment easier and more reliable:

- Activities of daily living (ADL) (Appendix C4)
- Instrumental activities of daily living (IADL) (Appendix C5).

Assessment of function is also included more briefly in more general instruments:

- Health assessments (Appendix A1 and A2)
- GPCOG (Appendix B2).

If the patient demonstrates impaired functional ability on these tests, further cognitive testing should be conducted if not already done (see Appendices B1-B2), (Grey Matters 7:6).

In addition to established physical or intellectual deficits, it is important to be aware that gender and cultural factors may influence the utility of these lists (for instance, men normally do less around the house than women).

The following issues also need to be considered:

- safety issues in the home and on the road (see below)
- personal hygiene
- financial competency
- self-monitoring of medications
- ability to attend to adequate nutrition
- present and future legal capacity regarding: advance care directives, Enduring Guardianship or Enduring Power of Attorney (see below).
Older road users
Advice from medical practitioners is often heeded by older patients in relation to their ability to drive. Using resources such as the Austroads publication *Assessing fitness to drive* will aid the general practitioner in making an informed decision in relation to this (Appendix F2).

Legal capacity for decision-making
Determination of a patient’s capacity to make decisions may be an important role of the doctor. This may apply in one of three situations:
- consent for medical treatment
- giving an advance care directive
- making a will.

It may also apply to other tasks such as managing financial affairs or arranging living circumstances (see p44).

Co-morbidity
Common conditions which can cause or aggravate dementia need to be thought of and excluded or managed are:
- depression
- drugs
- thyroid disorders (hypo/hyperthyroidism)
- subdural haematoma
- neoplasms
- alcohol
- intracerebral lesions (tumour, normal pressure hydrocephalus)
- vitamin B12 deficiency
- folate deficiency
- metabolic disturbances (hypo/hyperglycaemia, uraemia, hypo/hypercalcaemia)
- water and electrolyte disturbances (dehydration and hyponatraemia)
- infections (urinary tract, respiratory tract)
- renal failure
- hypoxia
- malnutrition.

Not only may these conditions aggravate dementia, but also the onset of dementia may lead to deterioration in such conditions, particularly by reducing compliance with medications.

In order not to avoid overlooking any co-morbidity which should be managed optimally, it may help to use the assessment form checklist developed by the RACGP (see Appendix A1).

Family/social support and environment
Assess carer and family
Mrs D, aged 81, has COAD and early dementia which she admits, although denying the seriousness of her problems. She lives alone, helped by a daughter who lives in the next suburb. When her driving licence is cancelled she expects the daughter to take her out every day, and blames her for all difficulties. She complains to the GP that her daughter often becomes explosively angry.

The stress associated with caring for a person with dementia should never be underestimated. It places an extraordinary burden on those who undertake the caring role. Carers are often elderly, or stressed by other family responsibilities. Higher levels of depression, psychological morbidity and use of psychotropic medications are seen in carers of those with dementia.

Difficulties experienced with caring can be enough to produce sufficient stress to place either the person with dementia or the carer at risk, or jeopardise the success of community care.

Grief is a constant feature of dementia. Initially this sense of loss and bereavement may be shared by both the person with dementia and those who are close to him or her, but later these feelings are experienced by the carer, often in isolation from patient, other family members or community or all three.

Signs of stress need to be looked for, the stress level assessed, and reviewed at least six-monthly; three-monthly would be ideal.
Ask the carer ‘How is this affecting you? What has changed for you?’ Ask about the carer’s mood level. Note any changes in the carer’s health which could be stress related. The Caregiver Burden Scale may be useful. (Appendix D1) (NZ Guideline 6:32), (see p39).

If the carer has a different GP they should be referred to that GP with an offer of cooperation in management.

Assess environment
Is the home environment safe? Consider:
- floor coverings
- cooking facilities
- bathroom
- toxic substance storage
- heating.

Action plan
Once the question of dementia arises assessment cannot be achieved in one consultation and a plan needs to be agreed with patient and family or carers, together and separately. The Enhanced Primary Care health assessment or care planning items in the Medical Benefits Schedule can be used.

At the initial consultation it is necessary to:
- determine the problems
- sort out priorities with patient and family
- manage urgent problems
- deal with the priority problems
- arrange a plan for further assessment and management.

This will be encouraged by a focus on dealing with the patient and family’s perceived problems, with follow-up to see that the desired goals are achieved.

There is often considerable fluctuation in the patient’s condition and functioning over time and in different places, and this needs to be considered.

The action plan needs to be considered in terms of weeks, months and years, since dementia is a chronic progressive condition, though the rate of progression varies in different people.

What, how and when to tell patient and family
Mrs F, aged 82, lived in a retirement village with her husband. She had been very active with no obvious health problems. She presented with concern about her memory and inability to control her aggressive feelings when things went wrong. Her husband and family felt it was merely ‘old age’ and denied any difficulties. After assessment, the GP was sure she had early dementia.

While the patient, carer and family have a right not to be informed of the diagnosis, where possible it is best to inform them so that they will know what to expect and can begin making any necessary arrangements, such as altering the home environment, changing wills and contacting Alzheimer’s Australia.

Listen first
Before imparting information, it is important to find out what the patient and family already know about dementia, to reinforce what is correct, and to correct what is not.

What to tell
This depends on what the patient and family need and want to know, but consider:
- what the diagnosis is, and its prognosis
- how this may affect the person’s personality, behaviour and functioning
- when and how to ask for help
- what services are available and how to access them
- legal and financial matters, eg enduring power of attorney, operation of bank accounts
- emotional support systems available
- support and respite care available
- financial assistance available
- how to deal with challenging behaviours and difficult issues such as giving up driving
- residential care options and how to access and evaluate these
- Enduring Power of Attorney or Guardianship
- making of will.
Be careful not to overload people with too much information at one time.

Encourage all involved to read the excellent resources available from Alzheimer’s Australia.

**How and when to tell**

Listed below are ways to help minimise the distress that breaking the news of dementia may cause:

- Allow adequate time and ensure privacy.
- Let the patient decide how much they want to know.
- Tell the patient and carer separately.
- Be empathetic and encourage expressions of feelings.
- Break the news in stages over several consultations.
- Assess patient’s understanding frequently.
- Be aware that both patients and carers may suffer reactive depression or anxiety after hearing the diagnosis.
- It is perfectly acceptable to refer the patient to a specialist to hear the diagnosis if you feel that passing on the diagnosis will damage your relationship with the patient and/or family.

Patients and families should be encouraged to contact the Alzheimer’s Australia, which can provide information and support. Its help sheets are a useful resource available on request to general practitioners to give to patients and families (Appendix F1).

**Prognosis**

Most dementia is progressive, but it will affect different individuals in different ways. On average, the time from onset of the disease to diagnosis is about 2-3 years, while from onset to death is usually within 10 years, but varies greatly for individuals. At some time during the dementia, behavioural complications will affect 90% of patients. Psychological/psychiatric complications include depression, anxiety, psychosis or hallucinations, while non-psychological behavioural complications include agitation, wandering, screaming and aggression (see pp42-3).

**Referral**

Most patients with early dementia can be managed successfully in general practice, without the need to refer to specialists (although other groups such as solicitors and community services may be required). However, some reasons for referring patients to (where appropriate) a neurologist, geriatrician, psychogeriatrician, memory clinic or an Aged Care Assessment Team (ACAT) are:

- confirmation of diagnosis
- uncertain diagnosis or unusual/complicated presentation
- rapid deterioration
- significant psychiatric co-morbidity (especially depression)
- access to dementia drugs (under current PBS arrangements)
- patient is less than 60 years old
- possible industrial exposure to heavy metals
- patient or family request a referral/second opinion
- access to multi-disciplinary team to assist in assessment or management
- difficult behavioural problems
- respite care or other community support services needed
- patient or family in denial and at unacceptable risk.

In addition to assessment, specialist services will ideally offer ongoing monitoring and management advice for a proportion of referred cases (usually those living alone or otherwise at risk), in liaison with the general practitioner.
2.3 Management

When dementia has been diagnosed, severity determined, abilities and disabilities clarified and family/social support and environment assessed, management can continue.

The management of the patient will be guided by the assessments made. Assessment and management will not necessarily be sequential, but will be undertaken in an iterative way following the priorities determined in formulating an action plan. It is important that patient, family and carers are kept involved as the plan is developed and modified in the light of further assessments and progress in meeting objectives.

Dementia and disability

Treatment of specific causes

Sometimes medical or surgical treatment can be offered for potentially reversible causes of or conditions associated with dementia, eg hypothyroidism, vitamin deficiency, hypercalcaemia, normal pressure hydrocephalus, subdural haematoma and brain tumours. Psychiatric illnesses such as major depression or schizophrenia may sometimes present with a dementia-like clinical appearance but can be improved with appropriate treatment.

Drug treatments for dementia

There are currently no drugs proven to prevent dementia or modify the neuropathology of the disease once established. However clinical studies have shown that acetylcholinesterase inhibitors can improve cognitive function and/or delay or lessen the rate of cognitive and functional decline in patients with mild to moderately severe Alzheimer’s disease. A number of acetylcholinesterase inhibitors are currently available under the Pharmaceutical Benefits Scheme, provided the patient meets the guidelines (see the PBS Handbook for current guidelines and arrangements). Evidence of benefit is now accumulating for Lewy-body dementia, but not for other types of dementia, including vascular dementia.

Cholinesterase inhibitors

Donepezil and rivastigmine constitute symptomatic treatments with varying degrees of efficacy and safety. So far the longest studies have used donepezil. Side effects were generally mild and transient in nature, usually resolving without dose modification, and were related to the nervous and digestive systems.

Other drugs

- Aspirin in vascular dementia is of benefit in preventing vascular events or death in patients with a history of prior transient ischaemic attack or stroke.
- Vasodilators – there is no consistent evidence of clinical benefit from vasodilators in dementia.
- Oestrogen – evidence of benefit is controversial.
- Vitamin E – evidence of benefit is controversial and applies only to very high doses.
- Nonsteroidal anti-inflammatory drugs (NSAIDS) – evidence of benefit is controversial.
- Hydergine may lead to a small improvement of variable sustainability in some patients, but those who will respond cannot be predicted in advance.
- Tacrine has a moderate effect on cognitive function, but this effect does not seem to translate to differences in activities of daily living scores, and it has potentially serious side effects so should not be used.

Psychotropic drugs

- Antidepressants – when doubt remains as to the extent of depression in a patient with early dementia, a trial of antidepressant therapy is warranted, with careful monitoring to determine the extent of benefit or adverse effect.
- Other psychotropic drugs – medication can be very helpful in treating some behavioural problems, but should not be regarded as first-line treatment (except in emergencies). Other strategies should be tried first and continued in parallel with drug treatment.
The golden rule is to start with low doses and increase slowly, whilst carefully monitoring both beneficial and adverse effects.

Adverse effects are unfortunately very common. These include: sedation, confusion, decreased mobility, low blood pressure and Parkinsonism, and paradoxical worsening of behaviour. Psychotropic drugs should not be prescribed indefinitely and their use needs regular review.

Major tranquillisers are the usual first-line drug treatment for agitation or aggression (especially if associated with psychosis) and have shown modest efficacy in controlled trials.

If anxiety appears to be driving the behaviour problem, shorter-acting minor tranquillisers may be tried, eg chlormethiazole, oxazepam or alprazolam.

For sleep disturbance a course of a shorter-acting sleeping tablet, eg temazepam or zopiclone can be useful. (see p42).

Managing behavioural concomitants of dementia

Some general practical strategies which carers can adopt:

- Establish a simple, regular routine that suits the person with dementia.
- Establish a physical environment that suits the person with dementia (safe, comfortable, familiar, interesting).
- Be prepared for change, understand that dementia is due to a disorder/disease of the brain and that the affected person has reduced ability to control/think/act.
- Ignore unwanted behaviour or walk away; positive reinforcement of adaptive behaviour.
- Expect inconsistencies – patient can sometimes do things, sometimes not (like faulty wiring).
- Distract – try to focus attention away from what is upsetting the person with dementia.
- Use empathy and humour to defuse tension.
- Maintain respect, avoid infantilisation, don’t say to the person ‘I just told you that’.
- Slow pace, avoid rush.

- Give repeated explanation and reassurance.
- Use clear, direct, short and simple communication; importance of eye contact, gestures and appropriate touch.
- Break tasks down into small steps.
- Look at activities in terms of the steps required to perform them. The person may be able to do some but not all of these eg get dressed, if clothes are selected and put out by someone else.
- If resistance encountered with task, try again later.
- Tolerate the behaviour (avoid arguing or scolding).
- Ensure consistency and avoid change wherever possible.

An important principle in minimising the difficulties that dementia will cause is to change the environment, not the person:

- Install a whiteboard near the telephone to write messages on.
- Display clocks prominently.
- Use calendars where the current date is obvious.
- Remove loose rugs and low furniture which may cause falls.
- Provide the patient with frequent reminders, explanations and orientation cues (see p43).

Co-morbidity

The patient’s general medical problems and treatments should be managed optimally and reviewed regularly to minimise adverse effects on mental functioning, particularly medications which may produce central nervous system side-effects. Polypharmacy should be avoided in light of the potential for additive drug toxicity or complex interactions.

Supervision of medication-taking, especially in those living alone, may be vital. This may require the use of aids such as the Webster pack, and domiciliary medication reviews from time to time.
**Depression**

Social stimulation, appropriate activities, plus counselling when appropriate are first-line strategies for depressed mood. Antidepressant drugs are often worth trying, newer antidepressants such as selective serotonin re-uptake inhibitors (SSRIs) usually being preferable to tricyclics.

**Anxiety states**

High anxiety levels may respond to social or environmental manipulation. If not, patients may benefit from behaviour modification, counselling, or anti-anxiety, anti-panic or anti-phobic drug treatment.

**Cerebrovascular disease**

The medical management of vascular dementia is the same as for stroke disease.

**Other conditions**

Other medical conditions particularly needing optimum treatment are: dehydration, diabetes (particularly, avoidance of hypoglycemia), hypoxia, anaemia, postural hypotension, epilepsy, infective illness, pain and urinary or faecal retention.

**Health promotion**

It is important to focus on the remaining strengths, skills and resources of people with dementia, and work toward the maintenance of these, encouraging customary activities. Support groups such as those run by Alzheimer’s Australia under its Living With Memory Loss Program may improve insight and coping skills and assist patients and carers in coming to terms with disability. Regular review and care planning, with referral on to counselling support groups or other support agencies, is vital.

**Diet**

Adequate diet is very important, particularly to avoid obesity or unwanted loss of weight, and to ensure an adequate dietary intake of vitamins and other essentials. Meals-on-Wheels should be considered for those living alone.

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**Exercise**

‘Use it or lose it’ applies to physical as well as mental activity. Patients need to be encouraged to maintain physical activity appropriate to their interests and physical state, and this needs to be built into their routine.

**Drug Use**

The patient’s consumption of alcohol and other potential drugs of abuse (especially minor tranquillisers) should be reviewed since usual doses (previously tolerated) may produce more obvious toxic effects once dementia ensues.

**Prevention**

This most commonly needs to be directed towards preventing:

- worsening of co-morbid conditions (see p22)
- falls and other accidents
- preventable infections.

**Falls**

Prevention of falls requires recognition and alteration of environmental risks, modification of risk behaviours, and appropriate physical assistance.

**Immunisations**

Routine immunisations such as tetanus should be checked, and updated if necessary, for all patients. Annual influenza immunisation should be given, and many may need pneumococcal vaccine every five years.
Patient/family and social support

This is probably the area of management which will make most impact on the quality of life for both patient and family or carer.

Providing information

Once the presence of dementia is established, information and support become crucial to the management of the condition for the medical practitioner, the person with dementia, and the family. Carers need to be able to access information in small, manageable ‘bites’, checking their understanding frequently and reviewing and updating information at each consultation.

People with dementia living alone will usually need support. There are complex ethical issues involved in ensuring that a person’s wish to continue living alone is balanced with their safety and that of others.

Decision making capacity

The patient’s capacity to make decisions about matters such as consent to treatment, living circumstances and financial arrangements needs to be determined (see pp44-5).

Risk management assessment

An early priority is to assess whether there is any evidence of danger to the person with dementia or to others. Falls, accident risks in the home (eg stove, appliances, open fires), impaired driving, malnutrition, suicide threats or apparent abuse or neglect may require urgent action.

Dementia and driving

The issue of fitness to drive must be assessed. Even mild dementia increases the risk of traffic accidents; the risk increases with concomitant morbidities and as the disease advances.

Writing ‘DO NOT DRIVE’ on a prescription pad may help. If there is a dispute, the patient should be referred to the local office of the Roads and Traffic Authority. Options for alternatives should be discussed including the offer of a second opinion or the suggestion of a formal driving assessment or a simulated test (Appendix F2).

Financial support

The patient may be eligible for superannuation on medical grounds, sickness benefits or a disability support pension.

The carer may be eligible for a carer payment or carer allowance. The latter is not means-tested but based on the severity of the dementia being at a level where the patient would be approved for nursing home admission. Information can be obtained from Centrelink Tel. 13 27 17.

Carer and Patient Support

Further support for carers can be obtained via the Commonwealth Carer Resource Centre, which has a Carer Information Pack Support Kit that provides information about the support and services available to carers, and offers practical assistance. A copy of the information kit can be obtained by phoning the Commonwealth Carer Resource Centre on 1800 242 636 (toll free from anywhere in Australia). Community services can be accessed via Carelink on 1800 052 222, and respite services via Commonwealth Carer Respite Centres on 1800 059 059.

In addition, Alzheimer’s Australia coordinates a large number of support groups throughout Australia and also offers free specialist counselling services for both carer and patients (Appendix F1). These services can be accessed through the National Dementia Helpline on 1800 639 331.

Aged Care Assessment Teams are also an avenue for further resources for carers and patients.

Other assistance may be obtained via Community Health teams, community nursing services, specialist services (eg psychogeriatricians) and community support services such as Meals-on-Wheels, community transport, personal home nursing care and home help and specialists.
Legal issues
Forward planning of legal and business administration together with discussion of treatment decisions are best addressed as soon as diagnosis is confirmed when the person with dementia may still be able to express their views. Testamentary capacity, Enduring Power of Attorney or Enduring Guardianship, and advanced care directives should be considered (see pp44-5).

Leaving home
Institutionalisation offers the best duration of survival for people with dementia, survival in this context meaning time until death rather than quality of life. However most patients would prefer to remain living in the community, and usually their carer agrees. A patient should not be assessed for optimal home care independently of the carer, and often both patient and carer prefer a formal care package while remaining at home.

Ultimately the requirements of caring become too much for carer and family, and often adversely affect their health. The decision to relinquish full-time care is rarely easy, particularly for spouses, and usually involves emotional turmoil, grief and guilt. Carers need support at this time, available from the Carer Resource Centre, Alzheimer's Australia or face-to-face counselling. Institutional care needs to be considered early because it often takes considerable time to arrange a placement. ACAT assessment is required for admission to a residential aged care facility.

The carer: the ‘second patient’
Patients sometimes make life very difficult for their carers.

Mrs B, aged 82, woke one morning and turned to her husband in alarm. ‘What are you doing there? Get out of the bed. I don't know you.’ The husband’s distress was compounded when the GP arrived and Mrs B greeted him warmly: ‘How nice to see you doctor. Why are you here?’

GPs need to be vigilant about the health of the carer as well as the patient with Alzheimer’s disease even if the carer is not their patient. Encourage carers to join the Alzheimer’s Australia and to contact the Carer Resource Centre for education and support. Suggest alternative or respite care arrangements rather than waiting for carers to mention them.

Particularly stressful are: sleep disturbance, incontinence, immobility/falls, repetitive demanding behaviour and aggression. ‘Negative’ symptoms grind down the carer and produce a build-up of strain over time. Spouses are generally more stressed than other kin. Problems may be exacerbated by grief at the loss of the relationship that previously existed.

Carers’ stress can be worsened if other family members or close friends have differing views about management. Such difficulties should be sought out and dealt with tactfully.

A problem-focused approach, compared to an emotion-based approach to caregiving appears to protect against strain. Similarly, those carers adopting a managerial rather than ‘hands-on’ style of caregiving tend to be less strained.

There is a great deal of descriptive and anecdotal data indicating that support services are helpful in many ways to carers and people with dementia. Training programs for carers have been shown both to relieve strain and to delay institutional placement.

Respite care
Consideration of respite care is an essential part of a long-term plan. This can give carers the opportunity to have a break, and allow the patient to experience another environment without it being a permanent break. However it must be planned carefully, as respite care in a unit with severely demented patients may be traumatic for a patient with early dementia. Respite care needs to be planned well in advance as it is rarely available at short notice. It must usually be arranged through an Aged Care Assessment Team.

Abuse
Recognition of abuse may be difficult and requires awareness of the possibility and tactful inquiring about the stresses of caring. Abuse can be physical, psychological, financial or sexual. The person with dementia can sometimes be the abuser (see p48).
Initial stage management

Management of dementia – the early stage

Management priorities and urgency will vary from patient to patient, but in the early stages there will be a need to address:

- assessment
- diagnosis
- deficits
- assets
- other health issues
- counselling and education
- patient
- carer
- medical management of dementia, behaviour and co-morbidity
- extended family interview
- legal planning
- driving
- financial planning
- support from others.

As these are dealt with, a long-term plan needs to be developed with formulation of potentially achievable objectives against which progress can be measured.

Long-term management plan

The long-term plan needs to be modified as time goes by to take into account changes in the patient, family, carer and social situation. Areas to consider include:

- support for the person with dementia
- support for the carer
- increasing dependence
- personality changes
- behavioural disturbances
- psychiatric co-morbidity
- aged care services
- social services.

Follow-up

As dementia is a progressive disease, ongoing follow-up and continuity of care are essential. Management should aim to anticipate developments and therefore minimise difficulties they might cause. During follow-up visits with the patient and their carer, it is important to explore:

- cognitive function, including any changes (especially if they are acute)
- functional ability, especially alterations in daily living skills such as shopping or travelling
- behaviour, including mood and motivation
- general health, including sleep, nutrition, continence, balance and mobility/gait.

Ask the carer:

- How they are coping with looking after the patient
- Whether they need assistance or respite care
- How their own health is and how they are looking after themselves.

Audit of care for persons with dementia

An audit checklist for the general practice management of a person with dementia is useful (see p7).

Referral

Referral should be considered when:

- progress with any of the problems is unsatisfactory to doctor, patient, family or carer
- there are multiple unresolved problems
- symptoms are causing acute distress
- there are difficult behavioural problems
- respite care or other community support services are needed.

Referrals should be made with specific stated objectives.
Background and supporting evidence

**General background**

**Prevalence**

For every 1,000 patients that an Australian GP sees, he or she can expect to find 10 patients with moderate to severe dementia and another 10 patients with mild dementia. The incidence of Alzheimer’s disease is dependent on age, with the prevalence doubling every 5 years from the age of 65 (Figure 1). It is estimated that 5-10% of elderly people and up to 50% of those aged over 85 years have some degree of Alzheimer’s disease (Katzman 1994).

**Figure 1. Prevalence of dementia in the population**


**Dementia in general practice in Australia**

The BEACH study of morbidity and treatment in general practice 1998–2000 contains information from 2,031 GPs who each recorded information about 100 consecutive encounters. There were 863 encounters (0.4%) with patients with dementia, with 7% of these encounters being a new diagnosis. The encounters were reported by only 431 of the GPs (21%); 39% occurred in nursing homes.

Most (82%) of the patients with dementia were over 75 years of age, and 69% were female. At their encounters they presented 154 reasons for encounter per 100 encounters, of which 28% were dementia, 15% were check-up, 13% were memory disturbance, and 9% were psychological or behavioural symptoms. Apart from check-up, these were uncommon reasons for encounter overall.

Other problems were dealt with in 96% of patients, most often hypertension (7%), heart disease (8%), diabetes (4%), depression (2%), and cerebrovascular disease (2%). Other problems in general, and all those mentioned except hypertension, were much more common in patients with dementia than in patients overall.

Prescriptions were issued much less frequently for patients with dementia (29 per 100 encounters) than for patients overall (64 per 100 encounters) and most were for psychotropic drugs (Bridges-Webb 2002).

**Genetics**

Genetic factors are important in the development of dementia, particularly Alzheimer’s disease, but most cases are sporadic; it is familial in less than 10% of cases (Panegyres 2000).

The presence of the apolipoprotein E4 (ApoE4) allele on chromosome 19 increases the probability that a patient with dementia has Alzheimer’s disease, while its absence makes it less likely.

Although the ApoE4 allele and Alzheimer’s disease are closely linked, not everyone with ApoE4 develops the disease and, conversely, not all patients with Alzheimer’s disease carry the allele. Therefore, although it is a risk factor, the use of ApoE4 genotyping to predict future risk of Alzheimer’s disease is currently not recommended (National Institute on Ageing 1996), (Grey Matters 7:3).
Neuropathology

The most characteristic neuropathological features of Alzheimer's disease are amyloid plaques, neurofibrillary tangles, neuronal loss and cortical and central atrophy. Amyloid protein is believed to play an important role in the pathogenesis of Alzheimer's and may be critical for the formation of amyloid plaques, which appear to reflect damage to the surrounding nerve endings. This neuronal damage causes impaired neurotransmission and results in the cognitive deficits associated with Alzheimer's (Grey Matters 7:3).

Risk factors

There are multiple risk factors for Alzheimer's disease. Almost certainly a variety of factors, both genetic and environmental, can contribute concurrently to its development; however, a number of specific risk factors have been associated with its onset, and should be inquired about.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increasing age</td>
<td>The prevalence of Alzheimer's disease doubles every 5 years in the elderly</td>
</tr>
<tr>
<td>Family history</td>
<td>A family history of Alzheimer's disease increases risk 2 to 4 times</td>
</tr>
<tr>
<td>Sex</td>
<td>Women appear to be at greater risk than men, but this may be linked to the longer life expectancy of women</td>
</tr>
<tr>
<td>Head trauma</td>
<td>Repeated trauma increases the risk of developing Alzheimer's disease</td>
</tr>
<tr>
<td>Down's syndrome</td>
<td>All patients with Down's syndrome develop the neuropathological (although not necessarily the clinical) features of Alzheimer's disease by the age of 40</td>
</tr>
<tr>
<td>Education</td>
<td>Patients with a lower level of formal education are more likely to develop Alzheimer's disease</td>
</tr>
</tbody>
</table>

The links between Alzheimer's disease and other potential risk factors such as aluminium and environmental pollutants have not yet been proven (Grey Matters 7:3).

Other risk factors may be important in other sub-types of dementia, such as arteriosclerotic vascular disease for vascular dementia (see the section on the sub-types of dementia pp38-9).
Ageing and cognitive decline

During normal ageing the speed at which individuals acquire information gradually declines, but their ability to recall information remains relatively preserved. In Alzheimer’s disease, there is a progressive decline in learning and the ability to recall information. There is also an intermediate state – cognitive impairment without dementia – which is in fact twice as prevalent as dementia in elderly populations (Graham 1997). Such individuals have lower than normal cognitive ability but it is insufficient to cause functional decline (refer to Appendix E1 for DSM IV criteria for Alzheimer’s disease).

The usual diagnostic standard for dementia consists of detailed assessment of mental status and careful investigation to rule out other causes of cognitive impairment. A variety of abbreviated instruments have been examined for their ability to screen for dementia in the outpatient setting (see Appendix B).

Advantages of early recognition of dementia

1. Opportunity to discuss concerns and feelings and gain some control of the situation.

2. Treatment issues – effective therapy, including drugs and behavioural therapy, is more likely to be of use in the early stages of the condition. The newer specific drug treatments for Alzheimer’s disease are most effective if used in the early stages. Treatment of conditions that may cause dementia, such as hypothyroidism or vitamin B12 deficiency, is essential, and optimal treatment of other co-existent conditions can improve the patient’s functioning.

3. Medico-legal issues – an early diagnosis allows for the following:
   - an Enduring Power of Attorney and Enduring Guardianship can be arranged
   - any advance directives can be discussed
   - car driving safety can be assessed
   - safety around the house can be assessed.

4. Education and Support of Carers – a good understanding of dementia by carers will assist in better management and lessen breakdown in relationships. Early diagnosis helps family and carers to make contact with support agencies that will help develop the support strategies and services that will be vital as the condition progresses. Forward planning is aided by access to accurate information and education (NZ Guidelines 6:15).

‘Use it or lose it’ applies to physical as well as mental activity. Patients need to be encouraged to maintain physical activity appropriate to their interests and physical state, and this needs to be built into their routine.

Recognition of dementia

The GP may become aware of the possibility of dementia in three ways:

- presenting problems
- noting early pointers when treating other conditions
- screening.

Presenting problems

A ‘typical’ presentation of early dementia

The patient:

- is brought to the doctor by a spouse, family member or friend
- tends to look at his or her carer when asked a question (the ‘head-turning sign’)
- has difficulty recalling the present date and finding words
- may forget recent events but immediate and long-term memory tend to be intact
- tends to minimise or rationalise problems
- has had a ‘memory problem’ for at least six months, with insidious onset and gradual progression
- shows mild impairment on cognitive screening, including impaired recent memory and difficulty drawing a clock.
With Alzheimer’s disease (the commonest type of dementia), the patient:

- is unlikely to have a history of cerebrovascular events, headaches or seizures
- is unremarkable on medical and neurological examination apart from higher cortical functions (Grey Matters).

There are, however, other less common types of dementia which may present in different ways. These are discussed in the section on the sub-types of dementia (pp38-9).

### Psychiatric symptoms/syndromes in dementia

#### Delusions (false beliefs) (30% of cases*)
- usually paranoid type: theft, infidelity, persecution, abandonment
- often evanescent rather than fixed/systematised
- usually occur in mid-stages; early onset predicts poor prognosis.

#### Misidentifications (30% of cases*)
- failure to recognise own home, delusion of ‘phantom boarder’
- misidentification of other people, accusations of others being imposters
- mistaking TV for reality, mistaking mirror image.

#### Misperceptions (illusions) and hallucinations (25% of cases*)
- visual more common than auditory: eg deceased relatives
- more common in later stages; indicates poor prognosis.

#### Depressive symptoms (20-40% of cases*)
- more common in earlier stages of dementia and in vascular dementia
- correlates with degree of disruption to brain monoamine systems (and possibly to retained insight)
- more common if previous history of depression
- carers’ observations important in making diagnosis
- diagnosis difficult as many symptoms (eg apathy, loss of interest, sleep/appetite disturbance
- agitation/retardation (can be due to dementia alone).

#### Anxiety states (up to 40% of cases*)
- mostly situational anxiety with unfamiliar situations or if left alone
- catastrophic reactions, panic attacks, compulsive rituals and phobias can also occur, and may require specific intervention.

#### Mania (2-3% of cases*)
Dementia may occasionally present with a syndrome which is indistinguishable from hypomania: overactivity, sleep disturbance, talkativeness, disinhibition, and cheerfulness or irritability.

*At some stage in the course of dementia.*

(From NZ Guideline 6:24)

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### Non-psychotic behavioural disorders

An association exists between acute underlying medical illness and outbursts of aggressive behaviour in people with dementia. A placebo response is seen in 67% of people treated with neuroleptic agents for the control of behavioural disorders in dementia; there is no difference between neuroleptic agents used and no identifiable differences between responders and non-responders. A high proportion of people with dementia of the Levy-body type are sensitive to neuroleptic agents, and appreciable number of these experience a severe reaction. Delusions or misidentifications are associated with a high number of aggressive episodes.

- Any underlying causes of behavioural disorder, eg an acute physical illness, environmental distress, or physical discomfort, should be excluded.
- Where underlying causes are identified they should be managed before prescribing drugs for the behavioural disorder.
- Tranquillisers should not be used routinely to control behaviour disorders in dementia. In crisis situations, the short-term use of neuroleptic drugs may be appropriate.
- Patients with dementia of the Levy-body type should not be treated with neuroleptics.

There is a relation between delusions and aggressive behaviour; aggressive behaviour should be assessed with this in mind.

The care setting and the attitudes of carers (or care teams in an institutional setting) may influence the emergence of behavioural problems (Eicles 1998).
Background and supporting evidence – Patient presentation

Early pointers

Case-finding and warning signs

Up to half of all cases of Alzheimer’s disease may remain undiagnosed and may become apparent only when the individual’s carer dies or becomes unable to cope.

GPs may note early pointers to dementia when treating other conditions. Early diagnosis is important because drug treatment is now available, and much can be done at this stage to improve lifestyle and reduce risks, and provide information and support for carer and family.

It is important to be alert to cognitive impairment in elderly patients and this should be kept in mind during routine appointments.

<table>
<thead>
<tr>
<th>Warning sign</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Memory problems</td>
<td>- trouble recalling time or date</td>
</tr>
<tr>
<td></td>
<td>- impaired ability to recall recent events or conversations</td>
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<tr>
<td></td>
<td>- losing items</td>
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<tr>
<td></td>
<td>- repetitive questioning</td>
</tr>
<tr>
<td>Cognitive problems</td>
<td>- abandonment of complex activities (e.g. finances)</td>
</tr>
<tr>
<td></td>
<td>- difficulty recognising familiar objects or people</td>
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<tr>
<td></td>
<td>- cannot follow the plot of a story</td>
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<tr>
<td></td>
<td>- language problems</td>
</tr>
<tr>
<td></td>
<td>- delirium</td>
</tr>
<tr>
<td>Behavioural changes</td>
<td>- withdrawal and/or inertia</td>
</tr>
<tr>
<td></td>
<td>- inflexible</td>
</tr>
<tr>
<td></td>
<td>- attitude or stubbornness</td>
</tr>
<tr>
<td></td>
<td>- irritability</td>
</tr>
<tr>
<td></td>
<td>- reduced planning and decision making</td>
</tr>
<tr>
<td></td>
<td>- lack of attention to detail</td>
</tr>
<tr>
<td>Specific incidents</td>
<td>- confusion or unhappiness while on holiday</td>
</tr>
<tr>
<td></td>
<td>- inability to recognise familiar faces at family/social gatherings</td>
</tr>
<tr>
<td></td>
<td>- neglect of long-established behaviours (e.g. writing Christmas cards)</td>
</tr>
</tbody>
</table>

These changes are likely to have developed slowly and have no clear date of onset.

(Canadian Consensus Conference 1998, p.3)

Screening

There is insufficient evidence to recommend for or against routine screening for dementia with standardised instruments in asymptomatic persons.

The routine physical examination and patient history is not sensitive for dementia, especially if family members are not present to corroborate patient self-report. The most commonly used short test of cognitive functioning, the MMSE, applied to a population of asymptomatic 65 to 74-year-old people would yield false positive rate of 93%.

The inability to recall the correct date or place is reasonably specific (92-100% – few false positives), but highly insensitive (15-53% – many false negatives) for dementia.

In dementia due to Alzheimer’s disease, neurologic findings, such as release signs, gait disorders, and impaired stereognosis, are usually late findings and are not sufficiently sensitive or specific to screen for dementia.
### 3.2 Assessment

**History and functional assessment**

As the dementing process progresses, awareness of memory problems decreases, leading to less reliable histories from patients. People with dementia cannot be relied on to complain of memory difficulties. The short mental questionnaire is a screening tool that is sensitive to mild dementia. It can be completed by the carer and may have a useful place in identifying people with dementia. Memory complaints by patients correlate with depression. Carers’ complaints about the memory of their relatives correlate with dementia.

Recommendations and important points:

- Insight diminishes as dementia progresses, making the patient’s history less reliable.
- In assessing a person with cognitive impairment, a history of memory problems should be sought from the carer as well as the patient.
- Dementia and other psychiatric symptoms (delusions or hallucinations, or both, usually persecutory in nature and simple in type) may coexist (Eccles 1998).

Remember that the patient must be asked whether he or she consents to others being consulted about his/her health. If an interpreter is required, it is preferable to use an independent person as members of the patient’s family may (consciously or unwittingly) compensate for the patient’s problems (Grey Matters 7:5).

### History from the patient and a reliable informant

The history should include:

- general medical (including vascular risk factors)
- neurological history
- neuropsychiatric history, including behavioural changes
- drugs, alcohol
- family history, particularly in younger onset
- description of onset and progression of cognitive deficits
- initial depression screen (eg ‘Are you feeling sad/down?’) (LoGiudice 1999).

Questions to ask both the patient and the carer include the following:

- Who first noticed a problem?
- What changes have been noticed?
- How and when did it start? Can a specific date or period be given?
- How is the situation progressing? Are the changes slow and smooth or sporadic?
- Is there a family history of behavioural or memory problems in old age? (Grey Matters 7:5)

When consulting the carer or family members alone, ask whether the patient has:

- changed their behaviour (eg become irritable, withdrawn, unhappy)
- become forgetful
- become lost in familiar surroundings
- failed to recognise – or shown a lack of interest in – family members
- exhibited difficulties driving, shopping or using the telephone
- not been performing well in their usual work or home duties
- suffered any delusions or hallucinations.

Giving carers checklists to fill in, or asking them to keep a diary of the patient’s behaviour, can help assess the patient’s decline and allow the progression of the condition to be monitored (Grey Matters 7:5). The Neuropsychiatric Inventory Questionnaire (NPI-Q) is one such useful instrument. (Appendix C3) (American Academy of Family Physicians Guidelines 2002, 1).

### Functional assessment

It is important to assess the extent to which the patient’s problems with memory and cognition are interfering with his or her ability to undertake daily activities (See p17).
Physical examination

A small proportion of people with dementia have an underlying abnormality, and when this is treated cognitive function improves. The exact number of people thus affected is uncertain because of problems of study populations. People with Alzheimer-type dementia do not complain of common physical symptoms, but experience them to the same degree as the general population.

Recommendations and important points:
- Health care professionals should be aware of the existence of reversible causes of dementia.
- People with dementia experience physical morbidity to the same degree as the general population, but are likely to under report their symptoms.
- General practitioners should ensure that the following routine tests are performed:
  - haematology (including erythrocyte sedimentation rate)
  - biochemistry
  - serum calcium and phosphate
  - thyroid function
  - simple urine analysis (Eccles 1998).

Does the cognitive loss affect more than one part of the brain?

If the dementia symptoms cannot be attributed to delirium, depression or drugs then a neurological evaluation should be undertaken. In particular, the following should be assessed:
- focal deficits – gait disturbance, motor deficit, sensory deficit
- abnormalities of muscle tone, movement or reflexes.

The neurological history and examination should look for current or past conditions which may produce signs of dementia, including cerebrovascular events, head trauma, epilepsy or infections of the central nervous system such as syphilis or human immunodeficiency virus (HIV) (Grey Matters 7:9).

Investigations

CT scan

The principle reason for conducting a CT scan is to eliminate non-Alzheimer's disease causes of dementia, such as mass lesions or subdural haematomas (Chan 1997). Although the detection of diffuse cerebral atrophy on CT may suggest Alzheimer's disease, its diagnostic specificity is low. A CT scan without contrast should be ordered at some stage in the dementia, but is of more use early in the disease. A CT scan becomes more urgent if the patient with dementia is under 60 years of age, has had a recent head trauma, is using anticoagulants or has undergone a rapid, unexplained decline (Grey Matters 7:9).

Potentially reversible dementia

This refers to syndromes that are, at least partly, reversible following early recognition and treatment of the underlying condition. Potentially reversible components of dementia are important to detect as they have enormous implications for the patient and his/her carer. This has led to the development of a list of tests which should be undertaken in any person with dementia (see p8), to ensure that reversible causes will not be overlooked. However these are encountered rarely when the case is typical of Alzheimer's disease. The use of such tests may reveal abnormalities, which can be corrected and can lead to total reversibility if the dementia is only related to these abnormalities. However there may not be any improvement in cognition if the abnormalities are aggravating a dementia which is fundamentally caused by another disease process (NZ Guideline 6:12-13).
Background and supporting evidence – Assessment

Cognitive assessment

Short assessment tests for cognitive impairment

Mini-mental state examination (MMSE) (Appendix B1)

At present the full mini-mental state examination should be used for assessment though, there is some evidence that it can be shortened for use in primary care with only a small reduction in specificity. Four items of the mini-mental state examination are predictors of dementia:

- orientation to day
- spell WORLD backwards
- recall three words
- write a sentence.

Reducing the mini-mental state examination to two items – recall and orientation for place – reduces the specificity only slightly. The mini-mental state examination may be influenced by verbal fluency, age, education, social grouping and cultural background.

Clock drawing test

In the clock drawing test, the accuracy of the fourth quadrant of the clock face shows the greatest sensitivity (87.5%) and specificity (82.3%) for dementia.

GPCOG (Appendix B2)

This Australian-designed test for use in general practice includes elements of both the above plus an informant interview, and has the advantage of brevity and efficiency. (Brodaty et al. 2002)

(Rowland) Universal Dementia Assessment Scale

This Australian-designed version of the mini-mental state examination is a brief, administratively simple dementia assessment tool that is designed to be culturally and linguistically fair. (Accepted for publication in International Psychogeriatric Association (IPA) Journal, 2003)

Activities of daily living (Appendix C5)

Deterioration in four domains of instrumental activities of daily living are significantly associated with cognitive impairment. These domains are:

- managing medication
- using the telephone
- coping with a budget
- using transportation.

Recommendations

Health care professionals should consider using the following instruments to identify cognitive impairment:

- the mini-mental state examination
- the clock drawing test
- the GPCOG
- (Rowland) Universal Dementia Assessment Scale

The most widely studied of these instruments is the mini-mental state examination, a short, structured examination that takes 5–10 minutes to administer. The MMSE contains 30 items and is reproducible using a standardised version. Various studies suggest that an MMSE score of less than 24 out of 30 has a reasonable sensitivity (80–90%) and specificity (80%) for discriminating between dementia cases and normal controls. There are only limited data, however, on its performance as a screening test for early dementia among a representative population of outpatients. The positive predictive value (PPV) of MMSE for dementia depends on the definition of an abnormal score and the prevalence of dementia. Based on its performance in one community study, a MMSE score of 20 or less has a PPV of only 48% when the prevalence is 10% (eg a population of 75–84 year olds), but a much higher PPV (73%) when prevalence of dementia is 25% (eg age over 85).

The predictive value of intermediate MMSE scores (21–25) appears to be low (21–44%) for dementia in most populations (Guide to Clinical Preventive Services, 1996).
Home visit
(Refer to Guidelines p12)

Delirium

Delirium in the elderly is often a first warning sign that dementia may develop within the next three years and may highlight underlying Alzheimer’s disease (Grey Matters 7:8 and NZ Guidelines 6:23).

Some of the causes include medication effects, infections, vascular changes, hypoxia, metabolic problems, surgery and trauma. Delirium has an increased mortality, increased rate of institutionalisation and increased likelihood of readmission to hospital. One source of diagnostic confusion between delirium and dementia is caused by the mistaken belief that delirium is always of short duration and of florid symptomatology. A sub-acute confusional state can last for months. Recent research has shown that ‘quiet signs’ are common in delirium, such as plucking at bedclothes, poor attention, incoherent speech, slow vague thought and fluctuating mental state (LoGiudice 1999).

Depression

Depression can manifest as dementia; conversely, dementia can present with depressive symptoms early in the illness. To differentiate between depression with cognitive impairment and dementia consider using the Geriatric Depression Scale (Appendix C1), or the short EBAS-DEP (Appendix C2). If in doubt, psychiatric referral is required.

Depression is common in the elderly. The longer the depression is left untreated, the less likely it is to get better. Failure to recognise and treat depression can lead to long-term suffering, disability and even suicide.

In the elderly depression is often overlooked or under treated; major depression occurs in 20-30% of people diagnosed with Alzheimer’s disease (Ames 1994). The criteria for making a clinical diagnosis of major depression are noted in following table.

Depression is common, especially in the physically ill, those in hospital, attendees at GP surgeries, alcoholics, the socially isolated and residents of aged care facilities.

Criteria for major depressive disorder

Before a diagnosis of depression can be made, one of the following two symptoms must be present:

- low (sad, miserable, depressed) mood most of the time which is not relieved by pleasant circumstances
- loss of interest and the capacity to take pleasure in things which the sufferer previously enjoyed (that is not due to circumstances only).

Other symptoms may be:

- loss of energy, tiredness, fatigue and ability not due to other physical factors
- unreasonable feelings of guilt or self reproach
- suicidal behaviour or recurrent thought of death or suicide
- subjective impairment of concentration or thinking ability
- agitation or psychomotor retardation
- excessive or disturbed sleep (especially early waking)
- loss of appetite and/or weight (increase in appetite or weight can occur)
- loss of self esteem and confidence.

The diagnosis of major depression is made when five or more symptoms are present for two or more consecutive weeks.

(American Psychiatric Association 1994)

Depression in patients with dementia

Depressive illness is commoner in people with dementia than those without. Mortality is increased in people with dementia and depression. The prevalence of depression in patients with dementia varies widely according to the study population. In a general population, the prevalence varies from 10-40% of patients with dementia. Depression is more commonly diagnosed or recognised in early dementia. Treatment is likely to be of value, with reported response rates of up to 85%. Depression commonly leads to difficulties in communication and independent activities of daily living and has a less common effect on cognitive function. Presenting symptoms relate to inner feelings (anxiety, mood, loss of interest, helplessness, hopelessness and worthlessness) and less to vegetative symptoms.
Recommendations and important points:

- Depression can occur in patients with dementia at any stage in the dementing process.
- The history should be gathered from both the patients and their carers.
- Relevant risk factors for depressive illness, such as personal or family history of depression, or recent adverse events, such as bereavement or relocation, should be considered.
- Consider a trial of antidepressant medication evaluated against explicit criteria such as activities of daily living, level of functioning, behavioural disturbance, and biological features of recent onset (Eides 1998).

**Differential diagnosis**

The most common cause of dementia is Alzheimer's disease, which accounts for about half of the cases seen. Other types of dementia are listed below.

**Features that may distinguish depression from dementia**

<table>
<thead>
<tr>
<th>Feature</th>
<th>Primary depression</th>
<th>Primary dementia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>General features</strong></td>
<td>– family aware of illness</td>
<td>– family often unaware of illness</td>
</tr>
<tr>
<td></td>
<td>– onset more acute and can be dated</td>
<td>– insidious onset, only vaguely dated</td>
</tr>
<tr>
<td></td>
<td>– symptoms of short duration</td>
<td>– symptoms of long duration</td>
</tr>
<tr>
<td></td>
<td>– rapid progression</td>
<td>– slow progression</td>
</tr>
<tr>
<td></td>
<td>– family history of affective disorder</td>
<td>– possible family history of dementia</td>
</tr>
<tr>
<td><strong>Patient’s history</strong></td>
<td>– past history of depression</td>
<td>– no history of depression</td>
</tr>
<tr>
<td></td>
<td>– seeks help with complaints of memory loss</td>
<td>– few complaints of memory loss</td>
</tr>
<tr>
<td></td>
<td>– complaints given in great detail</td>
<td>– vague, non-specific complaints</td>
</tr>
<tr>
<td></td>
<td>– cognitive deficits emphasised</td>
<td>– cognitive deficits concealed</td>
</tr>
<tr>
<td></td>
<td>– failings highlighted by patient</td>
<td>– accomplishments highlighted by patient</td>
</tr>
<tr>
<td><strong>Mental state observations</strong></td>
<td>– history consistent and sequential</td>
<td>– inconsistent history with poor temporal sequencing</td>
</tr>
<tr>
<td></td>
<td>– patient makes little effort with tasks and readily gives up</td>
<td>– patient struggles with tasks</td>
</tr>
<tr>
<td></td>
<td>– subjective distress common</td>
<td>– efforts sustained and may use cues or evasions</td>
</tr>
<tr>
<td></td>
<td>– affective symptoms pervasive</td>
<td>– unconcerned attitude common</td>
</tr>
<tr>
<td></td>
<td>– complaints greater than observed dysfunction</td>
<td>– affect may be shallow or labile</td>
</tr>
<tr>
<td><strong>Cognitive testing</strong></td>
<td>– ‘don’t know’ answers common</td>
<td>– observed dysfunction greater than complaints</td>
</tr>
<tr>
<td></td>
<td>– recent and remote memory loss more equal</td>
<td>– frequent ‘near miss’ answers</td>
</tr>
<tr>
<td></td>
<td>– poor memory for specific periods common</td>
<td>– orientation tests poor</td>
</tr>
<tr>
<td></td>
<td>– concentration worse than general knowledge or memory</td>
<td>– recent memory worse than concentration</td>
</tr>
<tr>
<td></td>
<td>– test performance may be highly variable</td>
<td>– consistently poor test performance</td>
</tr>
<tr>
<td></td>
<td>– no typical WAIS-R pattern</td>
<td>– WAIS-R performance scores worse than verbal scores</td>
</tr>
<tr>
<td><strong>Neurological</strong></td>
<td>– no primitive frontal release reflexes</td>
<td>– frontal reflexes may be present</td>
</tr>
<tr>
<td></td>
<td>– no dyspraxias or agnosias</td>
<td>– dyspraxias and agnosias common</td>
</tr>
<tr>
<td></td>
<td>– no language difficulties, corrects</td>
<td>– word finding problems and paraphasia common</td>
</tr>
<tr>
<td></td>
<td>– paraphasic errors</td>
<td>– CT head scan usually abnormal, with cerebral atrophy</td>
</tr>
<tr>
<td></td>
<td>– CT head scan more commonly normal</td>
<td></td>
</tr>
</tbody>
</table>

(NZ guidelines 6:23)
Background and supporting evidence – Assessment

Types of dementia

<table>
<thead>
<tr>
<th>Type of Dementia</th>
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</thead>
<tbody>
<tr>
<td>Alzheimer’s disease</td>
</tr>
<tr>
<td>Vascular dementia</td>
</tr>
<tr>
<td>Lewy-body dementia</td>
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<tr>
<td>Frontal lobe dementia</td>
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<tr>
<td>Parkinson’s disease with dementia</td>
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<tr>
<td>Normal pressure hydrocephalus</td>
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<tr>
<td>Post-traumatic, toxic (particularly alcohol) or anoxic encephalopathy</td>
</tr>
<tr>
<td>Prion diseases eg Creutzfeldt Jakob disease</td>
</tr>
<tr>
<td>Multi-infarct dementia</td>
</tr>
<tr>
<td>Down’s syndrome</td>
</tr>
<tr>
<td>AIDS</td>
</tr>
<tr>
<td>Other</td>
</tr>
</tbody>
</table>

(LoGiudice, 1999)

Dementia sub-types

Alzheimer’s disease

In addition to progressive memory impairment (especially recently acquired memories), language impairment is an important sign of Alzheimer’s disease. The earliest difficulties may be in finding words in spontaneous speech and in the increased use of automatic phrases and cliches (eg social speech such as ‘How are you?’ ‘I’m fine’ etc). The ability to repeat phrases is usually preserved. Other deficits occur with visual and spatial abilities such that there may be difficulties in recognising familiar faces or objects. Apraxias (difficulty completing complex motor tasks) may interfere with abilities to carry out activities of daily living. Impairment in arithmetic (acalculia) may interfere with managing accounts and/or a cheque book (NZ Guidelines 6:11).

Vascular dementia

Risk factors include hypertension, diabetes, atrial fibrillation and a history of myocardial infarction. A computed tomography (CT) scan without contrast may help confirm or exclude a vascular aetiology (Grey Matters 7:9).

In up to 90% of pathologically verified cases of vascular dementia, there is history of acute unilateral motor or sensory dysfunction consistent with a stroke. Vascular dementia, however, can occur in the absence of overt strokes. Urinary dysfunction and gait disturbance are possible early markers. Parkinsonian motor features, asymmetric reflexes and/or extensor plantar responses are useful signs. Cognitive decline tends to be discontinuous and deficits are often patchy (NZ Guidelines 6:11).

Dementia of the frontal lobe type

This syndrome probably accounts for 1-5% of all cases of dementia. Dementia of the frontal lobe type describes the syndrome of disordered executive function (impairment of initiation, goal setting, and planning) and disinhibited behaviour with only mild abnormalities on cognitive testing. These people are prone to angry catastrophic reactions. The apathy may be difficult to distinguish from depression. The apraxias of Alzheimer’s disease are usually absent and the language deficits are more characterised by abundant unfocused speech (logorrhea), echo-like spontaneous repetition of words or phrases (echolalia) or compulsive repetition of phrases (palilalia).

One cause of this syndrome is Pick’s disease which is associated with focal atrophy of one or both frontal and/or temporal lobes. (NZ Guidelines 6:11).

Lewy-body dementia

The clinical course of dementia of the Lewy-body type differs from that of Alzheimer’s disease, showing clear fluctuations with the following clinical features:
- complex visual hallucinations (48%)
- auditory hallucinations (14%)
- paranoid delusions (57%)
- clouding of consciousness (81%)
- falls or collapses (38%)
- depression (38%)
- extrapyramidal features (9.5%).

There is high neuroleptic sensitivity (61.5%) and a high risk of increased morbidity and mortality if neuroleptic drugs are prescribed.

Recommendations and important points:
- Differential diagnosis of dementia of the Lewy-body type is important because of the high risk of increased morbidity and mortality with neuroleptic agents in these patients.
Doctors should be aware of the importance of avoiding neuroleptic drugs in people known to have dementia of the Lewy-body type (Eccles 1998).

**Subcortical dementia syndrome**
This refers to a clinical syndrome characterised by slowing of cognition, memory disturbances, difficulty with complex intellectual tasks such as strategy generation and problem solving, visuospatial abnormalities and disturbance of mood and affect. Unlike Alzheimer's disease there is relative preservation of language, calculation and tasks requiring co-ordinated motor function. This syndrome may be seen in conditions such as Parkinson's disease, Huntington's disease, progressive supra-nuclear palsy, Wilson's disease and other disorders affecting predominantly the basal ganglia and/or thalamus (NZ Guidelines 6:11).

**Ability/disability**

**Older road users – issues for general practitioners**
Advice from medical practitioners is often heeded by older patients in relation to their ability to drive. Using resources such as the Austroads publication *Assessing Fitness to Drive* and *Medical examinations of commercial vehicle drivers* will aid the GP in making an informed decision in relation to this (Appendix F2).

**Co-morbidity**
(Refer to Guidelines p18).

**Family/social support and environment**

**Assess carer and family**
The stress associated with caring for a person with dementia should never be underestimated. It places an extraordinary burden on those who undertake the caring role.

Some people find themselves unwittingly and unwillingly in the role of carer. Other family members may look to one member of the family to take on this role without considering whether this person has the desire, ability or emotional capacity and physical health to cope. For some families, geographical location may place responsibility for care on one member only.

Several factors should be considered when evaluating the strengths of a caregiving relationship and the degree of burden likely to be experienced.

The ability to cope with caring depends on the:
- symptoms exhibited by the person with dementia
- type, frequency and disruptive effects of aberrant behaviour
- duration and severity of the dementia symptoms
- carer’s response to these symptoms and tolerance of aberrant behaviour
- formal and informal support services available to assist
- carer’s emotional and physical health
- carer’s perception of whether they have sufficient emotional support
- quality of the carer’s relationship with the person with dementia prior to the onset of dementia
- carer’s ability to make lifestyle adjustments
- carer’s ability to take over responsibilities and decision-making within the home
- carer’s other commitments.

Difficulties experienced with any one of the above areas can be enough to produce sufficient stress to place either the person with dementia or the carer at risk, or jeopardise the success of community care. It needs to be recognised that carers often become physically and mentally exhausted over time. This can have a profound effect on their decision-making ability. It is important to be aware that carer stress can lead to abuse and neglect of the person with dementia.

Stress may also be caused by the need for carers to take on the roles and functions formerly performed by the person with dementia. A son or daughter may find themselves in the role of parent to their own parent. The person with dementia may resist and oppose this carer’s good intentions.

Grief is a constant feature of dementia. Carers have described the journey through dementia as ‘the funeral that never ends’. As losses continue throughout the progress of the dementia, so the grief process is ongoing. There is potential for carers to become depressed.
Signs of stress:
- self reported stress
- increased dependency on alcohol or other drugs
- reported weight loss or gain
- sleep disturbance.

Assessing stress levels
Ask the carer ‘How is this affecting you?’
‘What has changed for you?’ Ask about the carer’s mood level. Note any changes in the carer’s health which could be stress-related.

The Caregiver Burden Scale is a self-administered 22-item scale that is a useful tool for measuring the distress of caregivers (Appendix D1) (American Academy of Family Physicians Guidelines 2002, 1).

Action plan
Overarching principles for the general practitioner:
- not all dementias are the same
- quality of life for the person with dementia
- the hidden second patient
- dementing not demented – the evolving, ever-changing picture
- dementia – the long haul
- vulnerability of people with dementia
- liaising with other services
- treating patients who can’t consent – medico-legal issues
- advances in knowledge (Brodaty 1996).

What, how and when to tell patient and family
Giving patients a diagnosis of Alzheimer’s disease is often seen by many as equivalent to giving a diagnosis of incurable cancer (Grey Matters 7:11).

Prognosis
As Alzheimer’s disease is a progressive neurodegenerative disease, it will affect different individuals in different ways. Throughout the dementing process, changes will continue to occur; some problems may become exaggerated, others will paradoxically subside.

On average, the time from onset of the disease to diagnosis is about 2-3 years, while from onset to death is usually 10 years. Although the progress of Alzheimer’s disease is gradual, the following table gives some indication of the changes that will occur as the disease progresses. (Grey Matters 7:12).

At some time during the dementia, behavioural complications will affect 90% of patients with Alzheimer’s disease. Psychological/psychiatric complications include depression, anxiety, psychosis or hallucinations, while non-psychological behavioural complications include agitation, wandering, screaming and aggression. These problems warrant treatment when they impair self-care or social interactions, or when they are likely to lead to institutionalisation. Patients with these behaviours can be difficult to manage but they are often over-medicated; as these behaviours change over time, medication for such complications should be reviewed at least every 6 months (Grey Matters 7:12).

Referral
(Refer to Guidelines p18).

Typical stages in the development of Alzheimer’s disease; each individual will be affected differently

<table>
<thead>
<tr>
<th>Moderate Alzheimer’s disease</th>
<th>Severe Alzheimer’s disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>– Approximately 4-7 years from onset.</td>
<td>– Approximately 7-10 years from onset.</td>
</tr>
<tr>
<td>– Deficits in working memory, attention span and language comprehension.</td>
<td>– Loss of most memories and inability to use or comprehend language.</td>
</tr>
<tr>
<td>– Personality traits become flattened or exaggerated (eg lack of inhibition, impulsiveness, coarseness).</td>
<td>– Motor symptoms become more pronounced: extrapyramidal signs and gait abnormalities.</td>
</tr>
<tr>
<td>– Problem behaviours emerge (eg wandering, shouting, clinging).</td>
<td>– Problem behaviours may abate; patient may become easier to care for.</td>
</tr>
<tr>
<td>– Psychiatric complications (eg hallucinations, delusions, paranoid ideation, suspiciousness).</td>
<td>– Increasing dependence on others for basic needs; residential care and eventually death.</td>
</tr>
</tbody>
</table>
3.3

Management

### Dementia and disability

#### Drug treatments for dementia

This is a rapidly developing area of research, and changes are occurring quickly. Two recent reviews are recommended for more detailed information (American Academy of Neurology 2001, 4; Brodaty H et al. 2001).

#### Cholinesterase inhibitors

The evidence that cholinesterase inhibitor drugs benefit patients with Alzheimer’s disease by delaying the onset and rate of functional decline and preserving the ability to perform certain activities of daily living has recently been reviewed (American Academy of Neurology 2001, 4; Brodaty H et al. 2001). Although an improvement in cognition has been correlated with plasma drug concentrations and the level of acetylcholinesterase inhibition in red blood cells, there has been no direct evidence to show that these drugs specifically alter the neuropathology of the disease process.

Cholinesterase inhibitor drugs including donepezil, rivastigmine and galantamine stop the breakdown of acetylcholine in the brain to reduce the apparent loss of cholinergic neurotransmitter activity in individuals with Alzheimer’s disease. Most patients benefit to some extent, but only 50-60% show a measurable response to treatment, and it is not possible to predict which ones before treatment is started. Four to eight patients need to be treated for one to show measurable benefit (Brodaty H et al.).

Some patients, 15-50% in various studies, experience predictable cholinergic adverse effects, predominantly gastrointestinal – nausea, vomiting, diarrhoea – but also including many others such as bradycardia, muscle cramps, fatigue, dizziness, headaches, agitation and insomnia. These are dose-related and usually occur within the first few weeks of treatment. The side effect profile of the different drugs varies somewhat, but there is no direct evidence as to their relative merits.

The drugs’ effects on cognition equate to about 6-9 months of preserved cognitive function. There are no known patient or disease characteristics that predict a positive response to treatment.

These treatments have an as yet undefined role in the overall management plan for individuals with Alzheimer’s disease. Treatment should be initiated only for patients with ‘probable’ Alzheimer’s disease of mild to moderate severity (ie MMSE scores of >10), and where there is a family member or other caregiver available to monitor compliance, effectiveness and adverse effects. There is now increasing evidence of benefit in Lewy-body dementia, but no evidence of benefit for patients with other types of dementia. Availability of the drugs under the Pharmaceutical Benefits Scheme is restricted.

Treatment should be initiated with clearly defined treatment goals and with an ability to assess effectiveness. The chosen measures and outcomes for monitoring should be meaningful to the patient and/or caregiver.

Treatment should be initiated at low dosages and titrated according to tolerability up to the maximum recommended by the manufacturer. Patients should be monitored for adverse effects in the first 6 weeks of commencing treatment or after dosage adjustment. Effectiveness should be assessed after 3 months of treatment at the highest tolerated dosage.

Treatment for longer than 6 months should be based on a clear response, which may include stabilisation of symptoms, preferably as assessed by objective measures. Treatment should be discontinued if there are significant adverse effects, poor compliance, failure to meet the chosen treatment outcomes, or a significant deterioration in the patient’s condition. All patients on long-term treatment should be reassessed at least every 6 months and consideration should be given to the cessation of treatment in order to judge the adequacy of response.
Other drugs

- Anti-inflammatory drugs – epidemiological evidence suggests that these may prevent or delay the onset of Alzheimer's disease, but there is insufficient evidence that they are effective in its treatment, and their side effect profile means that their use is not recommended.

- Vitamin E – one randomised controlled trial supports epidemiological evidence that delays clinical decline, but more evidence is needed.

- Selegiline has beneficial effects, but the evidence is as yet insufficient to recommend routine clinical use.

- Many other drugs have been thought to be beneficial, including gingko biloba and oestrogen, but the evidence is as yet unconvincing.

\[(American \ Academy \ of \ Neurology \ 2001, \ 1; \ Brodaty \ H \ et \ al. \ 2001; \ National \ Guidelines \ Committee \ for \ Anticholinesterase \ Inhibitors \ 2000)\]

Psychotropic drugs

General

Medication can be very helpful in treating some behavioural problems, but should not be regarded as first-line treatment (except in emergencies). Other strategies should be tried first and continue in parallel with drug treatment.

Psychotropic medication will not solve disinhibition or wandering without producing over-sedation; nor will it help negative symptoms or incontinence.

The specific goals of treatment should be clear at the outset.

The golden rule is to start with low doses and increase slowly, whilst carefully monitoring both beneficial and adverse effects. Dosage times should be tailored to the target problem – behaviour is often most difficult in the latter part of the day. The benefits and risks of treatment should be openly discussed with the patient (if possible) and carers. In this way unrealistic expectations and fears can be dispelled.

Adverse effects are unfortunately very common. These include: sedation, confusion, decreased mobility, low blood pressure and Parkinsonism.

It is also important to be alert to the possibility of paradoxical worsening of behaviour. Once instituted, drug treatment should be reviewed on an ongoing basis and attempts made to reduce or withdraw it. Many behavioural problems are relatively short-lived, so psychotropic drugs should not be prescribed indefinitely.

Drugs used

Major tranquillisers are the usual first-line drug treatment for agitation or aggression (especially if associated with psychosis) and have shown modest efficacy in controlled trials. Haloperidol and thoridazine are the most commonly used. Other neuroleptics include pericyazine, loxapine, thiothizene and pimozide. However these drugs are not recommended for treatment of Lewy-body dementia. In addition, a prospective study has suggested that neuroleptic drugs may hasten cognitive decline in dementia. Whilst further studies are needed to confirm this finding, it emphasises the need for caution and judgment in the use of these drugs.

The place of newer neuroleptics (olanzapine, risperidone) is being evaluated. They have a lower incidence of extrapyramidal side effects and may be more efficacious (Canadian Consensus Conference 3:10). These drugs are not currently PBS listed and so if used patients would be charged the full cost.

If anxiety appears to be driving the behaviour problem, shorter-acting minor tranquillisers may be tried, eg chlormethiazole, oxazepam or alprazolam. For sleep disturbance a course of a shorter-acting sleeping tablet, eg temazepam or zopiclone can be useful (NZ Guideline 6:26).

Managing behavioural concomitants of dementia

There is controversy about how to classify these symptoms, but the importance of assessment and intervention for behavioural problems is increasingly recognised, due to their impact on carers’ burden and coping ability. Due to problems with definition, there is a lack of good data on prevalence of particular symptoms, but it is agreed that behavioural problems of some sort arise in the great majority of dementia cases.
Recent reports have highlighted the occurrence of ‘negative’ symptoms (apathy, withdrawal and emotional blunting) in addition to positive behaviours. Behavioural problems are commonly associated with psychiatric symptoms; eg aggression may be attributable to psychosis. It is particularly important to be aware that a depressive or anxiety state may underlie behavioural disturbance, in which case management should be focused accordingly (see section on Co-morbidity below) (NZ Guidelines 6:25).

Non-drug management strategies
Recent management practices in dementia care are moving away from a problem-oriented focus, toward the assessment and fostering of an individual’s personhood and residual strengths.

Social work support and other counselling interventions such as those listed below, have been advocated for people with dementia, but few have been scientifically validated. The availability of these approaches varies widely, depending on local interest and expertise. All of these may be beneficial.

Specific activities or therapies which may be available from aged care services:
- reality orientation
- reminiscence
- validation therapy
- behaviour modification
- cued recall
- music therapy and dance
- motivational therapy
- doll therapy
- water therapy (NZ Guidelines 6:26).

Co-morbidity
Depression
Many clinicians feel that the newer antidepressants such as selective serotonin reuptake inhibitors (SSRI’s) are preferable to tricyclics, due to a more benign side-effect profile (in particular, less anticholinergic action which can make confusion worse). However, agitation, sleep disturbance and low blood sodium levels can sometimes prove troublesome with SSRI’s. Moclobemide also has proven efficacy and is generally well tolerated (NZ Guidelines 6:24).

Anxiety states
Sometimes patients may benefit from more formal anxiety management strategies, behaviour modification, or counselling. If these strategies have failed or are unrealistic, anti-anxiety, anti-panic or anti-phobic drug treatment can be helpful (NZ Guidelines 6:24).

Cerebrovascular disease
The medical management of vascular dementia is the same as for stroke disease. There should be careful treatment of hypertension (whilst avoiding hypotensive episodes), hyperlipidemia, carotid atherosclerosis and atrial fibrillation (NZ Guideline 6:19). Aspirin (75mg) may reduce the risk of further vascular events (Eccles 1998).

Other conditions
Other medical conditions warranting preventive measures or optimal management in the dementing patient are: diabetes (particularly, avoidance of hypoglycemia), hypoxia, anaemia, postural hypotension, epilepsy, infective illness, pain and urinary or faecal retention. Even apparently trivial medical problems (eg mild dehydration) can worsen dementia (NZ Guideline 6:19).

Health promotion
There is evidence that cognitive decline in dementia may be delayed by participation in stimulating intellectual activities (the ‘use it or lose it’ theory). People with dementia should be encouraged, as far as is possible, to maintain their customary hobbies and activities (NZ Guidelines 6:19).

Diet
Adequate diet is even more important in patients with dementia than in other older patients. Special attention needs to be paid to avoid or dealing with obesity or loss of weight, and ensuring an adequate dietary intake of vitamins and other essentials, since dementing patients may become difficult about taking their meals. Regular inquiry about what is being eaten should be made.
Background and supporting evidence – Management

**Prevention**

**Falls**

Fracture of the hip is the commonest fracture in falls associated with dementia. Medication increases the risk of falling in people with dementia. Falls are not associated with the severity of the dementia but are associated with wandering and reversible confusion. Those people who fall are more likely to fall again and falls are associated with their doing too much, eg wandering, restlessness. Falls are increased in the more capable groups of people with dementia (Eccles 1998).

Prevention of falls requires when possible recognition and alteration of environmental risks, modification of risk behaviours and appropriate physical assistance. This usually requires a multifactorial approach, but specific interventions may be needed in individual patients, such as recommending against the use of bifocals, awareness of appropriate footwear or use of hip protectors (Monagle 2002).

**Patient / family / social support**

Not all people with dementia will have a carer available, and some will have family members who do not wish to take on the care-giving role. Adequate support systems must be set up to enable these people to remain at home for as long as desired and practicable.

It is recognised that people with dementia living alone will usually need to access residential care sooner than those living with a carer. There are complex ethical issues involved in ensuring that a person’s wish to continue living alone is balanced with their safety and that of others (NZ Guideline 6:32).

**Legal issues**

People with dementia often become unable to manage their business, financial or personal affairs. They may be unaware that a problem exists. This makes them at risk from the unscrupulous. Forward planning of legal and business administration together with discussion of treatment decisions are best addressed as soon as diagnosis is confirmed when the person with dementia may still be able to express their views.

Advance care directives relate to advance decisions about the level of medical care to be adopted in specified circumstances when the patient is unable to make decisions. Enduring Guardianship provides for nomination of a person or persons who are to make decisions about personal care on behalf of the patient when the patient is unable to make decisions. Enduring Power of Attorney is similar, but relates only to financial and business matters.

Advance care directives, enduring guardianship, and enduring power of attorney are three aspects of legal decision-making which need to be considered in early dementia while the patient may still be able to make legal decisions. If not done, later there may arise a need for a medical decision about the patient’s capacity to make decisions about medical and personal care, and financial and testamentary matters.

**Decision making capacity**

(see Appendix B3)

Determination of a patient’s capacity to make decisions may be an important role of the doctor. This usually applies in one of three situations:

2. Arranging enduring guardianship or giving an advance care directive.
3. Making a will or giving power of attorney.

It may also apply to other tasks such as managing financial affairs or arranging living circumstances.

Whatever the task, it is important that:

- capacity is task-specific, and must be assessed separately for each decision
- assessment is best made over time, rather than at only one interview, because determination of consistency of response is important
- information from others, with the patient’s consent, is desirable.

Records should be kept as fully as possible, with emphasis on information that explains the basis for the decision.

The following factors need to be considered in determining capacity:

- Attention – can the patient maintain attention for long enough? Maintaining conversation for at least one minute is a minimum.
- Language – comprehension, by hearing or reading; this can be tested by conversation and/or with suitable simple multi-choice questions given orally and/or in writing.
- Language – reply, may be made by the patient in speech or writing, or by gesture, pointing or other understandable means. These forms of communication should not be overlooked when speech or writing is not possible.
- Memory – short and long-term memory need not be perfect, but should be relevant to the task.
- Awareness of the significance of the interview: does the patient understand who is doing it, what it is about and the likely consequences?
- Judgement – can the patient appreciate likely outcomes of decisions made?

**Consent to medical treatment**

In assessing capacity to consent to medical treatment the following factors must be clear:

- what are the options?
- the benefits and risks of each
- the values the patient wants to uphold or goals they wish to reach
- the stability of the decision over time; the consent must be given on at least two different occasions
- the patient must always be included in the decision process to the extent possible
- there must be no coercion or undue pressure from others.

If the patient does not have the capacity to consent, then the decision MUST be made by someone other than the treating team members.

**Capacity to arrange Enduring Guardianship or advance care directives**

In making such arrangements the patient must understand that:

- the choices being made are for the future
- it will be used only if the patient has become incapable
- some choices are about future treatment
- some choices are about who will then decide
- the choices made could threaten life

- coma or dementia means that no choice in the future will be possible
- choices may change over time
- directives should be updated and changed if necessary each year
- choices made in the directive override later choices if the patient has become incapable.

**Capacity to make a will**

In determining capacity to make a will, there are a number of specific requirements:

- the patient’s lawyer should first be consulted
- assessment should occur on two different occasions, the second preferably on the day of executing the will
- the presence or absence of witnesses to the assessment, and if any, who they should be, should be considered
- the patient must be free of undue influence, such as from family member or carer
- the patient must not have delusions or hallucinations which could influence the decisions.

In the assessment, the patient must:

- understand the nature and purpose of the interview, and what he/she is doing
- understand what a will is and when it would come into effect
- be able to describe the extent and nature of his/her property
- be able to understand and state the claims of potential heirs
- state who is to benefit, in what way each will benefit, and give a sensible explanation of why that benefit to that person is desired.

**Dementia and driving**

The issue of fitness to drive is an easier issue for GPs to tackle than some others because there are specific guidelines to be followed that are widely recognised in the community (though not always by the patient with dementia).

Even mild dementia increases the risk of traffic accidents; the risk increases with concomitant morbidities and as the disease advances.
Mildly impaired patients should be asked to stop driving or confine themselves to familiar routes; those with moderate to severe disease should be instructed not to drive at all. In some states it is mandatory to report patients whom one considers unfit to drive; if there is a dispute, the patient should be referred to the local office of the Roads and Traffic Authority. Many of these strategies also apply to patients who could endanger others if they continue working (eg doctors, engineers) (Grey Matters 7:12).

The practitioner, in making the notification, must be satisfied not only about the person's medical unfitness to drive, but also be aware that the licence holder may probably continue to drive despite medical advice.

Information should also be sought from the caregiver about the person's continuing ability to drive safely. Many small incidents can illustrate deteriorating ability long before a serious or life-endangering accident occurs. The role of the medical practitioner in encouraging the person to give up driving cannot be understated. This task must not be left to caregivers or families. Medical practitioners may find this difficult, involving as it does a loss of independence for someone who may have been a patient for many years. The discussion may provoke anger on the part of the patients. Some may feel that there is an ethical dilemma at stake. This should be seen, however, as a situation where the safety of others outweighs the rights of the individual.

Some people will acknowledge the problems of slowed reaction time and judgement. Others may recognise potential problems with insurance cover. Options for alternatives should be discussed including the:

- offer of a second opinion
- suggestion of a formal driving assessment or a simulated test available in occupational therapy departments in some hospitals
- use of mobility vouchers to reduce taxi costs (see Appendix F2) (NZ Guidelines 6:29).

**Leaving home**

Factors in patients that lead to an increased risk of institutionalisation are physical dependence, irritability, nocturnal wandering and incontinence. Stress in carers can lead to an increased risk of institutionalisation. Institutionalisation offers the best duration of survival for people with dementia followed by a formal care package at home. Survival in this context means time until death rather than quality of life. Day care for people with dementia can delay institutionalisation.

A patient should not be assessed for optimal home care independently of the carer (Eckles 1998).

**The carer: the ‘second patient’**

Many patients with a dementing illness have a primary carer. Carers may suffer physical, social and financial burdens associated with caring for the patient, as well as depressive disorders that can affect as many as 30% of them. Carers are often called the ‘second patient’ and GPs need to be vigilant about the health of the carer as well as the patient with Alzheimer’s disease. Encourage carers to join Alzheimer’s Australia for education and support. Suggest alternative or respite care arrangements rather than waiting for carers to mention them, and – when appropriate – remind carers that in the later stages of Alzheimer’s disease, a patient’s quality of life may actually improve in a nursing home. This will also allow the carer to spend more quality time with the patient as they will no longer be completely preoccupied with, or exhausted by, caring for the patient (Grey Matters 7:12).

**Epidemiology**

At least 80% of people with dementia are cared for at home, only 10-20% reside in facilities. 75% of dementia carers are female, many of whom are elderly. Within families there is an unspoken hierarchy of obligation to give care in Western society. Primary obligation falls to spouses, followed (in order) by unmarried daughters, married daughters, daughters-in-law, sons and other kin.
**Strain and burden**

Dementia carers have been shown to have poorer physical health status and impaired immune function, as well as higher levels of emotional distress, compared to equivalent samples of carers for other disabled groups. A substantial minority suffers from psychiatric illness, especially depression. There is minimal data on strain among professional carers.

It should be noted that caring for a person with dementia, whilst often stressful, may also be a positive, life-enhancing experience.

**Determinants of poor emotional health in carers**

**Factors related to dementing illness**

Neither severity of cognitive impairment nor duration of dementia seems to be correlated with strain as strongly as behavioural problems of the person with dementia. Particularly stressful are:
- sleep disturbance
- incontinence
- immobility/falls
- repetitive demanding behaviour
- aggression.

‘Negative’ symptoms grind down the carer and produce a build-up of strain over time. These include:
- loss of initiative
- loss of good company/conversation
- the need for constant supervision.

**Relationship factors**

Spouses are generally more stressed than other kin. The quality of the past relationship has a major bearing on strain, with ambivalent, conflictual or highly mutually dependent premorbid relationships predicting high levels of stress.

**Caregiving factors**

Dysfunctional caregiving is characterised by:
- inability to set limits when the cared-for person behaves unreasonably
- inability to leave the cared-for person, even when adequate arrangements are made
- difficulty engaging with outside agencies when help is offered
- marked discrepancy in the cared-for person’s level of functioning between home and other care settings.

**Support**

The relationship between support levels and strain is highly complex and variable. Informal (unpaid) support appears to be protective in some circumstances but scientifically sound evidence for any protective benefit of formal (paid) support services is lacking. There is, however, a great deal of descriptive and anecdotal data indicating support services are helpful in many ways to carers and people with dementia.

**Gender**

Studies have consistently shown that female carers experience, on average, much more strain and morbidity than their male counterparts. This appears to be mainly related to attitudinal factors and differences in coping style.

**What can be done to relieve carer strain and distress?**

Carer surveys have shown carers would like GPs to:
- include them in care planning and decision-making
- provide plain language information about the patient’s condition, prognosis and treatment
- refer to support groups such as carer associations, health care services and respite care providers
- discuss and assess the carer’s own physical and psychosocial health needs
- engage other family members in understanding and sharing care responsibilities
- recognise grief and loss on cessation of caring (Nankervis et al. 2002).

Training programs for carers have been shown both to relieve strain and to delay institutional placement, and are therefore cost-effective. Such programs can include the above and provide a group within which the carer can be encouraged to come to terms with losses involved through expression of feelings.
Despite the dearth of solid empirical evidence for effectiveness of formal support services, demand for these services among carers (especially for respite care) tends to be high; once provided, they tend to be well accepted. Support groups and various other forms of counselling are helpful for some carers, although scientific evidence for this is also scarce.

There is good evidence that institutional placement of the person with dementia usually results in reduction of measured strain levels in the carer (although it may also produce a new set of adjustment difficulties) (NZ Guidelines 6:27).

**Abuse**

People with dementia are at increased risk of abuse. This can be physical, psychological, financial or sexual. The stresses associated with caring for someone with dementia can tax the resources of even the most patient person and this can lead to physical or psychological abuse.

This can occur more readily if the carer is unaware of supports. It should also be recognised that the person with dementia can sometimes be the abuser.

Recognition of situations where abuse is occurring may be difficult and requires awareness of the possibility by health professionals and tactful inquiring about the stresses of caring.

The prospect of financial gain may sometimes be a source of abuse. As one GP said ‘One of the saddest things is watching the family come in like a pack of sharks for the will’ (NZ Guidelines 6:31).
References

Guidelines


These guidelines are ‘most relevant to the management of community-dwelling patients being cared for by family members’.

2. Dementia update. Medicine Today supplement June 2002

This Australian supplement has four articles dealing with the management of behavioural disorders in dementia; diagnosis and management of delirium; diagnosis and management of vascular dementia; and Alzheimer’s disease: a new guide to management in general practice.


This is a good summary, with quite specific conclusions ‘attempting to distil the available evidence and wisdom into statements helpful to primary care physicians’, but they date from the conference in February 1998.


This review is a report of the Quality Standards Subcommittee of the Academy and makes evidence based recommendations for practice. It covers pharmacotherapy for both cognitive and non-cognitive symptoms, educational interventions and other non-pharmacological interventions.

5. The primary care management of dementia North of England Evidence Based Guideline Development Project Department of Primary Care, and Centre for Health Services Research, University of Newcastle upon Tyne, England.

This guideline is a thorough and methodologically rigorous meta-analysis of literature on various aspects of dementia, up to 1996.


A practically orientated guideline/resource document for those caring for people with dementia.


A guide published by Pfizer Pharmaceuticals (makers of Aricept) prepared by international advisers and adapted for use in Australia with input from Australian advisers, now largely superseded by item 2 above.
Other references


Fourth Edn. Washington DC.

Bridges-Webb C. Dementia care in general practice: what can the BEACH survey tell us? Australian 
Family Physician 2002; 31:381-383.

Cat. No. GETP 2. Canberra: Australia Institute of Health and Welfare (General Practice Series No. 2).


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### Appendix A.
Patient health assessments

#### A1. Royal Australian College of General Practitioners (RACGP) assessment form checklist

**People with dementia and behaviours of concern – assessment form**

<table>
<thead>
<tr>
<th>Presenting behaviour and characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>(include duration, frequency, antecedents, consequences)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Physical/psychological health status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exclusion of delirium Considered</td>
</tr>
<tr>
<td>Systems review Reviewed</td>
</tr>
<tr>
<td>Infections</td>
</tr>
<tr>
<td>Respiratory</td>
</tr>
<tr>
<td>Urinary</td>
</tr>
<tr>
<td>Neurological</td>
</tr>
<tr>
<td>Metabolic disorders</td>
</tr>
<tr>
<td>Electrolyte disturbance</td>
</tr>
<tr>
<td>Renal failure</td>
</tr>
<tr>
<td>Hepatic failure</td>
</tr>
<tr>
<td>Vascular disease</td>
</tr>
<tr>
<td>Hypertensive encephalopathy</td>
</tr>
<tr>
<td>Shock</td>
</tr>
<tr>
<td>Hypoxia</td>
</tr>
<tr>
<td>Hypotension</td>
</tr>
<tr>
<td>Cardiac failure</td>
</tr>
<tr>
<td>Carbon monoxide poisoning</td>
</tr>
<tr>
<td>Endocrine disorders</td>
</tr>
<tr>
<td>Hypo- or hyper- thyroidism</td>
</tr>
<tr>
<td>Hypo- or hyper- adrenocorticism</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Drugs</td>
</tr>
<tr>
<td>Alcohol</td>
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<tr>
<td>Withdrawal</td>
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<tr>
<td>Cholinergic drugs</td>
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<tr>
<td>Psychotropics</td>
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<tr>
<td>Cardiac drugs (beta blockers)</td>
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<tr>
<td>Toxins</td>
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<tr>
<td>Heavy metals</td>
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<tr>
<td>Pesticides</td>
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<tr>
<td>Trauma</td>
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<tr>
<td>Head trauma</td>
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<tr>
<td>Subdual haematoma</td>
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<tr>
<td>Heat stroke</td>
</tr>
<tr>
<td>Postoperative</td>
</tr>
<tr>
<td>Systems review Reviewed</td>
</tr>
<tr>
<td>Eyes (spectacles, reduced acuity)</td>
</tr>
<tr>
<td>Medication review</td>
</tr>
<tr>
<td>(drug interactions, compliance, usage or non prescription medication medicines)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mental state</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance (stance, facial expression, disheveled)</td>
</tr>
<tr>
<td>Behaviour (general behaviour apart from focal problem eg early morning awakening)</td>
</tr>
<tr>
<td>Form of speech (thoughts slowed)</td>
</tr>
<tr>
<td>Content of speech (hallucinations sad and hopeless thoughts)</td>
</tr>
<tr>
<td>Mood (anxious, agitated, depressed, unresponsive)</td>
</tr>
<tr>
<td>Diurnal variation in mood</td>
</tr>
<tr>
<td>Awareness</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pre dementia personality, hobbies, occupation, education</th>
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</table>

| Social assessment |
| (support network, physical supports, financial support, lifestyle/cultural issues, social activities) |

| Carer assessment |
| (dementia knowledge, coping skills, stress levels, physical, social supports; including Alzheimer’s Association) |

| Physical environment |
| Reviewed Comment |
| Safety considerations (restraints, wandering, smoking, night, time lighting) |
| Environmental stimulation (level of interest orienting, cues mouse level individual alternation) |
A2. Health Assessment Checklist
A Guide for General Practitioners
Health Assessments - Medicare items 700 to 706

This assessment checklist utilises a series of key questions identified as being useful from the DVA Preventive Care Trial for older persons. However, the information from these questions are to be used in conjunction with clinical assessment. Support for this assessment process is available in a DVA internal publication 'Clinical Supplement - Screening Tools to Support Health Care Planning and Assessment'. The information provided may also be used for the progressive validation of the checklist.

A health assessment means the assessment of a patient’s health and physical, psychological and social function and whether preventative health care and education should be offered to the patient, to improve that patient’s health and physical, psychological and social function.

Who qualifies for an assessment?
All Australians aged 75 and over and Aboriginal and Torres Strait Islanders aged 55 and over.

Assessment Guidelines
The assessment must include keeping a record of the health assessment, signed by the patient and giving the patient a written report about the health assessment, with recommendations about the matters covered by the health assessment.

These items do not apply to in-patients of a hospital or day hospital facility or residents of a nursing home.

For items 704 and 706, a person is of Aboriginal or Torres Strait Islander descent if the person identifies himself or herself as being of that descent.

The annual health assessment should not take the form of a health screening service, in particular the assessment should not include category 5 (diagnostic imaging) services or category 6 (pathology) services. See General Notes 13.3.

The information collection component of the assessment may be rendered ‘on behalf of’ a medical practitioner in accordance with accepted medical practice, acting under the supervision of the medical practitioner. The other components of the health assessment must include a personal attendance by the medical practitioner.

Practitioners should establish a register of their patients seeking annual health assessments and remind registered patients when their next health assessment is due.

DVA health assessment enquiries may be directed to 1300 301 610.

What do I do with the completed form?

- You should provide a copy of the health assessment to the patient/veteran/carer.
- Retain the health assessment in your patient’s record for your future reference and referral resource.
- Consider attaching a copy of the health assessment to referrals to other health care providers.
- Do NOT send this completed Health Assessment Checklist to DVA. Send your claim for payment to the HIC in the usual manner.

<table>
<thead>
<tr>
<th>Name</th>
<th>Date</th>
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Date of birth
/ /

Medicare/DVA No.  

D677 5/09 - P1
### Appendix A. Patient health assessments

1. **Are you living:**
   - [ ] Alone
   - [ ] As a couple
   - [ ] With others

2. **Self Rated Health**
   - Excellent
   - Very Good
   - Good
   - Fair
   - Poor

3. **Current health problems:**

4. **Community services currently provided:**
   - Daycare
   - Respite care
   - HACC - Home Help
   - Community nursing
   - Meals on Wheels
   - Podiatry
   - Physiotherapy
   - Other (please specify)

5. **Vaccinations**
   - Influenza
   - Pneumococcus
   - Tetanus
   - Date

6. **Medications including OTC items** (including OTC & complementary items)

<table>
<thead>
<tr>
<th>Preparation</th>
<th>Strength</th>
<th>Dose</th>
<th>Expiry date</th>
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</table>

Manages own medicine
- [ ] No
- [ ] Yes

Compliance difficult
- [ ] No
- [ ] Yes

Comments

7. **Smoking**
   - Currently smokes
   - [ ] No
   - [ ] Yes

   Wishes to quit
   - [ ] No
   - [ ] Yes

   Comments

8. **Alcohol Consumption**
   - How often do you have a drink containing alcohol?
     - [ ] Never
     - [ ] 2 - 4 times a week
     - [ ] Monthly or less
     - [ ] 5+ times a week
     - [ ] Once a week

   How many standard drinks do you have on a typical day when you are drinking?

   How often do you have 6 or more standard drinks on one occasion?
     - [ ] Never
     - [ ] 2 - 4 times a week
     - [ ] Monthly or less
     - [ ] 5+ times a week
     - [ ] Once a week

9. **Mental Status**
   - What is the year, season, date, day, month?
     - [ ] Score 1 point for each correct answer
   - Where do you live?
     - State, country, town, street number, street name
     - [ ] Score 1 point for each correct answer
   - Repeat 5 objects - house, bus, dog
     - [ ] Score 1 point per word on first trial only
   - Spell world backwards
     - [ ] Score 1 point for each correct letter

   TOTAL score
   - Scale: 14 - 18 indicates no cognitive difficulty

   Comments

10. **Social Support**
    - During the last 4 weeks... Was someone available to help you if you needed and wanted help? For example if you:
      - [ ] felt very nervous, lonely or blue
      - [ ] got sick and had to stay in bed
      - [ ] needed someone to talk to
      - [ ] needed help with daily chores
      - [ ] needed help just taking care of yourself
    - Yes, as much as I wanted
      - [ ] Yes
      - [ ] Yes, quite a bit
      - [ ] Yes, some
      - [ ] Yes, a little
      - [ ] No, not at all

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Appendix A. Patient health assessments

11 Feelings
During the last 4 weeks... How much have you been bothered by emotional problems such as feeling anxious, depressed, irritable or downhearted or blue?

- Not at all [ ] 1
- Slightly [ ] 2
- Moderately [ ] 3
- Quite a bit [ ] 4
- Extremely [ ] 5

12 Nutrition
a. Do you have an illness or condition that made you change the kind and/or amount of food you eat?
   - Yes [ ] 2 No [ ] 1
b. Do you eat at least 3 meals per day?
   - Yes [ ] 0 No [ ] 3
c. Do you eat fruit or vegetables most days?
   - Yes [ ] 0 No [ ] 2
d. Do you eat dairy products most days?
   - Yes [ ] 0 No [ ] 2
e. Do you have 3 or more glasses of beer, wine or spirits almost every day?
   - Yes [ ] 3 No [ ] 0
f. Do you have 6-8 cups of fluids most days?
   - Yes [ ] 0 No [ ] 1
g. Do you have teeth, mouth or swallowing problems that make it hard to eat?
   - Yes [ ] 4 No [ ] 0
h. Do you always have enough money to buy food?
   - Yes [ ] 0 No [ ] 5
i. Do you eat alone most of the time?
   - Yes [ ] 2 No [ ] 0
j. Do you take 3 or more prescribed or over the counter medicines every day?
   - Yes [ ] 3 No [ ] 0
k. Without wanting to, have you lost or gained 5kg in the last 6 months?
   - Yes [ ] 2 No [ ] 0
l. Are you always able to shop, cook and/or feed yourself?
   - Yes [ ] 0 No [ ] 2

TOTAL score

0 - 5 ‘good’, 6 - 20 ‘moderate risk’, 21 - 29 ‘high risk’

Comments

---------------------------------------------------------------

13 Home Safety

a. Can you get up from your lounge chair easily?
   - Yes [ ]  No [ ]
b. Can you get in and out of bed easily and safely?
   - Yes [ ]  No [ ]
c. Can you switch a light on easily from your bed?
   - Yes [ ]  No [ ]
d. Are your loose mats securely fixed to the floor?
   - Yes [ ]  No [ ]
e. Do you slip resistant mats in the bath/bathroom/shower recess?
   - Yes [ ]  No [ ]
f. Can you carry meals easily and safely from the kitchen to your dining area?
   - Yes [ ]  No [ ]
g. Difficulty gripping utensils/handrails?
   - None [ ]
   - A little [ ]
   - A lot [ ]
h. Are the edges of the steps/stairs easily identifiable?
   - Yes [ ]  No [ ]

Comments

---------------------------------------------------------------

14 Mobility

a. Difficulty climbing one flight of stairs?
   - None [ ]
   - A little [ ]
   - A lot [ ]
b. Difficulty bending, kneeling or stooping?
   - None [ ]
   - A little [ ]
   - A lot [ ]
c. Difficulty walking 100 metres?
   - None [ ]
   - A little [ ]
   - A lot [ ]
d. Difficulty bathing or dressing self?
   - None [ ]
   - A little [ ]
   - A lot [ ]
Appendix A. Patient health assessments

15 Feet
a. Problems with one or both feet?
   Yes □ No □

Comments

16 Vision
Acuity (with glasses)

Comments

17 Hearing
a. Whisper test
   Heard □ Not heard □

b. Hearing aid
   N/A □ Adequate □ Poor □

Comments

18 Continence
a. Leaking urine?
   Never □ Sometimes □ Often □

b. Is this related to coughing or sneezing?
   Yes □ No □

c. Faecal incontinence/change of bowel habit?
   Yes □ No □

d. Family history of bowel cancer?
   Yes □ No □

Comments

19 BP/Pulses
a. Blood Pressure □

b. Pulse
   Regular □ Irregular □

Comments

20 Urine analysis
   Clear □

   Glucose □

   Blood/protein □

21 Other areas for examination and/or follow up (eg skin, breast, prostate)

Comments

22 Future plan of action (include agreed action/goals of patient and/or carer)

Comments

23 Patient and Doctor declaration
I agree with this plan
Patient’s signature

Doctor’s signature

24 General Practitioner/Local Medical Officer details
Provider No.

Name

Address

Postcode

Phone No.

□
Appendix B.
Cognitive functioning

B1. Mini-mental state examination

(Folstein et al. 1975)

This test could not be included for copyright reasons. It is available from:
Psychological Assessment Resources Inc (PAR) 16204 North Florida Avenue, Lutz, Florida 33549
Website: www.parinc.com
Email: custserv@parinc.com
B2. GPCOG patient examination

This is a new screening test developed in Australia for use in general practice. It is shorter than the MMSE and incorporates the clock drawing test (Appendix 2.2). Only if the test result is in the doubtful area does it need to include some additional information to be obtained from a suitable informant.

The test is set out on next page.

**Scoring**

Add correct scores from items 2-6:

9 = cognitively intact – no need for informant interview
4 or less = cognitively impaired – no need for informant interview
5-8 = uncertain – needs informant interview

Informant interview score:

No, 3 or less = cognitively impaired

**Appendix B. Cognitive functioning**

**GPCOG Patient Examination**

Unless specified, each question should only be asked once.

**Name and address for subsequent recall test**

1. “I am going to give you a name and address. After I have said it, I want you to repeat it.
   Remember this name and address because I am going to ask you to tell it to me again in a few minutes: John Brown, 42 West Street, Kensington.” (Allow a maximum of 4 attempts but do not score yet)

<table>
<thead>
<tr>
<th>Time Orientation</th>
<th>Correct</th>
<th>Incorrect</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. What is the date? (exact only)</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>

**Clock Drawing** (visuospatial functioning) - use pen with printed circle

3. Please mark in all the numbers to indicate the hours of a clock (correct spacing required)
4. Please mark in hands to show 10 minutes past eleven o’clock (11:10)

<table>
<thead>
<tr>
<th>Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>5. Can you tell me something that happened in the news recently? (recently = in the last week)</td>
</tr>
</tbody>
</table>

**Recall**

6. What was the name and address I asked you to remember?

<table>
<thead>
<tr>
<th>John Brown 42 West (St) Kensington</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐</td>
</tr>
</tbody>
</table>

---

**Scoring guidelines**

**Clock drawing:** For a correct response to question 3, the numbers 12, 3, 6, and 9 should be in the correct quadrants of the circle and the other numbers should be approximately correctly placed. For a correct response to question 4, the hands should be pointing to the 11 and the 2, but do not penalize if the respondent fails to distinguish the long and short hands.

**Information:** Respondents are not required to provide extensive details, as long as they demonstrate awareness of a recent news story. If a general answer is given, such as "war," "a lot of rain," ask for details—if unable to give details, the answer should be scored as incorrect.

---

**GPCOG Informant Interview**

**Ask the informant:** “Compared to a few years ago,

<table>
<thead>
<tr>
<th>Does the patient have more trouble remembering things that have happened recently?</th>
<th>Yes</th>
<th>No</th>
<th>Don’t Know</th>
<th>N/A</th>
</tr>
</thead>
<tbody>
<tr>
<td>I.</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Does he or she have more trouble recalling conversations a few days later?</th>
</tr>
</thead>
<tbody>
<tr>
<td>II.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>When speaking, does the patient have more difficulty in finding the right word or tend to use the wrong words more often?</th>
</tr>
</thead>
<tbody>
<tr>
<td>III.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Is the patient less able to manage money and financial affairs (eg paying bills, budgeting)?</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Is the patient less able to manage his or her medication independently?</th>
</tr>
</thead>
<tbody>
<tr>
<td>V.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Does the patient need more assistance with transport (either private or public)?</th>
</tr>
</thead>
<tbody>
<tr>
<td>VI.</td>
</tr>
</tbody>
</table>
B3. Decision making capacity

C Bridges-Webb, RACGP NSW Projects, Research and Development Unit.

Determination of a patient’s capacity (a word preferred to the more strictly legal one of competency [1]) to make decisions may be an important role of the doctor. This usually applies in one of three situations:

- consent for medical treatment
- giving an advance care directive
- making a will.

It may also apply to other tasks such as managing financial affairs or arranging living circumstances.

Whatever the task, there are a number of important principles to be observed:

- Capacity is task specific, and must be assessed separately for each decision.
- Assessment is best made over time, rather than at only one interview, because determination of consistency of response is important.
- Information from others, with the patient’s consent, is desirable.
- There are no studies which define threshold, and only modest correlation between scores on scales such as MMSE or IADL and capacity to make decisions (2).

Records should be kept as fully as possible, with emphasis on information that explains the basis for the decision.

Factors to be considered

The following factors need to be considered in determining capacity (3):

- **Attention** – can the patient maintain attention for long enough? Maintaining conversation for at least 1 minute is a minimum. Assess using subtraction of serial 7’s from 100 (need 7 correct responses), or ability to count forward by 3’s from 1 (no more than one error in 30 seconds), or counting backwards from 20 (finishing within 20 seconds) (3).

- **Language – comprehension**, by hearing or reading. This can be tested by conversation and/or with suitable simple multi-choice questions given orally and/or in writing.

- **Language – reply**, may be made by the patient in speech or writing, or by gesture, pointing or other understandable means. These forms of communication should not be overlooked when speech or writing is not possible.

- **Memory** – short and long term memory need not be perfect, but should be relevant to the task.

- **Awareness** of the significance of the interview: Does the patient understand who is doing it and why? How does it relate to the patient’s social situation, family, interests, activity? What are the likely consequences?

- **Judgement** – can the patient appreciate outcomes, control impulses? How does what they say compare with what they do? How consistent are their responses? History from others is important for this.

**Consent to medical treatment**

In assessing capacity to consent to medical treatment the following factors must be clear (1):

- what are the options?
- the benefits and risks of each
- the values the patient wants to uphold or goals they wish to reach
- the stability of the decision over time; the consent must be given on at least two different occasions
- the patient must always be included in the decision process to the extent possible
- there must be no coercion or undue pressure from others.

If the patient has not the capacity to consent, then someone other than the treating team members MUST make the decision.

Even in the presence of an advance care directive it is important to ‘try to understand also the present subjective experiences’ of the patient (1).
Appendix B. Cognitive functioning

Advanced care directives
In making an advanced care directive the patient must understand that (4):
- the choices being made are for the future
- it will be used only if the patient has become incapable
- some choices are about future treatment
- some choices are about who will then decide
- the choices made could threaten life
- coma or dementia means that no choice in the future will be possible
- choices may change over time
- directives should be updated and changed if necessary each year
- choices made in the directive override later choices if the patient has become incapable.

A protocol to ensure a patient centred approach for the assessment of competence to complete advance care directives, using two vignettes of hypothetical medical problems and ten questions in a semi-structured interview, has been found to be valid and reliable in one British trial (5).

Making a will
In determining capacity to make a will there are a number of specific requirements (2):
- The patient's lawyer should first be consulted.
- Assessment should occur on two different occasions, the second preferably on the day of executing the will.
- The presence or absence of witnesses to the assessment, and if any, who they should be, should be considered.
- The patient must be free of undue influence, such as from family member or carer.
- The patient must not have delusions or hallucinations that could influence the decisions.

In the assessment the patient must:
- understand the nature and purpose of the interview, and what he/she is doing. Ask them to explain what a will is
- be able to describe the extent and nature of his/her property
- be able to understand and state the claims of potential heirs
- state who is to benefit, in what way each will benefit, and give a sensible explanation of why that benefit to that person is desired.

Corroborative information should be sought, with permission of the patient and his/her lawyer, from medical records, other clinicians, family or others involved with the patient.

References
Appendix C.
Emotional state and behaviour

C1. The geriatric depression scale

Choose the best answer for how you felt over the past week

1. Are you basically satisfied with your life? yes/no N*
2. Have you dropped many of your activities and interests? yes/no Y*
3. Do you feel that your life is empty? yes/no Y*
4. Do you often get bored? yes/no Y*
5. Are you hopeful about the future? yes/no N
6. Are you bothered by thoughts that you just cannot get out of your head? yes/no Y
7. Are you in good spirits most of the time? yes/no N*
8. Are you afraid that something bad is going to happen to you? yes/no Y*
9. Do you feel happy most of the time? yes/no N*
10. Do you often feel helpless? yes/no Y*
11. Do you often get restless and fidgety? yes/no Y
12. Do you prefer to stay at home, rather than go out and do new things? yes/no Y*
13. Do you frequently worry about the future? yes/no Y
14. Do you feel that you have more problems with memory than most? yes/no Y*
15. Do you think it is wonderful to be alive now? yes/no N*
16. Do you often feel downhearted and blue? yes/no Y
17. Do you feel pretty worthless the way you are now? yes/no Y*
18. Do you worry a lot about the past? yes/no Y
19. Do you find life very exciting? yes/no N
20. Is it hard for you to get started on new projects? yes/no Y
21. Do you feel full of energy? yes/no N*
22. Do you feel that your situation is hopeless? yes/no Y*
23. Do you think that most people are better off than you are? yes/no Y*
24. Do you frequently get upset over little things? yes/no Y
25. Do you frequently feel like crying? yes/no Y
26. Do you have trouble concentrating? yes/no Y
27. Do you enjoy getting up in the morning? yes/no N
28. Do you prefer to avoid social gatherings? yes/no Y
29. Is it easy for you to make decisions? yes/no N
30. Is your mind as clear as it used to be? yes/no N

This scale can be self-administered or read in an interview

Count 1 point for each depressive answer (as indicated by N or Y)

Scores
- 0-10: normal range
- 11-20: mild depression
- 21-30: moderate to severe depression

Cut of points: usually 10/11 but 13/14 and 14/15 have also been used

* = items to include in an abbreviated version to reduce problems of fatigue and lack of focus

Scores
- 0-4: normal range
- 5-9: mild depression
- 10-15: moderate to severe depression

Appendix C. Emotional state and behaviour

C2. A screening instrument for depression in later life

EBAS DEP (Even briefer assessment scale for depression)

The eight items of this schedule require raters to make a judgement as to whether the proposition in the middle column is satisfied or not. If a proposition is satisfied then a depressive symptom is present and raters should ring ‘1’ in the right hand column, otherwise ‘0’ should be ringed. Each question in the left-hand column must be asked exactly as printed but follow-up or subsidiary questions may be used to clarify the initial answer until the rater can make a clear judgement as to whether the proposition is satisfied or not. For items which enquire about symptoms over the past month, note that the symptom need not have been present for the entire month nor at the moment of interview, but it should have been a problem for the patient or troubled him/her for some of the past month.

1. Do you worry? In the past month? Admits to worrying in past month. 1 0
2. Have you been sad or depressed in the past month? Has had sad or depressed mood During the past month. 1 0
3. During the past month have you ever felt that life was not worth living? Has felt that life was not worth living at some time during the past month. 1 0
4. How do you feel about your future? What are your hopes for the future? Pessimistic about the future or has empty Expectations (ie nothing to look forward to). 1 0
5. During the past month have you at any time felt you would rather be dead? Has wished to be dead at any time during past month. 1 0
6. Do you enjoy things as much as you used to – say like you did a year ago? Less enjoyment in activities than a year previously. 1 0

If question 6 rated 0, then rate 0 for question 7 and skip to question 8.
If question 6 rated 1, ask question 7

7. Is it because you are depressed or nervous that you don’t enjoy things as much? Loss of enjoyment because of depression/nervousness. 1 0
8. Are you – very happy, fairly happy, not very happy, not happy at all? Not very happy or not happy at all. 1 0

**Total score 8**

A score of 3 or greater indicates the probable presence of a depressive disorder which may need treatment and the patient should be assessed in more detail or referred for psychiatric evaluation.

### Appendix C. Emotional state and behaviour

#### C3. Neuropsychiatric inventory questionnaire (NPI-Q)

**Name of patient:** ____________________________  **Date:** ____________________________

**Informant:** Spouse: __________  Child: __________  Other: __________

Please answer the following questions based on changes that have occurred since the patient first began to experience memory problems. Circle "yes" only if the symptom has been present in the past month. Otherwise, circle "no".

For each item marked "yes":

Rate the severity of the symptom (how it affects the patient):
1 = Mild (noticeable, but not a significant change)
2 = Moderate (significant, but not a dramatic change)
3 = Severe (very marked or prominent; a dramatic change)

Rate the distress you experience because of that symptom (how it affects you):
0 = Not distressing at all
1 = Minimal (slightly distressing, not a problem to cope with)
2 = Mild (not very distressing, generally easy to cope with)
3 = Moderate (fairly distressing, not always easy to cope with)
4 = Severe (very distressing, difficult to cope with)
5 = Extreme or very severe (extremely distressing, unable to cope with)

---

<table>
<thead>
<tr>
<th><strong>Delusions</strong></th>
<th>Does the patient believe that others are stealing from him or her, or planning to harm him or her in some way?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Hallucinations</strong></th>
<th>Does the patient act as if he or she hears voices? Does he or she talk to people who are not there?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Agitation or aggression</strong></th>
<th>Is the patient stubborn and resistive to help from others?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Depression or dysphoria</strong></th>
<th>Does the patient act as if he or she is sad or in low spirits? Does he or she cry?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Anxiety</strong></th>
<th>Does the patient become upset when separated from you? Does he or she have any other signs of nervousness, such as shortness of breath, sighing, being unable to relax, or feeling excessively tense?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Elation or euphoria</strong></th>
<th>Does the patient appear to feel too good or act excessively happy?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Apathy or indifference</strong></th>
<th>Does the patient seem less interested in his or her usual activities and in the activities and plans of others?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Disinhibition</strong></th>
<th>Does the patient seem to act impulsively? For example, does the patient talk to strangers as if he or she knows them, or does the patient say things that may hurt people's feelings?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Irritability or lability</strong></th>
<th>Is the patient impatient and cranky? Does he or she have difficulty coping with delays or waiting for planned activities?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Motor disturbance</strong></th>
<th>Does the patient engage in repetitive activities, such as pacing around the house, handling buttons, wrapping string, or doing other things repeatedly?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Nighttime behaviors</strong></th>
<th>Does the patient awaken you during the night, rise too early in the morning, or take excessive naps during the day?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Appetite and eating</strong></th>
<th>Has the patient lost or gained weight, or had a change in the food he or she likes?</th>
<th>Severity:</th>
<th>Distress:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

---

**FIGURE 3. Neuropsychiatric Inventory Questionnaire.** This tool provides a reliable assessment of behaviors commonly observed in patients with dementia.

### C4. Activities of daily living (ADL)

Name of patient: ___________________________ Date: ___________________________

For each area of function listed below, check the description that applies. (The word ‘assistance’ means supervision, direction or personal assistance.)

<table>
<thead>
<tr>
<th><strong>Bathing</strong> – sponge bath, tub bath, or shower</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Receives no assistance (get in and out of tub by self, if tub is usual means of bathing)</td>
<td>☐</td>
</tr>
<tr>
<td>Receives assistance in bathing only one part of the body (such as back or a leg)</td>
<td>☐</td>
</tr>
<tr>
<td>Receives assistance in bathing than one part of the body (not bathed)</td>
<td>☐</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Dressing</strong> – gets clothes from closets and drawers, including underclothes and outer garments, and uses fasteners (including braces, if worn)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Gets clothes and gets completely dressed without assistance</td>
<td>☐</td>
</tr>
<tr>
<td>Gets clothes and gets dressed without assistance, except for help in trying shoes</td>
<td>☐</td>
</tr>
<tr>
<td>Receives assistance in getting clothes or in getting dressed, or stays partly or completely undressed</td>
<td>☐</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Toileting</strong> – going to the ‘toilet room’ for bowel and urine elimination, cleaning self after elimination and arranging clothes</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Goes to ‘toilet room’, cleans self, and arranges clothes without assistance (may use object for support, such as cane, walker, or wheelchair, and may manage night bedpan or commode, emptying same in morning)</td>
<td>☐</td>
</tr>
<tr>
<td>Receives assistance in going to ‘toilet room’. Or in cleaning self or arranging clothes after elimination, or in use of night bedpan or commode</td>
<td>☐</td>
</tr>
<tr>
<td>Does not go to room termed ‘toilet’ for the elimination process</td>
<td>☐</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Transfer</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Moves in and out of bed, as well as in and out of chair, without assistance (may use object for support, such as cane or walker)</td>
<td>☐</td>
</tr>
<tr>
<td>Moves in and out of bed or chair with assistance</td>
<td>☐</td>
</tr>
<tr>
<td>Does not get out of bed</td>
<td>☐</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Continence</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls urination and bowel movements completely by self</td>
<td>☐</td>
</tr>
<tr>
<td>Has occasional ‘accidents’</td>
<td>☐</td>
</tr>
<tr>
<td>Supervision helps keep urine or bowel control; catheter is used, or person is incontinent.</td>
<td>☐</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Feeding</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Feeds self without assistance</td>
<td>☐</td>
</tr>
<tr>
<td>Feeds self except for cutting meat or buttering bread</td>
<td>☐</td>
</tr>
<tr>
<td>Receives assistance in feeding or is fed partly or completely by using tubes or intravenous fluids</td>
<td>☐</td>
</tr>
</tbody>
</table>

**Scoring**

Score ‘0’ for each box checked in column 1 or 2, and score ‘1’ for each box checked in column 3. **Total score:** ___________________________

SCORING Key: 0 = independent in all 6 functions; 1 to 5 = independent in 1 to 5 function; 6 = dependent in all 6 functions.

**FIGURE 1. Activities of Daily Living scale.** This Instrument evaluates the degree of assistance the patient received during set period (eg the previous week) for each of six basic activities.

*Adapted with permission from Katz S, Ford AB, Moskowitz RW, Jackson BA, Jaffe MW. Studies of illness in the aged. The index of ADL, a standardized measure of biological and psychosocial function. JAMA 1963; 185:914-9.*
Appendix C. Emotional state and behaviour

C5. Instrumental activities of daily living (IADL)

Name of patient: ____________________________ Date: __________

This form may help you assess the functional capabilities of your older patients. The data can be collected by a nurse from the patient or from an informant such as a family member or other caregiver; (I = independent; A = assistance required; D = dependent)

<table>
<thead>
<tr>
<th>Obtained from:</th>
<th>Activity</th>
<th>Guidelines for assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient</td>
<td>Informant</td>
<td></td>
</tr>
<tr>
<td>I A D</td>
<td>I A D</td>
<td>Using telephone</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I = Able to look up numbers, dial telephone, and receive and make calls without help</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A = Able to answer telephone or dial operator in an emergency, but needs special telephone or help in getting numbers and/or dialing</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D = Unable to use telephone</td>
</tr>
<tr>
<td>I A D</td>
<td>I A D</td>
<td>Traveling</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I = Able to drive own car or to travel alone on buses or in taxis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A = Able to travel, but needs someone to travel with</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D = Unable to travel</td>
</tr>
<tr>
<td>I A D</td>
<td>I A D</td>
<td>Shopping</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I = Able to take care of all food and clothes shopping with transportation provided</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A = Able to shop, but needs someone to shop with</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D = Unable to shop</td>
</tr>
<tr>
<td>I A D</td>
<td>I A D</td>
<td>Preparing meals</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I = Able to plan and cook full meals</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A = Able to prepare light foods, but unable to cook full meals alone</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D = Unable to prepare any meals</td>
</tr>
<tr>
<td>I A D</td>
<td>I A D</td>
<td>Housework</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I = Able to do heavy housework (ie scrub floors)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A = Able to do light housework, but needs help with heavy tasks</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D = Unable to do any housework</td>
</tr>
<tr>
<td>I A D</td>
<td>I A D</td>
<td>Taking medicine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I = Able to prepare and take medications in the right dose at the right time</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A = Able to take medications, but needs reminding or someone to prepare them</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D = Unable to take medications</td>
</tr>
<tr>
<td>I A D</td>
<td>I A D</td>
<td>Managing money</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I = Able to manage buying needs (ie write checks, pay bills)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A = Able to manage daily buying needs, but needs help managing checkbook and/or paying bills</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D = Unable to handle money</td>
</tr>
</tbody>
</table>

FIGURE 2. Instrumental Activities of Daily Living scale. This instrument evaluates the patient’s ability to perform the more complex activities that are necessary for optimal independent functioning.

Appendix D.
Caregiver coping

D1. Caregiver burden scale

Caregiver’s name: ___________________________ Date: _______________________

The following questions reflect how people sometimes feel when they are taking care of another person. After each question, circle how often you feel that way: never, rarely, sometimes, frequently, or nearly always. There are no right or wrong answers.

<table>
<thead>
<tr>
<th>Question</th>
<th>Never</th>
<th>Rarely</th>
<th>Sometimes</th>
<th>Frequently</th>
<th>Nearly always</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Do you feel that your relative asks for more help than he or she needs?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2. Do you feel that because of the time you spend with your relative, you do not have enough time for yourself?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3. Do you feel stressed between caring for your relative and trying to meet other responsibilities for your family or work?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4. Do you feel embarrassed over your relative’s behavior?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5. Do you feel angry when you are around your relative?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6. Do you feel that your relative currently affects your relationship with other family members or friends in a negative way?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7. Are you afraid about what the future holds for your relative?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8. Do you feel your relative is dependent on you?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9. Do you feel strained when you are around your relative?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10. Do you feel your health has suffered because of your involvement with your relative?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11. Do you feel that you do not have as much privacy as you would like, because of your relative?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>12. Do you feel that your social life has suffered because you are caring for your relative?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>13. Do you feel uncomfortable about having friends over, because of your relative?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14. Do you feel that your relative seems to expect you to take care of him or her, as if you were the only one he or she could depend on?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15. Do you feel that you do not have enough money to care for your relative, in addition to the rest of your expenses?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>16. Do you feel that you will be unable to take care of your relative much longer?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>17. Do you feel you have lost control of your life since your relative’s illness?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>18. Do you wish you could just leave the care of your relative to someone else?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>19. Do you feel uncertain about what to do about your relative?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>20. Do you feel you should be doing more for your relative?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>21. Do you feel you could do a better job in caring for your relative?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>22. Overall, how burdened do you feel in caring for your relative?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

Total score: ________________________

SCORING KEY:
0 to 20 = little or no burden; 21 to 40 = mild to moderate burden; 41 to 60 = moderate to severe burden; 61 to 88 = severe burden.

FIGURE 4. Caregiver Burden Scale. This self-administered 22-item questionnaire assesses the “experience of burden.”
Appendix E.
Diagnostic criteria

**E1. Diagnostic criteria for dementia of the Alzheimer's type (DSM-IV)**

A. The development of multiple cognitive deficits manifested by both

1. Memory impairment (impaired ability to learn new information or to recall previously learned information).

2. One (or more) of the following cognitive disturbances:
   a. aphasia (language disturbance)
   b. apraxia (impaired ability to carry out motor activities despite intact motor function)
   c. agnosia (failure to recognise or identify objects despite intact sensory function)
   d. disturbance in executive functioning (planning, organising, sequencing, abstracting).

B. The cognitive deficits in criteria A1 and A2 each cause significant impairment in occupational and social functioning, and represent a decline from previous higher levels of functioning.

C. The course is characterised by gradual onset and continuing cognitive decline.

D. The cognitive deficits in criteria A1 and A2 are not due to any of the following:

1. Other CNS conditions that cause progressive deficits in memory.
2. Systematic conditions known to cause dementia (e.g. hypothyroidism).
3. Substance-induced conditions.

E. The deficits do not occur exclusively during the course of a delirium.

F. The disturbance is not better accounted for by another disorder (e.g. major depression).

*(DSMIV, American Psychiatric Association, 1994)*
### Appendix E. Diagnostic criteria

#### E2. Criteria for the clinical diagnosis of Alzheimer’s disease (NINCDS-ADRDA)*

1. The criteria for the clinical diagnosis of PROBABLE Alzheimer’s disease include:
   - dementia established by clinical examination and documented by the Mini-Mental Test, Blessed Dementia Scale or some similar examination and confirmed by neuropsychological tests
   - deficits in two or more areas of cognitive functions
   - no disturbance of consciousness
   - onset between ages 40 and 90, most often after age 65: and absence of systemic disorders or other brain diseases that in and of themselves could account for the progressive deficits in memory and cognition.

2. The diagnosis of PROBABLE Alzheimer’s disease is supported by:
   - progressive deterioration of specific cognitive functions such as language (aphasia), motor skills (apraxia) and perception (agnosia)
   - Impaired activities of daily living and altered patterns of behavior
   - Family history of similar disorders. Particularly if confirmed neuropathologically; and

   Laboratory results of:
   - Normal lumber puncture as evaluated by standard techniques.
   - Normal pattern or non-specific changes in EEG, such as increased slow-wave activity.
   - Evidence of cerebral atrophy on CT with progression documented by serial observation.

3. Other clinical features consistent with the diagnosis of PROBABLE Alzheimer’s disease after exclusion of causes of dementia other than Alzheimer’s disease include:
   - plateaus in the course of progression of the illness
   - associated symptoms of depression, insomnia, incontinence, delusions, illusions, hallucinations, catastrophic verbal emotional or physical outbursts, sexual disorders and weight loss
   - other neurologic abnormalities in some patients especially with more advanced disease and including motor signs such as increased muscle tone myoclonus or gait disorder
   - seizures in advanced disease; and
   - CT normal for age.

4. Features that make the diagnosis of PROBABLE Alzheimer’s disease uncertain or unlikely include:
   - sudden apoplectic onset
   - focal neurologic findings such as hemiparesis sensory loss, visual field deficits, and incoordination early in the course of the illness; and
   - seizures or gait disturbances at the onset or very early in the course of the illness.

5. Clinical diagnosis of POSSIBLE Alzheimer’s disease:
   - may be made on the basis of the dementia syndrome, in the absence of other neurologic psychiatric or systemic disorders sufficient to cause dementia and in the presence of variations in the onset, in the presentation or in the clinical course
   - may be made in the presence of a second systemic or brain disorder sufficient to produce dementia, which is not considered to be the cause of the dementia
   - should be used in research studies when a single, gradually progressive severe cognitive deficit is identified in the absence of other identifiable cause.

6. Criteria for diagnosis of DEFINITE Alzheimer’s disease are:
   - the clinical criteria for probable Alzheimer’s disease and histopathologic evidence obtained from a biopsy or autopsy.

7. Classification of Alzheimer’s disease for research purposes should specify features that may differentiate subtypes of the disorder such as:
   - familial occurrence
   - onset before age of 65
   - presence of trisomy 21
   - coexistence of other relevant condition such as Parkinson’s disease.

*NINCDS – ADRDA: National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer’s Disease and Related Disorders Association

### Appendix F. Alzheimer’s Australia and other services and resources

#### F1. Alzheimer’s Australia list of contacts and help sheets

<table>
<thead>
<tr>
<th>National</th>
<th>Tel.</th>
<th>Fax.</th>
<th>Email</th>
</tr>
</thead>
<tbody>
<tr>
<td>National Secretariat</td>
<td>(02) 6254 4233</td>
<td>(02) 6278 7225</td>
<td><a href="mailto:secretariat@alzheimers.org.au">secretariat@alzheimers.org.au</a></td>
</tr>
<tr>
<td>Alzheimer’s Australia</td>
<td>(02) 6254 2522</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PO Box 108</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HIGGINS ACT 2615</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frewin Centre</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frewin Place</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scullin ACT 2615</td>
<td></td>
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<table>
<thead>
<tr>
<th>Australian Capital Territory (ACT)</th>
<th>Tel.</th>
<th>Fax.</th>
<th>Email</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s Australia</td>
<td>(02) 6254 5544</td>
<td></td>
<td><a href="mailto:alzact@netspeed.com.au">alzact@netspeed.com.au</a></td>
</tr>
<tr>
<td>PO Box 108</td>
<td>(02) 6254 2522</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higgins ACT 2600</td>
<td></td>
<td></td>
<td></td>
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<table>
<thead>
<tr>
<th>New South Wales (NSW)</th>
<th>Tel.</th>
<th>Fax.</th>
<th>Email</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s Australia</td>
<td>(02) 9805 0100</td>
<td></td>
<td><a href="mailto:alz@alznsw.asn.au">alz@alznsw.asn.au</a></td>
</tr>
<tr>
<td>PO Box 6042</td>
<td>(02) 9805 1665</td>
<td></td>
<td></td>
</tr>
<tr>
<td>North Ryde NSW 1670</td>
<td>Website: <a href="http://www.alznsw.asn.au">www.alznsw.asn.au</a></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Email: <a href="mailto:admin@alznsw.asn.au">admin@alznsw.asn.au</a></td>
<td></td>
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<table>
<thead>
<tr>
<th>Northern Territory (NT)</th>
<th>Tel.</th>
<th>Fax.</th>
<th>Email</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s Australia</td>
<td>(08) 8948 5228</td>
<td></td>
<td><a href="mailto:alz@octa4.net.au">alz@octa4.net.au</a></td>
</tr>
<tr>
<td>PO Box 515</td>
<td>(08) 8948 5229</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nightcliff NT 0814</td>
<td>Email: <a href="mailto:ang.alz@octa4.net.au">ang.alz@octa4.net.au</a></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/18 Bauhinia Street</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nightcliff NT 0814</td>
<td></td>
<td></td>
<td></td>
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<table>
<thead>
<tr>
<th>South Australia (SA)</th>
<th>Tel.</th>
<th>Fax.</th>
<th>Email</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s Australia</td>
<td>(08) 8372 2100</td>
<td></td>
<td><a href="mailto:alz@alzheimerssa.asn.au">alz@alzheimerssa.asn.au</a></td>
</tr>
<tr>
<td>27 Conyngham Street</td>
<td>(08) 8338 3390</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glenside SA 5065</td>
<td>Website: <a href="http://www.ozemail.com.au/~alzsa">www.ozemail.com.au/~alzsa</a></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tasmania (TAS)</th>
<th>Tel.</th>
<th>Fax.</th>
<th>Email</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s Australia</td>
<td>(03) 6278 9897</td>
<td></td>
<td>tasonline/tasalz</td>
</tr>
<tr>
<td>PO Box 1606</td>
<td>(03) 6278 9878</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hobart TAS 7001</td>
<td>Website: <a href="http://www.tased.edu.au/">www.tased.edu.au/</a> tasonline/tasalz</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Email: <a href="mailto:Debbie.slater@alztas.asn.au">Debbie.slater@alztas.asn.au</a></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Victoria (VIC)</th>
<th>Tel.</th>
<th>Fax.</th>
<th>Email</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s Australia</td>
<td>(03) 9815 7800</td>
<td></td>
<td><a href="mailto:alz@alzvic.asn.au">alz@alzvic.asn.au</a></td>
</tr>
<tr>
<td>Locked Bag 3002</td>
<td>(03) 9815 7801</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hawthorn VIC 3122</td>
<td>Website: <a href="http://www.alzvic.asn.au">www.alzvic.asn.au</a></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Email: <a href="mailto:alz@alzvic.asn.au">alz@alzvic.asn.au</a></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Western Australia (WA)</th>
<th>Tel.</th>
<th>Fax.</th>
<th>Email</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s Australia</td>
<td>(08) 9388 2800</td>
<td></td>
<td><a href="mailto:alzwa@alzheimers.asn.au">alzwa@alzheimers.asn.au</a></td>
</tr>
<tr>
<td>PO Box 1509</td>
<td>(08) 9388 2739</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subiaco WA 6904</td>
<td>Website: <a href="http://www.alzheimers.asn.au/wa">www.alzheimers.asn.au/wa</a></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Email: <a href="mailto:alzwa@alzheimers.asn.au">alzwa@alzheimers.asn.au</a></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Queensland (Qld)</th>
<th>Tel.</th>
<th>Fax.</th>
<th>Email</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s Australia</td>
<td>(07) 4434 4501</td>
<td></td>
<td><a href="mailto:info@alzgc.asn.au">info@alzgc.asn.au</a></td>
</tr>
<tr>
<td>PO Box 6842</td>
<td>(07) 5535 4186</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gold Coast Mail Center</td>
<td>Website: <a href="http://www.alzheimers.asn.au/wa">www.alzheimers.asn.au/wa</a></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Qld 9726</td>
<td>Email: <a href="mailto:info@alzgc.asn.au">info@alzgc.asn.au</a></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Melbourne (Vic)</th>
<th>Tel.</th>
<th>Fax.</th>
<th>Email</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s Australia</td>
<td>(03) 9815 7800</td>
<td></td>
<td><a href="mailto:alzvic@alzheimers.asn.au">alzvic@alzheimers.asn.au</a></td>
</tr>
<tr>
<td>Locked Bag 3002</td>
<td>(03) 9815 7801</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hawthorn VIC 3122</td>
<td>Website: alzheimers.asn.au</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Email: <a href="mailto:alzvic@alzheimers.asn.au">alzvic@alzheimers.asn.au</a></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### HELPLINE: 1800 639 331

### NATIONAL Website. www.alzheimers.org.au
Appendix F. Alzheimer’s Australia and other services and resources

Help sheets

About Dementia
- What is dementia?
- Diagnosing dementia
- Early planning
- Next steps
- Memory loss
- Progression of dementia
- Research
- Useful resources
- Alzheimer’s disease
- Vascular dementia
- Frontal lobe dementia, including Pick’s disease
- Dementia with Lewy bodies
- Alcohol related dementia
- AIDS related dementia
- Drug treatments and dementia
- Dementia and heredity
- Diagnosis: Informing the person with dementia
- Information for family and friends
- Down syndrome and Alzheimer’s disease

Caring for someone with dementia
- Intimacy and sexual issues
- Continence
- Wandering
- Driving
- Communication
- Eating
- Dressing
- Activities
- Sleeping
- Travelling
- Safety issues
- Hygiene
- Caring for someone who lives alone
- Sundowning
- Nutrition
- Dental care
- Therapies and communication approaches
- Going to hospital
- Later stages of dementia
- Working with doctors
- Pain
- Making the most of Respite Care

Changed behaviours and dementia
- Changed behaviours
- Hallucinations and false ideas
- Depression and dementia
- Changed behaviours: Useful resources
- Changed behaviours: Problem solving
- Agitated behaviours
- Aggressive behaviours
- Anxious behaviours
- Aggressive behaviours
- Disinhibited behaviours

Information for people with dementia
- What is dementia?
- Early planning
- Looking after yourself
- Driving and dementia
- Living alone
- Feelings and adjusting to change
- Keeping involved and active
- Talking about diagnosis
- Talking with your doctor

Residential care and dementia
- Deciding on residential care
- Coping with placement
- What is available and what will it cost?
- Which residential facility?
- Caring partnerships
- Good care in a residential facility
- Accreditation in residential facilities
- Rights and responsibilities
- Further information and support

Taking care of the carers
- Taking care of the carers
- Feelings
- Men and caring
- Useful resources
- Taking a break
- Coping after the death of someone with dementia

The environment and dementia
- At home with dementia
- The kitchen
- The laundry
- The living room
- The bathroom and toilet
- The bedroom
- The building
- Furnishings and décor
- Utilities
- Outside
- New housing and renovations
- Memory aids in the home
- Creating a calming environment
- Pets
- Health and safety for carers
- Useful resources

Young people and dementia
- Information about dementia for young people
- Information about dementia for parents and grandparents
- Young people and dementia: Useful resources

Younger onset dementia
- What is early onset dementia?
- Early planning
- Next steps
- Younger onset dementia: Useful resources
F2. Some relevant services

**Dementia Advisory Services (NSW)**
These locally-based services promote local awareness of dementia, provide information, education and support, and link people with dementia, their carers and families to assessment and support services.

**Aged Care Information Line**
The Aged Care Information Line provides information to consumers about aged and community care services, and can forward Aged Care Information Sheets on issues such as community care packages, respite care and other services for carers and residential aged care. Tel. 1800 500 853.

**Commonwealth Carelink Centre**
Carelink Centres provide information about services such as meal services, personal and nursing care, home help and other health, aged care, and disability services that help elders stay at home and in the community. Carelink is accessible via a national freecall telephone number. Tel. 1800 052 222.

**Aged Care Assessment Team**
Aged Care Assessment Teams (ACATs) are multidisciplinary teams including doctors, nurses, allied health professionals and social workers. Team members can provide an expert assessment of dementia and help to determine the level of care the person will need, and provide information about services such as respite care, community support services and residential care services.

**National Dementia Helpline**
This freecall national telephone service is provided by Alzheimer’s Australia and staffed by professional counsellors and trained volunteers who provide information, support, advice and local referrals. Tel. 1800 639 331.

**Dementia Education and Support Program**
Alzheimer’s Australia also provides education and support programs for people with dementia and their carers. For information, see Alzheimer’s Australia website. [www.alzheimers.org.au](http://www.alzheimers.org.au) or call the National Dementia Helpline 1800 639 331.

**Commonwealth Carer Resource Centre**
Carer Resource Centres provide carers with referral to services and practical information to support them in their caring role. Tel. 1800 242 636.

**Commonwealth Carer Respite Centre**
Carer Respite Centres can arrange emergency respite care and specific dementia respite care for carers who need a break. This service is accessible 24 hours a day via a national freecall number 1800 059 059.

**National Dementia Behaviour Advisory Service**
This is a national telephone advisory service for respite care staff, carers and relatives or friends who provide care and are concerned about the behaviours of people with dementia and any issues around accessing or providing respite. Tel. 1300 366 448.

**Community Support Services**
People with dementia can receive help through services for frail older people such as the Home and Community Care Program, Community Aged Care Packages, day care centres and residential aged care services. These services include dementia monitoring and support services, meal services, personal and nursing care, home help and respite care.

**Residential Aged Care Services**
Some people with dementia will need residential care (in a hostel or nursing home). There are some dementia-specific services that provide specialised residential care for people with more complex needs.
Appendix F. Alzheimer’s Australia and other services and resources

**General information and support for Carers NSW**
Tel. 1800 242 636 (toll free from anywhere in Australia)
Email. contact@carersnsw.asn.au
Website. www.carersnsw.asn.au

**Taxi transport subsidy scheme**
This Scheme operates through the NSW Department of Transport and may be appropriate for patients in the later stages of dementia.
Tel. (02) 9268 2800
Application forms are available by calling:
(02) 9270 6100 or 1800 623 724.

Similar schemes may operate in other states and information can be obtained from the following offices:
Department of Transport Queensland
Tel. (07) 3253 4700
Customer Service
Tel. (07) 3834 2011

Department of Infrastructure Victoria
Tel. (03) 9655 6666

Department of Transport Western Australia
Tel. (08) 9320 9300

Department of Transport Tasmania
Tel. (03) 6233 5201

Transport SA (for South Australia)
Tel. (08) 8343 2222

Department of Transport and Regional Services (for ACT)
Tel. (02) 6274 7111

Department of Transport and Works Road Transport Branch (Northern Territory)
Tel. (08) 8999 5511

**Assessing fitness to drive**

Order free copies on Tel. (02) 9264 7088 or contact the following state offices:

**ACT**
Manager – Licensing Registration
Department of Urban Services
PO Box 582, Dickson ACT 2606
Tel. (02) 6207 7122

**NSW**
Manager – Medical Unit
Driver and Vehicle Administration Section
PO Box K198, Haymarket NSW 1238
Tel. (02) 9218 6888

**NT**
Manager Customer Services
Department of Transport and Works
GPO Box 530, Darwin NT 0801
Tel. (08) 8999 3122
Fax. (08) 8999 3189
email. mvr@nt.gov.au

**QLD**
Executive Director
Land Transport Safety Division
PO Box 673, Fortitude Valley QLD 4006
Tel. (07) 3253 4132

**SA**
Office Manager – Licence Services
Department of Transport
60 Wakefield Street, Adelaide SA 5000
Tel. (08) 8226 7433

**TAS**
Manager – Driver Licensing Policy
Department of Infrastructure Energy and Resources
1 Collins Street, Hobart TAS 7001
Tel. (03) 6233 5389

**VIC**
Medical Review
Registration and Licensing
60 Denmark Street, Kew VIC 3101
Tel. (03) 9854 2666

**WA**
Supervisor Driver Assessment Section
Department of Transport
441 Murray Street, Perth WA 6000
Tel. (08) 9320 9392
F3. Resources for general practitioners (GPs), carers and families

Useful resources for general practitioners

**NSW**

- *A Dementia Care Guide for General Practitioners* (CD ROM) – Educational tool for GPs on various aspects of dementia care in general practice. Completion of the questions within the CD ROM attracts 6 CME points. Available from Central Coast Dementia Advisory Service
  Tel. (02) 4320 3677
  Fax. (02) 4320 2088

**Australia**

- *Dementia: What’s it all about?* (video) – Information on dementia including causes and risk factors, types of dementia, the progression of dementia, the early signs and benefits of early diagnosis and assessment, care management and treatment options, and the latest research. Available from the Rural Health Education Foundation.
  Tel. (02) 6232 5480
  Fax. (02) 6232 5484
  Email. rhef@hcn.net.au
  Website. www.rhef.com.au

**International**

- *A Guide to the Diagnosis and Management of Alzheimer's disease* for GPs, including differentiating AD from other forms of dementia; diagnosing AD in primary care, and AD in primary care: the diagnostic process. Available on the International Psychogeriatric Association website www.ipa-online.org

Resources for carers and families

**NSW**

Produced under the NSW Action Plan on Dementia 1996-2001

- *At Home with Dementia* – A practical manual on how to modify the home environment for a person with dementia. The publication includes principles and steps to problem solving, then a room-by-room description of problems, concerns and possible solutions plus a list of useful products. The information is useful for carers as well as for the range of community workers who provide services to people with dementia living at home.
  Available free of charge from the NSW Department of Ageing, Disability and Home Care (DADHC), Office for Ageing Tel. (02) 8270 2211
  • available on the DADHC website. www.dadhc.nsw.gov.au
  • available on audio cassettes from DADHC
  Tel. (02) 9364 6963
  • the information has been summarised into 16 Helpsheets and has been included in the Help Sheets Resource Set of Alzheimer’s Australia: 1800 639 331.

- *Planning Ahead Kit* – Resources for managing financial, health and lifestyle decisions into the future.
  • available on the DADHC website. www.dadhc.nsw.gov.au
  • available on tape from DADHC
  Tel. (02) 8270 2211.
Depression in the elderly

This week’s Update looks at the symptoms, types and treatment of late-life depression.

Introduction

ABOUT 13% of Australia’s population is aged 65 years or older, and this will increase to about 30% by 2051. Life expectancy has increased, with more Australians reaching late life in good health compared to past generations.

Older people are mostly active, reporting high levels of life satisfaction and fewer pressures and worries than earlier in life, and about two-thirds rate their health as at least good. Depression is not a normal part of ageing.

There is debate about the prevalence of depression in old age. The 2007 National Survey of Mental Health and Wellbeing found a decline in the prevalence of depression from midlife and particularly over the age of 65, where fewer than 3% had a 12-month affective disorder compared with the 16- to 85-year average of 6.2 per cent.

It is unclear whether this accurately reflects the situation, as the methodology used may underestimate mental disorders in older persons, for example, by the exclusion of residents in aged care facilities and hospitalised persons, where depression rates are known to be high. With the latter, psychiatric hospitalisation rates for all types of depression peak in men older than 75 years, while hospitalisation for psychotic depression also peaks in late life.

Depression is also more common in older general practice patients, with a recent Australian survey of more than 22,000 patients aged 60 years or older reporting that 8.2% had clinically significant depression.

Presentations of late-life depression

An important factor that contributes to difficulties in identifying depression in late life is the manner in which it presents.

Although the criteria used to diagnose depression do not vary with age, the way they are applied often requires an understanding of the nuances of symptom interpretation.

Identification of depression in general practice relies a lot on patient self-report as a starting point for diagnosis.

This is still the case in late life, but older patients tend to under-report feelings of depression and may not acknowledge being sad or depressed.

The extent to which this tendency is age-related, or a cohort effect reflecting a generation that was raised during the Great Depression and World War II where stoicism was a virtue, is not known.

As a consequence, non-dysphoric depression is more prominent in late life, and common depressive symptoms such as a loss of interest in life, lack of enjoyment in normal activities, fatigue, insomnia, weight loss, thoughts of death, chronic pain, poor concentration, or impaired memory are incorrectly attributed by the older person to old age, early Alzheimer’s disease or physical ill health.

Too frequently family, friends and doctors see it that way as well, with the result that depression may go undetected and untreated for a long time.

It is this fundamental feature that results in so-called atypical presentations of depression in late life.

An important take-home message for GPs is not to rely solely on self-report of depressive feelings for diagnosis of late-life depression.

Dr Draper is Conjoint Professor in the School of Psychiatry at the University of NSW, and Senior Staff Specialist in the Academic Department for Old Age Psychiatry at Prince of Wales Hospital.
Non-dysphoric presentations

There are three main non-dysphoric presentations of late-life depression:

**CHRONIC UNEXPLAINED PHYSICAL SYMPTOMS**

The older person complains of a range of physical symptoms for which no adequate medical explanation can be found. Common symptoms include dizziness, chronic pain, constipation, anorexia and weight loss.

While symptoms of melancholic major depression are usually apparent, the older person often denies that it could be the problem.

In the extreme, the patient may develop psychotic features and become convinced they have an incurable illness. This is not infrequently found to be a factor contributing to suicidal behaviour, which can include the passive form of food refusal and associated severe weight loss.

**MEMORY IMPAIRMENT**

Depression in late life is often accompanied by memory changes, but sometimes the memory impairment may appear to be the main problem, or, at times, the features of depression are not readily apparent. The older person and/or their family become concerned about dementia, or perhaps put the memory change down to normal ageing and do not seek help.

This type of presentation has historically been described as depressive pseudodementia.

Treatment of the depression usually improves the memory, although sometimes it doesn’t fully recover because depression can be a prodom to dementia.

**BEHAVIOURAL CHANGES**

Behaviours associated with depression in late life can be varied. A common scenario is social withdrawal and agoraphobia, in which the older person becomes homebound with fear of going out after a fall or stroke. The reaction is disproportionate to the physical ailment, and although the basic reaction is an anxiety disorder, many patients have co-morbid depression.

Self-neglect, in which the older person loses interest in their appearance, eating an inadequate diet and performing household chores, can in extreme cases result in the person living in squalor. Alcohol abuse might also occur in this context and can develop for the first time in late life. ‘Accidental’ overdoses of sleeping tablets and situations in which an older person becomes preoccupied with changing their will, giving away personal possessions, talking about death; has persistent feelings of hopelessness; or takes an unprecedented interest in firearms should not only alert friends and family of the possibility of depression, but also the risk of suicide.

Types of late-life depression

Some types of depression are more frequent in late life than in younger age groups. As indicated previously, more severe types of depression (particularly with psychotic features) are more prevalent in older people.

There is evidence from neuroimaging and post-mortem studies that cerebrovascular disease is often a factor underlying the development of first-episode depression in late life, and this often results in ‘vascular depression’.

This is characterised by clinical features of aphyria, psychomotor retardation, reduced depressive feelings, reduced insight and mild frontal executive cognitive deficits – in other words, a non-dysphoric depression.6

The most common type of depression in old age is minor or sub-syndromal depression that is characterised by having fewer depressive symptoms (2-4) than major depression (five or more) for at least two weeks. Many researchers have downplayed its significance, but in recent years good evidence has emerged that minor depression causes considerable disablement, distress and may persist. This is possibly because many older people with minor depressive disorders also have physical health impairment that is contributing to the depression.6

Causes of late-life depression

It is best to consider causal factors in terms of the age at which the older person first had an episode of depression. First-episode depression in late life is more likely to bear a relationship to physical health problems, although there are genetic factors (through predisposition to cerebrovascular disease).

Other factors that play a role include loss events (e.g. death of spouse), social isolation and loneliness, medications, alcohol misuse, prodomal dementia, personality factors and adverse early life experiences.

Recurrent depression, where there have been previous episodes earlier in life, such as post-partum depression, is more likely to have genetic, personality and adverse early life experience causal factors, although recent health and psychosocial issues may also be relevant.

A cognitively intact older person in good physical health has a relatively low risk of depression.8

**PHYSICAL HEALTH**

There is a complex relationship between physical illness, disability and depression.

Many physical illnesses cause depression through a variety of biological mechanisms, including structural damage in the brain due to disorders such as cerebrovascular disease, Alzheimer’s disease and Parkinson’s disease, and neuroendocrine disturbances such as hypothyroidism. This may occur in a person where no psychological reason for depression is apparent.

Depression may be present long before other symptoms or signs of the physical illness can be established. Other types of physical illnesses that can cause depression in old age include occult cancer, vitamin deficiencies, anaemia and infections. Therefore, it is essential for any person who becomes depressed for the first time in late life to have a thorough medical evaluation.

The diagnosis of a serious illness such as cancer or dementia may be a precipitant to an adjustment reaction that, at times, progresses to become a depressive illness, particularly in vulnerable individuals. There is also evidence that suicide risk is increased in the three months after a diagnosis of cancer or dementia.

Many physical illnesses result in permanent disabilities. Almost 50% of older people are restricted in at least one specific activity, with more than 20% having a profound or severe core activity restriction that usually affects their mobility and often requires assistance with self-care.2

Chronic pain is perhaps the most important factor that leads to depression and suicide. It is the handicap of being unable to perform a normal social role that appears to be linked with pervasive depression and is likely to be one of the main reasons that depression is more common in residential aged care facilities. Enforced dependency may cause a loss of dignity, a sense of being a burden on others and a fear of institutionalisation. In many circumstances, depression may be inappropriately assessed as a normal psychological reaction to the situation and left untreated.

Medications that are required to treat many of these problems can also cause or exacerbate depression, particularly anti-hypertensives, steroids, analgesics, benzodiazepines and antipsychotics.

**SOCIAL ISOLATION AND LONELINESS**

More than a third of older women, often widows, live alone. People who have never married may have relationships with nieces and nephews that are more tenuous than those between parents and children. Sometimes, a long history of family history may have alienated the older person from earlier relationships. Older migrants with poor English skills are particularly vulnerable to isolation.

Social isolation, in combination with physical disablement, may result in loneliness and demoralisation, with depression a frequent accompaniment. Sometimes there is overuse of alcohol to...
Consequences of untreated late-life depression

Untreated depression has many adverse effects on older people. These include psychological suffering; difficulty coping with chronic health problems; overdose of medication; substance abuse (alcohol and benzodiazepines); premature retirement; interruption and permanent loss of functional roles in the family and society; alienation of family and friends; overuse of social/medical services; reduced self-care and malnutrition; and premature mortality (including from suicidal behaviour).

Treatment of late-life depression

An important part of the evaluation of depression in late life is the assessment of suicide risk. Some tips on how to approach this are listed in Table 1.

High-suicide-risk patients include those who spontaneously express suicidal ideas, state intent to attempt suicide, have planned suicide, or express severe hopelessness about their situation. Such patients require urgent psychiatric review.

It is also important to exclude physical health factors, and, particularly with first-episode major depression, a similar workup as that used for the investigation of dementia is warranted.

Late-life depression treatments are similar to those used in earlier life, but may be applied differently. The effectiveness of treatment does not seem to be influenced by age. It is important to minimise the use of benzodiazepines for insomnia and anxiety, as these tend to provide only temporary symptom relief without addressing the actual depression. Suicide risk might also be increased with their use.

In less severe types of depression (minor depression or non-melancholic major depression), psychological treatments including problem-solving therapy and cognitive-behaviour therapy should be the first-line approach. Therapists need to take into account the limitations imposed by poor hearing, poor eyesight and physical discomfort. Other helpful treatments for depression include social activities, physical exercise (especially resistance training) and music therapy.

For more severe depression (melancholic major depression, psychotic depression), antidepressants are usually required, as well as specialist input.

SSRIs (see Table 2) are the first-line antidepressants due to their better tolerability when compared with other antidepressants. Of the SSRIs, sertraline, escitalopram and citalopram are the agents of choice because they are less likely to cause adverse effects due to pharmacokinetic drug interactions caused by an inhibitory effect of the SSRIs on the hepatic cytochrome P450 metabolic system. To minimise adverse effects, SSRIs should be started at half strength for a week before increasing to full strength.

There is some evidence that SSRIs are less effective than dual-action antidepressants such as venlafaxine, mirtazapine or the tricyclic antidepressant nortriptyline in the treatment of psychotic and melancholic depression, and these agents are often used first-line in this circumstance. Atypical antipsychotics may be used to augment treatment in the treatment of psychotic depression.

Electroconvulsive therapy (ECT) is a useful treatment in melancholic and psychotic depression which has failed to respond to medication. Extra precautions need to be taken with the anaesthetics, but older people tolerate the treatment well.

Prognosis

Antidepressant medication may take longer to work in older people, so trials of at least 6-8 weeks are required before being abandoned due to lack of effect.

Treatment resistance is more likely when there is physical and psychological co-morbidity. It is often best to use a different type of antidepressant for the second trial, although there is evidence that sequential SSRIs might work. It is important to convey a sense of optimism to the patient throughout treatment.

Depression in late life is a recurrent disorder. The prognosis of major depression that has responded to antidepressants is improved by maintaining the patient for at least two years on the dose of antidepressant that achieved the response. After ECT, a maintenance antidepressant and/or lithium is essential.

Psychosocial programs to improve quality of life are also important. Early relapse tends to occur if there is only a partial response to initial treatment, when there is co-morbidity, with more severe depression, and with premature discontinuation of antidepressants.

In conclusion, while there have been no major advances in the treatment of late-life depression in recent years, it is clear that gains can be made through better application of current knowledge.

The patient handout, ‘Depression in older people’, references and further reading are available at medicalobserver.com.au.

Table 2: Main antidepressants used in late life

<table>
<thead>
<tr>
<th>Antidepressant</th>
<th>Starting dose</th>
<th>Daily dose</th>
<th>Major side-effects and warnings</th>
</tr>
</thead>
<tbody>
<tr>
<td>SSRIs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sertraline</td>
<td>25 mg</td>
<td>25-100 mg</td>
<td>Nausea, vomiting, diarrhoea, agitation, anxiety, insomnia, hyponatraemia – monitor sodium levels</td>
</tr>
<tr>
<td>Citalopram</td>
<td>10 mg</td>
<td>10-40 mg</td>
<td></td>
</tr>
<tr>
<td>Escitalopram</td>
<td>5 mg</td>
<td>5-10 mg</td>
<td></td>
</tr>
<tr>
<td>Tricyclic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nortriptyline</td>
<td>25 mg</td>
<td>25-100 mg</td>
<td>Confusion, sedation, cardiac arrhythmia, constipation, dry mouth, falls, orthostatic hypotension, prostatism</td>
</tr>
<tr>
<td>Venlafaxine</td>
<td>37.5-75 mg</td>
<td>75-300 mg</td>
<td>Nausea, vomiting, diarrhoea, falls, agitation, insomnia, hypertension, dizziness, dry mouth, sweating, hyponatraemia – monitor serum sodium</td>
</tr>
<tr>
<td>Mirtazapine</td>
<td>15 mg</td>
<td>15-60 mg</td>
<td>Sedation, increased appetite, weight gain</td>
</tr>
</tbody>
</table>

KEY POINTS

- Depression is not a normal part of ageing.
- GPs should not rely solely on self-report of depressive feelings for diagnosis of late-life depression.
- The three main non-dysphoric presentations of late-life depression are chronic unexplained physical symptoms, memory impairment and behavioural changes.
- The most common type of depression in old age is minor or sub-syndromal depression, which can cause considerable disablement and distress, and may persist.
- The causes of late-life depression include loss events (e.g. death of spouse), social isolation and loneliness, medications, alcohol misuse, prodromal dementia, personality factors and adverse early life experiences.
- Late-life depression treatments are similar to those used in earlier life, but may be applied differently; assessing suicide risk is a crucial part of the evaluation of depression.
Child/Adolescent Disorders


professional and other middle-class parents performing better than children of skilled working-class parents and much better than children of unskilled working-class parents. However, behavioural and emotional disorders are found to be less strongly related to these measures of social class (Meltzer et al. 2003).

About a third of UK and about a half of US mothers of pre-school children go out to work. Whether a mother works or not has a major influence on the life of her children (Graham 1990). Except possibly in the first year of life when there may be some drawbacks, there is little evidence that maternal employment is, in itself, a significant advantage or disadvantage for children as far as behavioural or cognitive development is concerned. Of greater importance is the quality of substitute care available and whether the mother feels comfortable and satisfied with her decision about work. The quality of mother–child attachment is closely related to whether a mother is satisfied with her employment status whatever decision she has made about work.

1.4 Classification and prevalence of psychiatric disorders

The grouping of cases according to their distinguishing features is called classification. The defining characteristics of disorders are specified in classification systems such as the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (American Psychiatric Association 1994) or the International Classification of Diseases (ICD-10) (World Health Organization 1992). The DSM system is used particularly in the USA, whereas the ICD is used in much of the rest of the world. The USA is under treaty obligation to maintain a terminology which is compatible with the ICD. Therefore the newest editions of both systems (DSM-IV and ICD-10) are compatible (Table 1.1).

Both systems allow the classification of different facets of a condition on five axes that differ somewhat between the two systems. The official ICD-10, which contains the essential core, does not describe multiple axes. However, it was designed in such a way that it is possible to put the section on psychiatric conditions in a multiaxial format (Rutter 1989).

<table>
<thead>
<tr>
<th>Axis no.</th>
<th>ICD-10 (modified)</th>
<th>DSM-IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Behavioural and emotional disorders with onset usually occurring in childhood and adolescence</td>
<td>Clinical</td>
</tr>
<tr>
<td>2</td>
<td>Developmental disorders</td>
<td>Personality disorders</td>
</tr>
<tr>
<td>3</td>
<td>Intellectual level</td>
<td>Mental retardation</td>
</tr>
<tr>
<td>4</td>
<td>Medical conditions</td>
<td>General medical conditions</td>
</tr>
<tr>
<td>5</td>
<td>Associated abnormal psychosocial situations</td>
<td>Psychosocial and environmental problems</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Global assessment of functioning</td>
</tr>
</tbody>
</table>

an children of skilled working-class parents. This strongly related to children go out to the life of her children may be some draw- significant advantage in her employment status whether the mother quality of mother-child relationship employment status

is called classification. on systems such as the American Psychiatric CD-10 (World Health JSA, whereas the ICD obligation to maintain newest editions of both lition on five axes that each contains the essen- in such a way that it is format (Rutter 1989).

The official DSM-IV describes which areas of the child's functioning should be coded on each axis. There are axes for clinical psychiatric syndromes, developmental disorders, physical disorders, psychosocial and environmental problems, and the child's level of adaptive functioning. On each axis there must be a coding in all cases. There is also the provision of a "no abnormality" code. Multiple codings per axis are allowed.

The greatest difference between the two systems is the way in which the diagnostic criteria are specified. The ICD employs descriptions of disorders supplemented by global guidelines which read like a clinical text and offers a comprehensive picture of the core clinical concept, whereas the DSM offers operationalized criteria which must be met before the diagnosis can be made. The ICD system uses a prototypical approach in which the clinician decides whether or not the child's problems fit the conceptual picture. This approach involves decisions which are to some degree arbitrary, but it should be realized that many of the DSM-IV diagnostic criteria have also been determined through arbitrary decisions.

The advantage of the use of classification systems is comparability of an individual's problems with those diagnosed earlier by the same clinician or with those diagnosed by others. The coding of ICD or DSM axes also has the advantage that information can readily be stored and communicated.

This can be useful for clinicians as well as for policy makers. However, classification should be incorporated in the final diagnosis in the broader sense including possible aetiological, consequential, and background features in addition to the description of the disorder in ICD or DSM terms.

### 1.4.1 Classification of psychiatric syndromes

In both the classifications mentioned above, the first axis concerns psychiatric disorders. This is the axis most likely to be employed by physicians. Most disorders can be classified in the scheme shown in Table 1.2, which is an abbreviation of the two most relevant sections of ICD-10. Each of these categories has, to some degree, been validated, i.e. there is evidence available that the type of disorder mentioned has a characteristic presentation, aetiology, and course.

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>F90</td>
<td>Hyperkinetic disorder</td>
</tr>
<tr>
<td>0</td>
<td>Disorder of activity and attention</td>
</tr>
<tr>
<td>1</td>
<td>Hyperkinetic conduct disorder</td>
</tr>
<tr>
<td>F91</td>
<td>Conduct disorders</td>
</tr>
<tr>
<td>.0</td>
<td>Conduct disorder confined to the family context</td>
</tr>
<tr>
<td>.1</td>
<td>Unsocialized conduct disorder</td>
</tr>
<tr>
<td>.2</td>
<td>Socialized conduct disorder</td>
</tr>
<tr>
<td>.3</td>
<td>Oppositional defiant disorder</td>
</tr>
</tbody>
</table>

(Continued)
<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>F92</td>
<td>Mixed disorders of conduct and emotions</td>
</tr>
<tr>
<td>.0</td>
<td>Depressive conduct disorder</td>
</tr>
<tr>
<td>F93</td>
<td>Emotional disorders with onset specific to childhood</td>
</tr>
<tr>
<td>.0</td>
<td>Separation anxiety disorder</td>
</tr>
<tr>
<td>.1</td>
<td>Phobic disorder of childhood</td>
</tr>
<tr>
<td>.2</td>
<td>Social sensitivity disorder</td>
</tr>
<tr>
<td>.3</td>
<td>Sibling rivalry disorder</td>
</tr>
<tr>
<td>F94</td>
<td>Disorders of social functioning with onset specific to childhood or adolescence</td>
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<td>Elective mutism</td>
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<tr>
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<td>Reactive attachment disorder of childhood</td>
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<td>.2</td>
<td>Disinhibition attachment disorder of childhood</td>
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<td>F95</td>
<td>Tic disorders</td>
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<td>Transient tic disorder</td>
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<td>Chronic motor or vocal tic disorder</td>
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<td>.2</td>
<td>Combined vocal and multiple motor tic (Tourette syndrome)</td>
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<td>F98</td>
<td>Other emotional and behavioural disorders with onset usually occurring during childhood</td>
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<td>Enuresis</td>
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<td>Encopresis</td>
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<td>.2</td>
<td>Feeding disorder of infancy or childhood</td>
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<td>Stereotyped movement disorder</td>
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<td>Cluttering</td>
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<td>F80–89 Developmental disorders (abbreviated)</td>
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<td>Acquired aphasia with epilepsy</td>
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<td>Specific developmental disorders of scholastic skills</td>
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<td>Specific spelling disorder</td>
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<td>.3</td>
<td>Mixed disorder of scholastic skills</td>
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<td>Specific developmental disorders of motor function</td>
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<td>F83</td>
<td>Mixed specific developmental disorders</td>
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Table 1.2 (Continued) Comparison of the ICD-10 and DSM-IV classifications

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<tr>
<td>0</td>
<td>Childhood autism</td>
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<td>1</td>
<td>Atypical autism</td>
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<td>Rett syndrome</td>
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<td>3</td>
<td>Other childhood disintegrative disorders</td>
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<td>Overactive disorder associated with mental retardation and stereotyped movements</td>
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<tr>
<td>5</td>
<td>Asperger syndrome</td>
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</table>

1.4.2 Drawbacks of existing classification schemes

There are various problems with the existing schemes which make them less than satisfactory (Graham 1982) and indeed discourage some practitioners from using them at all.

- They necessarily involve a judgement of the nature of the problem "within the child". For many practitioners the essential nature of the disturbance is seen elsewhere—within the family, or within the interaction between child and family members, or between the child and wider society.
- The existing diagnostic categories, although reasonably well validated, provide only a very rough guide to aetiology, treatment, and prognosis. They generally need to be amplified by a formulation giving a more extended account of the child's condition and background features before action can be taken.
- Many situations of psychiatric concern frequently dealt with by family practitioners and paediatricians cannot readily be categorized within these classification systems. Non-accidental injury and monosymptomatic abdominal pain of psychogenic or uncertain origin are examples.
- The positioning of some categories is occasionally arbitrary and might be misleading. For example, the introductory section to Developmental Disorders in ICD-10 defines such disorders in terms of their early onset, delay in functions strongly related to biological maturation, and steady course, yet enuresis, which often satisfies all these criteria, is classified as a behavioural or emotional disorder. The problem is that some symptoms, like enuresis and perhaps overactivity, may occur either as developmental disorders or as signs of emotional disorder, and consequently their place in the classification is inevitably somewhat arbitrary.
- Diagnostic classifications are not defined in terms of specific assessment procedures. Thus the diagnostician has to decide whether or not the diagnosis is based on information from parents, child, psychological testing, teachers, or any combination of these. It follows that variations in these assessment procedures will inevitably lead to increased variation in diagnoses.
Nevertheless, the framework of these classification systems in child psychiatry does represent a real advance on previous attempts, and the organization of much of this book is based upon them.

1.4.3 Prevalence of psychiatric disorders

Epidemiology is concerned with the study of the distribution and determinants of disease frequency in human populations (Fletcher and Fletcher 2005). Information about the incidence (number of new cases in a defined period of time) and prevalence (number of cases in the population over a defined period of time) of child psychiatric disorders has accumulated rapidly over the last few decades. Prevalence studies provide helpful information on clinical service needs. Epidemiological studies of background factors and rates of disorder in different populations can indicate possible causes of disorders and thus rational prevention strategies.

The design and methods used in an early study of the prevalence of disorders on the Isle of Wight has set the pattern for many subsequent studies (Rutter et al. 1970a; Rutter 1989). The entire population of 10- and 11-year-olds was screened using teacher and parent behaviour questionnaires. On the basis of their scores on the questionnaires, children were selected as at risk for psychiatric disorder and, together with a comparison group, were seen for individual semistructured interviews and psychological testing. Parents and teachers were also interviewed.

An overview of 49 studies, involving over 240,000 children in many different countries, in which the prevalence of the overall level of psychiatric disorder had been determined, revealed that the median prevalence over a period of a year was 12 per cent (Verhulst 1995). Another overview including a few more recent studies showed median prevalence estimates of 8 per cent for pre-schoolers, 12 per cent for preadolescents, and 15 per cent for adolescents (R.E. Roberts et al. 1998). Different researchers use different methods to calculate rates of disorder. Those who calculate simply on the basis of symptom frequency arrive at high rates, sometimes as high as 49 per cent (Bird et al. 1988). Those who consider a disorder to be present only when there is a significant degree of social impairment find lower rates, such as in a Dutch national sample (7.9 per cent). This approach is considered to be much more typical of rates of actual psychological disturbance in children and young people (Verhulst et al. 1997).

Information about background factors and prevalence of different disorders is provided in the relevant chapters of this book. In this section we limit discussion to factors related to overall prevalence.

Age

Measuring rates of disorders in babies and infants is problematic, though attempts have been made at classification (National Center for Clinical Infant Programs 1994). Around 10–15 per cent of older pre-school children show clinically significant psychiatric disorders (Campbell 1995). Rates remain relatively steady during the rest of childhood and
adolescence, although there are suggestions of a rise during adolescence. However, types of disorder found vary considerably during this age period. Most so-called externalizing disorders (those characterized by aggressive and other types of antisocial behaviours such as stealing and lying) are stable during childhood and then decline after early adolescence, with the exceptions of behaviours that involve legal violations, such as truancy, substance abuse, and other rule-breaking behaviours, which tend to increase during adolescence (Bongers et al. 2003). Moffitt (1993) has hypothesized two developmental trajectories of antisocial behaviours: the life-course persistent pattern consisting of antisocial behavior spanning childhood and adolescence into adulthood, and the adolescence-limited pattern with antisocial behavior which usually starts in early adolescence and remits in late adolescence. So-called internalizing disorders (those involving depression and anxiety in particular) rise slowly during childhood but much more sharply during and after puberty, especially in females (Crijnen et al. 1997).

Social factors
Using relatively weak indicators of social advantage and disadvantage, such as social class as measured by parental occupation, there is only a slight association with overall prevalence. However, with stronger measures of social variables, including parental education, income, and housing conditions, the associations become much greater. This is particularly the case for conduct disorders and is much less marked for internalizing disorders. Disadvantaged neighbourhoods also have a negative impact on behavioural and emotional functioning of children, even when parental socio-economic status is taken into account (Schneiders et al. 2003).

Gender
Prepubertal boys are much more frequently referred to child psychiatric clinics than are girls. This is because of their much higher rate of conduct disorders. In contrast, during adolescence the proportion of girls referred increases because of their greater tendency to develop eating, depressive, and anxiety disorders.

Urbanization
Early studies reported higher rates of disorder in children living in cities than in those living rurally (Rutter et al. 1975). These findings have been generally confirmed, but have not been universally found (e.g. Achenbach et al. 1991). They are thought to be related to the increased stress involved in city living, with higher levels of social and family disorganization. However, rural poverty and social isolation are now being increasingly recognized as a potent cause of psychological difficulty which often goes unrecognized because of the scattered distribution of dwellings and individuals and the centralized and often distant nature of clinical services.

Ethnicity
Great caution is needed in comparing rates of disorder in different ethnic groups. Concepts of acceptable and unacceptable behaviour and ways of showing distress vary.
considerably. Generally, few differences have been found between ethnic groups (Earls and Richman 1980; Bird et al. 1989; Achenbach et al. 1991). However, in the UK, rates of disorder may be somewhat lower in Asian minorities (Kallarackal and Herbert 1976). This does not mean that referral rates are similar. For example, it is common for referred children to come much less frequently from minority than majority indigenous groups. Furthermore, the nature of how psychological problems present in young people may determine the advice route sought, with apparent physical symptoms prompting referral to hospital paediatricians, and classroom/public misbehaviour resulting in educational and social service involvement in the first instance (Daryanani et al. 2001).

Migrant groups
Immigrant children are often under special stress because of disrupted family ties, racism, stressors relating to the reasons for migration, social marginalization, financial hardship, and frequent poor living circumstances. On the other hand, community support and family cohesion can often be greater than in the receiving society generally. Some studies have shown higher rates of overall disorder in these groups. A study of Turkish migrant children living in The Netherlands (Bengi-Arslan et al. 1997) revealed higher rates of parent-rated disorder than in the indigenous population. This might have reflected higher standards of behaviour in the migrant parents or a true difference in prevalence. Parent-reported and self-reported problem scores for Moroccan youths were similar or even lower than scores obtained for Turkish youths and for Dutch youths in The Netherlands (Stevens et al. 2003). However, Dutch teachers reported substantially higher levels of externalizing problems for Moroccan youths than for Turkish and Dutch youths. Such findings may help clinicians in starting to adapt their interpretations of diagnostic findings for children from different cultures.

Secular trends
It is widely believed that rates of psychiatric disorder in children and young people increased considerably in the second half of the twentieth century. The evidence for changes over time in rates of different disorders in European countries has been reviewed by Rutter and Smith (1995). There is indeed evidence for an increase in some disorders such as depression and conduct disorder. However, some have not found any changes at all. A Netherlands study using exactly the same methods 10 years apart in samples of children aged 4–16 years found very small and inconsistent changes (Verhulst et al. 1997). Conversely, Collishaw et al. (2004) found that rates of antisocial disorder in 15–16-year-olds had doubled in the UK between 1974 and 1999.

Family circumstances
Unsurprisingly, family circumstances are strongly associated with rates of child psychiatric disorder. Disharmonious family relationships are those most significantly linked. The fact that rates of disorder are higher in children from families broken by separation and divorce, and are not higher in families broken by parental death, reflects this fact.
There are other family risk factors (variables that increase vulnerability to disorder). These include parental or sibling physical and mental illness. Child-rearing practices that involve inconsistent or harsh physical punishment, critical or hostile attitudes of parents towards their children, lack of affection, and poor parental supervision have also been shown to increase overall risk of disorder.

Factors within the child

Different disorders vary in which personal factors increase risk of occurrence, and this issue is discussed in relevant sections. However, genetic vulnerability, often shown in the early years by adverse temperamental characteristics, is often relevant. Chronic physical disability (especially if this arises from brain dysfunction) and educational failure are other risk factors. Intelligence is also relevant with brighter children showing lower rates of overall disorder. Children with mild intellectual disability have a slightly raised risk of disorder, but those with more severe intellectual disability have much higher rates.

Life events

Life events that have an adverse impact on a child generally increase the risk of disorder. Thus groups of children living in disadvantaged circumstances may be expected to have higher rates (Goodyer 1990). High rates of loss of or separation from parents, other loved relatives, friends, or pets, as occurs in areas plagued by natural disasters or armed conflict, will increase overall rates of disorder.

1.5 Assessment and diagnosis of psychiatric disorders

The specialist skills of child and adolescent psychiatrists are necessary for complex problems. However, most appraisals of behaviour and emotional disorders are made by non-psychiatrists—family practitioners, nurses, psychologists, educationalists, social workers, and other professional groups. This section aims to provide guidance to all professionals carrying out such assessments.

The aims of psychiatric assessment of a child with psychosocial problems or physical symptoms having psychosocial implications are to:

- appraise the nature and severity of the problem
- identify likely causes, be they social, familial, individual, physical, genetic or, as is commonly the case, a combination of these
- plan with child and family members a form of effective management.

Most psychiatric appraisals occur because a family seeks help with a problem in their child. Increasingly, psychiatrists and other professionals are requested to assess for other reasons, for example as part of a statement of a child’s special educational needs, to assist a juvenile court in coming to a decision concerning a child charged with an offence, or as part of a child protection investigation.

A secondary but important set of aims relates to the need to communicate sympathy and support for the child and other family members, to convey understanding of their problems, and to provide meaning and significance for their predicament.
Hyperactivity and attention disorders of childhood
Second edition

Edited by
Seija Sandberg
Royal Free and University College Medical School, London
Historical development

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Introduction

The constellation of overactivity, poor impulse control and inattention has been given a variety of diagnostic labels over the years. Although at present attention deficit hyperactivity disorder and hyperkinetic disorder are popular diagnoses, they are relative newcomers in the diagnostic classification of child psychiatry. For example, even the latest (1957) edition of the child psychiatry textbook by Kanner contained no reference to hyperactivity as a diagnostic entity. The same applied to the 1969 edition of a widely used text on child psychology by Johnson and Medinimus (1969), with even the term ‘attention span’ only mentioned on two of the total of 657 pages. A well-regarded textbook on experimental child psychology (Reese and Lipsitt, 1970) did include a section on attention processes, but without any reference to attention deficit hyperactivity disorder. Yet evidence of attention psychology going back at least to the latter half of the nineteenth century has been well documented (James, 1890; Spearman, 1937). On the European continent, however, the condition was recognized and referred to as ‘hyperkinetic disorder’ in Hoff’s (1956) major textbook of general psychiatry. So, whilst hyperactivity tends to be thought of as a particularly American phenomenon, the history of the use of the term tells us otherwise. Though new as a diagnostic entity, hyperactive behaviour in children has been detected and treated for much longer. Indeed, the diagnosis of ‘dextro-amphetamine response disorder’ was in common use in the former German Democratic Republic (DDR: Göllnitz, 1981).

The current conceptualization of the disorder represents a stage in a complex and varying developmental history, and therefore in order to appreciate our present perspective it is important to consider the chronological course in the unfolding of the concept itself. In this chapter, an overview of the history of hyperactivity and attention disorders will be presented, especially as they have appeared in the western texts. Relatively more attention will also be given to publications up to the 1960s, after which time studies on the condition began to
mushroom. This revised version contains some modifications and additions to the text of the first edition.

References to behavioural disturbance in childhood of a similar nature to that seen in hyperactivity disorders can be found in the writings of Hoffmann (1845), Maudsley (1867), Clouston (1899), Ireland (1877) and others from the middle of the 1800s. However, the earliest clear descriptions of the disorder are those by Still and Tredgold in the early 1900s. Their work will be discussed and examined in the context of the prevailing social and scientific climate. Both authors presented their analysis of the behavioural characteristics of a relatively small group of children, some of whom bear close resemblance to the hyperactive children of today. Still (1902) attributed such behaviour to a 'defect of moral control', believing it to be a biological defect, which was inherited or resulted from some pre- or postnatal injury. His ideas about causation are best understood in the context of the widespread support given at that time to social Darwinism.

The theory of damage occurring in the early stages of the individual's development, though often mild and undetected, was adopted by Tredgold (1908); and at a later stage by others, such as Pasamanick et al. (1956a), as an attempt to explain the behavioural problems seen in hyperkinetic children. The encephalitis epidemic in 1917–18 played a significant part in the history of hyperactivity. Following the epidemic, clinicians were confronted with children suffering from behavioural and cognitive sequelae, many presenting with the core features of what would today be termed hyperactivity.

For the first half of the twentieth century, the predominant view regarding the causation of hyperactivity was that of an association with brain damage. A plethora of terms such as 'organic drivenness' and 'minimal brain damage' were used to describe the disorder. During this period, the similarity between the behaviour of hyperactive children and that of primates subjected to frontal lobe lesions was noted. This was used by several investigators to support the idea that hyperkinetic disorders were due to defects in forebrain structures, despite the lack of evidence of such lesions in many children.

During the fourth and the fifth decades of the last century, a series of papers were published which marked the beginnings of child psychopharmacology in general, and the pharmacotherapy of behaviourally disturbed children in particular. By the end of the 1950s, the concept that brain damage was the single important factor in the development of hyperkinesis was challenged. 'Minimal brain dysfunction' was substituted for 'minimal brain damage'. At the time, a variety of hypotheses were put forward regarding the causation of hyperactivity, including the psychoanalytic theory of poor parenting. With the decline of
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the concept of 'minimal brain dysfunction', the idea of the 'hyperactive child syndrome' was being developed, with Stella Chess (Chess, 1960) as one of its chief proponents. Chess differed from her predecessors in that she viewed the prognosis of hyperactive children as reasonably good, with the condition having resolved by puberty in most cases. By the end of the 1960s the prevailing view was that hyperkinesis represented a form of brain dysfunction, and presented with a variety of symptoms, of which motor overactivity was the most predominant.

It was during the 1960s that the split developed between Europe and North America over the conceptualization of hyperkinesis. Clinicians in Europe maintained a narrower view of the disorder, seeing it as a rare syndrome of excessive motor activity, usually occurring in conjunction with some signs of brain damage. In North America, on the other hand, hyperactivity was seen as being relatively common, and in most cases not necessarily associated with overt signs of brain damage. The differences became highlighted in the diagnostic classificatory systems used (International Classification of Disease (ICD); World Health Organization, 1992 and Diagnostic and Statistical Manual of Mental Disorders (DSM: American Psychiatric Association, 1980), respectively).

In the 1970s, the emphasis moved away from motor overactivity and began to focus on the attentional aspects of the disorder. A number of authors demonstrated that hyperactive children had great difficulty with tasks involving sustained attention. At the same time, there was also a growing belief that hyperkinetic behaviour was due to environmental causes. This coincided with a move towards a healthier lifestyle and disquiet about the 'drugging' of schoolchildren. The idea that hyperactivity was, at least in every other case, due to an allergic reaction to food substances, particularly food additives, became popular at this time. Technological advance and other cultural influences were also put forward as causative factors. An additional development of the decade was the increase in the number of studies investigating the psychophysiology of hyperactivity.

During the 1980s the explosion of research in the field continued. With this came the development of research criteria and standardized assessment procedures. Advancements were also made in the area of treatment, with new methods involving cognitive-behavioural therapies. Hyperactivity came to be seen as a condition with a strong hereditary component, chronic in nature and causing significant handicap in the areas of academic achievement and socialization; treatment required the complementary skills of a variety of professionals.

Throughout the 1990s hyperactivity and attention disorders continued to
generate more research literature than any other child psychiatric disorder. The breadth and range of the subject matter of this literature are enormous and encompass research into the genetics and neurobiological bases of hyperactivity, together with studies examining the relative efficacy of different treatment methods. The 1990s also saw the development of management guidelines, including the European Society for Child and Adolescent Psychiatry guidelines (Taylor et al., 1998), and the American Academy of Child and Adolescent Psychiatry practice parameters (Dulcan, 1997), in an attempt to facilitate standardization in practice. These guidelines emphasize the importance of individualized, multimodal, multidisciplinary assessment and treatment of hyperactivity and attention disorders by practitioners experienced in the field. Increasingly, it is also recognized that these disorders can and do persist through adolescence and into adulthood. Hence, the past decade has also seen a surge of interest in their presentation and treatment in adults.

Early medical explanations

The modern history of hyperactivity disorders is traditionally seen to begin with the writings of Sir George Frederick Still (1902) and Alfred F. Tredgold (1908), with many authors acknowledging that our present concept of hyperactivity has its foundation in their work (Ross and Ross, 1982). Preceding the studies by Still and Tredgold, extensive descriptions of hyperkinetic children, mostly in the form of individual case studies, had already appeared in the psychiatric literature of the nineteenth century (Hoffmann, 1845; Maudsley, 1867; Ireland, 1877; Bourneville, 1897; Clouston, 1899) and continued to be published in the early decades of the twentieth century (Pick, 1904; Boncour and Boncour, 1905; Scholz, 1911; Heuver, 1914).

Overexcitability and mental explosiveness: forerunners of hyperactivity?

Clouston (1899) described a series of very difficult morbid conditions that 'occur in neurotic children, but lie on the borderland of psychiatry'. He hypothesized that the disorders were all due to some dysfunction in the 'brain cortex', and were pathogenically 'states of deranged reactiveness of the neurones of the higher regions of the brain'. The derangement had arisen because the higher centres of the brain, responsible for inhibiting activity, had in some way been weakened and therefore become unable to cope with the amount of 'energising they have to control'.

Clouston, however, emphasized that such disorders should not be classified as 'out and out mental disorders'. He believed that the conditions were
Historical development

...disorder. The numerous and informal treatment guidelines, often conflicting, have been insufficient to facilitate the management of hyperactivity. Adolescent hyperactivity is also seen as an extension of the hyperactive child. Hyperactivity, characterized by excessive energy, physical and verbal restlessness, and distractibility, is divided into categories: mild, moderate, and severe. The mild form may indicate a response to the transition from childhood to adolescence. The moderate form is associated with poor academic performance, and the severe form with severe cognitive and emotional difficulties.

...ultimately caused by 'hereditary and congenital peculiarities', with defects in the 'subtle and obscure process of central nerve development in childhood' acting as predisposing agents. He further postulated that certain areas of the brain 'running ahead' in their development, compared to others, were causing the derangement of its function. This in turn caused 'explosions which spread into other centres'.

A series of morbidities was outlined which, as Clouston proposed, all resulted from the same pathology; one of these resembled the attention deficit hyperactivity disorder of today. He called it 'simple hyperexcitability', arguing that the disorder resulted from 'undue brain reactiveness to mental and emotional stimuli'. It affected children from the age of 3 years until puberty, with overactivity and restlessness being the main symptoms. The disorder came in bursts and lasted from a few months to years. During the bursts the child would grow thin, not sleep and deteriorate in educational performance. Such clinical signs were ascribed to the children showing a 'delirium of pleasure in response to nice things', and interpreted as an exaggeration of the response one would expect from a child of nervous temperament.

The common feature of such disorders, according to Clouston, was the 'explosive condition of the nerve cells in the higher cortex'. In his view this was comparable to the overactivity of the motor cortex seen in persons suffering from epilepsy. Referring to the by then well-established findings that the process of brain development in childhood comprises rapid cell multiplication accompanied by the cells gradually becoming more stable, he argued that in the children suffering from 'hyperexcitability' this process of nerve stability does not occur, resulting in the children growing up with 'irregular explosive tendencies'.

The recommended treatment for such conditions was the use of bromides, 'fearlessly in large doses up to the point when symptoms of bromism are beginning to show themselves'. Clouston did, however, emphasize that the drugs should not be used in isolation. Instead, the children should at the same time be given a good diet, plenty of fresh air, 'suitable amusement, companionship and employment'. The aim of the treatment was to 'reduce the cell catabolism and the reactiveness of the cerebral cortex whilst not interfering with brain anabolism'. The treatment had to be carefully monitored in order to ensure that it did not 'go too far'.

Still and the defect of moral control

The first clear, systematic accounts of hyperactivity are attributed to Sir George Frederick Still (1868–1941), a paediatrician and the first professor of children's
diseases at King’s College Hospital, London. Professor Still is, however, most notable for his description of chronic rheumatoid arthritis in children, commonly known as Still’s disease. In 1902, Still presented the Cloustonian lectures to the Royal College of Physicians describing the case histories of 20 children, whose presentation was similar to what we at present would call hyperactivity. In his descriptions, features such as extreme restlessness and ‘almost choreiform’ movements were frequently mentioned. Another common characteristic was that of ‘an abnormal incapacity for sustained attention’, causing school failure even despite normal intellect. In their behaviour, many of the children were mischievous, destructive and violent, and appeared not to respond to punishment.

According to Still, this pattern occurred more often in boys than in girls, became frequently apparent by early school years, was sometimes accompanied by peculiarities of physical appearance (e.g. epicanthic folds and high arched palate), seemed often to be a function of temperament, generally showed little relationship to the child’s training and home environment, and commonly shared a poor prognosis. Thus, Still described many of the characteristics and associations we recognize today. Still suggested that the children he was describing suffered from a ‘defect of moral control’, whereby they demonstrated ‘the reckless disregard for command and authority in spite of such training and discipline as experience shows will render a healthy child law-abiding’. Instead, such children displayed ‘immediate gratification of self without regard either to the good of others or to the larger and more remote good of self’. Still also noted that, although many of the children with this condition came from chaotic families, a substantial proportion of them lived in homes where they appeared to receive an adequate upbringing. In fact, when refining his criteria for children with the disease, Still decided to exclude those who had been exposed to poor child-rearing. This led him to hypothesize that the ‘defect of moral control’ was due to some ‘morbid physical condition’, which was either inherited or resulted from a perinatal or postnatal injury.

The degree of uncertainty about the causation of the condition provided a logical basis for separating the children with such problems into subgroups. Still proposed a distinction between children with demonstrable gross lesions of the brain; those with a variety of acute diseases, conditions and injuries that would be expected to result in brain damage, although none could be demonstrated; and those with hyperactive behaviours that could not be attributed to any known cause. Thereby, Still laid the groundwork for the historical equivalents of three major diagnostic categories of brain damage, minimal brain dysfunction and hyperactivity. In doing this, he also sowed the seeds for a terminologi-
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cal confusion so prevalent in the literature of hyperactivity over decades to
come, but confusion that also gave the impetus for much excellent research on
the nature of hyperactivity and its treatment.

Still’s theories about his patients are best understood in the context of the
prevailing socioeconomic and scientific climate. During the nineteenth century
the UK underwent significant economic, political and social change. The
economy became increasingly centred on factories in small towns, moving
away from farming and the land. Unemployment was common and those in
employment often worked long hours and were poorly paid. A distinct class
hierarchy dominated society, with the lower classes being perceived as im-
moral and inferior (Rowntree, 1901). Considerable adversity afflicted the lower
classes as a result of the socioeconomic changes and this was reflected in rising
infant mortality, poor physical health in general, and learning difficulties and
delinquency in children. However, the intellectual and moral deficiencies of the
lower classes tended to be identified as the cause, rather than the consequence,
of their circumstances.

Concurrent with these developments was the rise of positivism in contem-
porary science, with beliefs that the progress of society could be achieved
through the development of objective science. It was especially the theories of
Darwin that provided a scientific rationale for various kinds of social deviance,
with hypotheses suggesting that the environment conferred a selective advan-
tage to some types of biological variation (Darwin, 1859). Deduced from his
theories, the notion for the ‘survival of the fittest’ became elevated to the status
of a ‘law’, in an attempt to explain social phenomena. Likewise, poor health
could easily be viewed as a form of inherited weakness and inferiority. This
social Darwinism soon found wide support among intellectuals and social
reformers.

In keeping with the prevailing trend of his day, Still was keen to adopt the
principles of social Darwinism and set out to explain the ‘defects of moral
control’ of the children he was asked to treat. He claimed that moral conscious-
ness and moral control were essentially innate characteristics. They were also
‘the highest and latest product of mental evolution’. However, because they
constituted a relatively recent evolutionary advance, they were also fragile and
showed ‘a special liability to loss and failure in development’.

Hyperactivity due to neuropathic diathesis

To support Still’s reports of hyperactive behaviour pattern occurring when
brain damage was suspected, but could not be substantiated, Tredgold (1908)
presented further evidence in children. He proposed that some forms of brain
damage, such as birth injury or relatively mild anoxia, though undetected at the
time, could express themselves as behaviour problems or learning difficulties
when the child is faced with the demands of early school years. Alfred E.
Tredgold was a member of the English Royal Commission on Mental Defi-
ciency. His book, Mental Deficiency (Amentia), was published in 1908, updated in
1914, and remained in publication until 1952. In the book, Tredgold described
many children who exhibited features of hyperactivity, and he is attributed by a
number of authors (Ross and Ross, 1982) as being the first to provide an
account of 'minimal brain damage'.

Tredgold's descriptions of hyperactivity derived from his observations of a
group of child patients whom he labelled as 'high-grade feeble minded'.
Although incapable of making use of education as provided in school, such
children would in his judgement nevertheless benefit from individual attention
and instruction. He also noted that a number of the children exhibited a variety
of physical anomalies, including abnormalities of the palate, soft neurological
signs, abnormal head shape and size and poor coordination.

Apart from being educationally inferior, the children were also prone to
criminal behaviour, despite having been raised in an adequate environment.
Tredgold shared Still's belief that moral deficiency resulted from the effect of
some 'organic abnormality on the higher levels of the brain', and argued that
the areas of the brain where the 'sense of morality' was located were the
product of the more recent development in the course of human evolution, and
were therefore more susceptible to damage. Tredgold believed that such moral
deficiency was caused by the inheritance of some brain defect that was being
passed on from generation to generation: being able to take various forms, it
resulted in hyperactivity, migraine, mild forms of epilepsy, hysteria and neurasthenia. He called the defect by various names, such as 'neuropathic diathesis',
'psychopathic diathesis', 'blastophoria' or 'germ corruption'. In his view, environ-
mental circumstances played no significant role in the causation of such
mental or moral deficiency.

With regard to the influence of slum life and all its associated conditions in producing amentia, it is
necessary to sound a note of warning. It does happen sometimes that the real mental defectives
of our large towns hail from the slums, although I do not think such is disproportionately the
case. Still, a sufficient number of defective children come from such areas to make the superficial
enquirer content with that which is apparent. Jumps on the conclusion that the pernicious
environment is therefore the cause of their defect. My own enquiries have convinced me that in
the great majority of these slum cases, there is pronounced morbid inheritance, and that their
environment is not the cause, but the result, of that heredity. (Schachar, 1986, p. 25).
In the decades that followed, a wide range of deviance was attributed to the interaction of brain disorder and constitutional predisposition by leading medical authorities on both sides of the Atlantic. Such biological variation could in turn have several different outcomes, ranging from school failure (Cornell, 1912), to criminality (Healy, 1915). In contrast, psychological and social explanations for cognitive and behavioural deficits were explicitly rejected. The core debate revolved around the relative contributions of inheritance and birth injury as factors of prime importance in leading to disturbed adaptability towards one’s surroundings (Henderson, 1913).

**Sequelae of the encephalitis epidemic**

The link between hyperactive behaviour and demonstrable brain damage was strengthened by the epidemic of encephalitis spreading across Europe and the USA in 1917–18. In its aftermath, many clinicians encountered children who, though having survived the infection, subsequently presented with behaviour problems and cognitive deficits. Hyperactivity, catastrophic changes in personality and learning difficulties were among the predictable sequelae of the disease (Hohman, 1922; Ebaugh, 1923). The term ‘postencephalitic behaviour disorder’ was adopted to encompass the various consequences. Observations of the child victims of subsequent outbreaks of encephalitis confirmed the same pattern of symptoms (Bender, 1943; Gibbs et al., 1964). Cantwell (1975) and many others date the development of North America’s interest in hyperactivity to the encephalitis epidemic.

Hohman (1922), Ebaugh (1923) and Strecker and Ebaugh (1924) argued that the children who showed persisting problems with behaviour following the epidemic were also the ones who had been most severely affected by the disease, and in most cases were left with severe brain dysfunction. As such, however, only the problems of few of the children described would fit the present-day criteria for attention deficit hyperactivity disorder. It is also to be acknowledged that the available evidence was for an association between severe damage and severe behavioural disturbance. For some reason, this was subsequently extrapolated to claim that a similar connection existed between minimal brain damage and lesser degrees of disordered conduct.

As was the case with Still’s disease, the influences of social Darwinism were also brought to bear on the sequelae of encephalitis, with the assumption that there was some inherited predisposition to developing the disease. People who contracted illnesses, such as encephalitis, were believed to be in some way constitutionally inferior (Bassoe, 1922; Bond and Appel, 1931).
Hyperkinetic disease

In the early 1930s, Kramer-Pollnow described a syndrome characterized by extreme restlessness, distractibility and speech disorder. He called it 'hyperkinetische Erkrankung' (hyperkinetic disease) and classified it as a form of childhood psychosis, usually of unknown origin (reported by Hoff, 1956, pp. 537–53). The extreme restlessness, commencing in the third or fourth year of life, often came on suddenly, 'after a period of quiet', and was frequently followed by an epileptic seizure. The restlessness reached its most severe form at the age of 6, and then gradually decreased, with most cases achieving almost complete recovery. Disturbances of speech and general mental development were often noted before the onset of restlessness.

The children were also described as being easily distracted. Their excessive motor activity, chaotic and aimless in nature, appeared to occur as a succession of uncorrelated impulses with no aim other than to respond to a stimulus. Their play also seemed to lack purpose, with toys tending to be broken rather than played with. The 'lack of discrimination', as noted, bears a resemblance to the 'impulsivity' described in children suffering from hyperkinetic disorders of today. The paucity of the children's interpersonal relationships was likewise remarked upon. They were also more often aggressive towards other children rather than playing with them. Any attempts to restrain the child would be met with opposition and struggling.

The speech disorders, as recorded, consisted of poor articulation and 'inarticulate labelling': the children's vocabulary was thought not to increase until their recovery from the condition. However, their intelligence, when observed daily at home, appeared to be higher than that detected on formal cognitive testing. It was also argued that the disorder could be differentiated from others such as schizophrenia, dementia infantilism, encephalitis and schizophreniform-like psychoses.

In total, 15 children presenting with 'hyperkinetic disease' were described. Of these one died, three suffered from definite mental defect, three recovered from the restlessness but were left with permanently impaired intelligence, four recovered partially, whilst another two recovered completely. The remaining two children were less than 7 years old and were therefore felt still to be within the 'hyperactive stage'.

A small compilation of cases with similar presentation had also been published in Italy a few years previously (de Sanctis, 1925). Indeed, after having examined the evidence collected by both Kramer-Pollnow and de Sanctis, Hoff concluded the topic of 'Hyperkinetische Erkrankung', as part of his three-lecture series on child psychiatry to his Viennese medical students, by adopting
a revised view. He stated clearly that, rather than a form of psychosis, the hyperkinetic disease was due to some kind of endogenous disturbance of brain metabolism – and quite possibly had a hereditary basis (Hoff, 1956, p. 544).

In the Soviet Union, on the other hand, hyperactivity appears to have been a well-acknowledged condition almost from the inception of the new federal state. According to Isaev and Kagan (1981), as early as the 1920s Soviet child psychiatrists Gurevich, Kashchenko (1919), Simson (1929) and Jogikhes (1929) are recorded as having emphasized that hyperactivity was a combination of a medical and a pedagogical problem. In addition, Gurevich (1925) had described in some detail the work done at a ‘psychoneurological sanatorium’ school, established in 1919. The school was meant for ‘near-normal children with minor aberrations’, such as nervousness and general neurological imbalance. It also seems that the view of hyperactivity as a condition closely akin to ‘hypersthenic form of neurasthenia’ guided Soviet child psychiatrists’ approach to the diagnosis, etiological explanations and treatment of the disorder for decades to come. The medication aspect of the treatment often consisted of a mixture of stimulants and tranquillizers, the relative proportions of which were determined by the assumed ratios of excitory and inhibitory processes operating in the child’s nervous system (Isaev and Kagan, 1978, 1981). Likewise, in the former DDR, where terms such as ‘an erethic child’ were used to describe hyperkinesis, the nature of medication treatment depended on whether or not the restlessness was accompanied by rapid tiring. In the former instance, stimulants were the choice, and in the latter it was tranquillizers (Göllnitz, 1981).

Organic drivenness

In 1934, Kahn and Cohen described three patients whose clinical condition was marked by hyperkinesis, an inability to remain quiet, abruptness, clumsiness and explosiveness of voluntary activity. The authors argued that all the symptoms were secondary to a central behaviour abnormality – hyperactivity. This, in turn, was a result of ‘organic drivenness, or a surplus of inner impulsion’, originating from an abnormality in the organization of the brain stem, often caused by trauma, or ‘prenatal encephalopathy or birth injury’. Acknowledging that in a number of such hyperactive children no history of trauma could be confirmed, Kahn and Cohen postulated that a congenital defect in the activity-modulating system of the brain stem was the cause of the condition. In their view, both the over- as well as the underdevelopment of certain areas of the brain were capable of leading to the same endresult. They also added that features such as soft neurological signs, e.g. twitching, and minor congenital
dysmorphisms were evidence of the constitutional nature of these deficits.

Further evidence of a causative link between brain damage and hyperactivity was provided by studies of epilepsy and other brain disorders (Clark, 1926; Lord, 1937; Preston, 1945). Also, children with confirmed lead poisoning were often found to sustain severe neurological and psychological sequelae, one of the latter being a behavioural expression of hyperkinesis, short attention span and impulsivity (Byers and Lord, 1943). The characteristics of the children suffering from 'motor restlessness syndrome', as it appeared in the early recorded descriptions in Soviet child psychiatry (Sukhareva, 1940), are strikingly similar.

**Associations with primate research**

Studies carried out in the latter half of the nineteenth century (Fernier, 1876) had shown that frontal lobe ablation in monkeys produced excessive restlessness and poor concentration. During the 1930s, several researchers noted that there was a similarity between the behaviour shown by primates who had undergone frontal lobe ablation and that of hyperactive children (Blau, 1936; Levin, 1938). This was taken as evidence in support of hyperkinetic behaviour in children as the result of some defect in forebrain structures, even though signs of such lesions could not always be demonstrated in all affected individuals.

**Psychostimulant treatments and their effect on theory of hyperkinesis**

The efficacy of amphetamines in the treatment of hyperkinesis was discovered purely by accident by Charles Bradley, working at the Emma Pendleton Bradley Home in Providence, Rhode Island, USA. The details of the discovery are described in a letter from Mortimer D. Gross to the editor of the *American Journal of Psychiatry* in 1995 (Gross, 1995). In this letter, Gross recalls the story of the breakthrough as recounted to him by M.W. Laufer. Bradley and his colleagues, having been impressed by the ideas of Kahn and Cohen (1934), also working at the centre, decided to join in the efforts to demonstrate the nature of structural abnormalities in the brain which they believed were the cause of disruptive behaviour in children. For this purpose, pneumoencephalograms were widely employed, with the consequence that many of the children were left with severe headaches. The headaches were thought to be due to the loss of spinal fluid and Bradley believed that if he could stimulate the choroid plexus to produce spinal fluid more rapidly, the headaches would be alleviated. He chose benzedrine as the most potent stimulant available at that time.

Most of the children given benzedrine (amphetamine derivative) for their
historical development

...e deficits.

peractivity lark, 1926; in some there was also a rise in intelligence test scores (Bradley and Green, 1940), which was attributed to the drug's favourable effect on the child's emotional attitude towards the test. Furthermore, it was noted that a wide range of children benefited from the medication, and that the therapeutic effect was not specific to any particular behaviour, although the children who improved least turned out to have a demonstrable structural neurological deficit (Bradley and Bowen, 1940, 1941).

The mechanism of the drug's action was at first attributed to its effect of stimulating higher inhibitory centres, thereby producing increased voluntary control. In addition, Bradley (1957) commented on the importance of the euphoriant effect of amphetamines. He believed that children with behaviour problems, many of whom showed marked overactivity and restlessness, were unhappy, and that their deviant behaviour was their way of conveying this. Therefore, giving them amphetamines made them less unhappy, and took away some of their need to behave badly.

Laufk (1975) later suggested that the reason the drug was not widely used until the mid 1950s was because of the prevailing psychoanalytic climate, which resisted the idea that hyperactive behaviour had an organic basis. Laufk and his colleagues took up the cause of amphetamines and set out to investigate the neurological mechanism underlying the 'hyperkinetic impulse disorder', and the action of amphetamines (Laufk and Denhoff, 1957; Laufk et al., 1957). Attempting to secure some sound evidence for the drug's effectiveness, in the face of strong competition from those who promoted the role of poor parenting as the cause for hyperactive-type behaviour problems, was an uphill struggle.

Laufk's research was heavily built on the work of two of his contemporaries, Gastaut (1950) and Magoun (1952). Gastaut had developed a photometrozo stimulation test, which involved administering the drug metrazol whilst lights were flashed at the child. The resulting myoclonic jerks of the forearm and spike-wave patterns on the EEG were then carefully monitored. Magoun's work centred on the importance of the ascending reticular activating system in preserving alertness.

Hypothesizing that abnormalities of the reticular activating system led children to be overalert and overactive, Laufk et al. (1957) embarked on a study of children with 'emotional disturbance', dividing them into those who showed evidence of 'hyperkinetic impulse disorder' and those who did not. They found that the hyperkinetic group required less metrazol to induce myoclonic jerks in response to the strobonoscope compared with the...
nonhyperkinetic group. However, when the hyperkinetic children were given amphetamines, the amount of metrazol required increased to the same level as in the nonhyperkinetic children. The authors postulated that the central nervous system deficit of overactive children was located in the area of the thalamus. As a result of this, excess stimulation was not filtered out, but instead passed through to the brain, causing the child to behave in an overactive manner.

It has later been pointed out that Laufer's research did suffer from certain methodological flaws; for example, there had been a failure to record the post-metrazol levels of the nonhyperkinetic children. However, even though the original study has never been replicated, Laufer's early work has definitely fuelled much interest in the study of biological treatments in childhood hyperactivity.

From minimal brain damage to minimal brain dysfunction

Minimal brain damage/minimal damage to the brain

The belief that brain damage could be inferred laid the foundation for the concept of minimal brain damage (Ehrenfeucht, 1926; Smith, 1926; Doll et al., 1932; Ingalls and Gordon, 1947). The publications and 'minimal stimulation' classroom programmes by Strauss and his associates (Strauss and Werner, 1942, 1943; Strauss and Lehtinen, 1947; Lehtinen, 1955) further helped to promote the idea that hyperactive behaviour was caused by minimal brain damage. The group studied the psychopathology and educational needs of children residing in the Wayne County Training School, Michigan, USA. The school provided rehabilitation for children considered to be of borderline intelligence, or 'higher grade morons'. Strauss and Kephart (1955) suggested that the borderline children should be divided into two distinct groups, which they called 'exogenous' and 'endogenous'. The 'exogenous' children had histories of physical damage to the central nervous system, either due to trauma or inflammatory process, but no family history of mental retardation. The medical histories of the 'endogenous' children did not contain any evidence of physical damage to the brain, but the children came from families with a history of mental retardation. The two groups also differed with regard to their behaviour and current functioning: the 'exogenous' children responded poorly to teaching, and appeared to be overactive and easily distracted. Prompted by their observations, the group set out to determine the nature of the brain-injured child's deficit, and to develop appropriate educational programmes for such children.

As a result of their observations, Strauss and Werner (1943) concluded that
The behaviour of the brain-injured children closely resembled that of brain-injured adults. This led them to postulate species-specific innate patterns of behaviour. When a child suffers a brain injury, these particular behavioural patterns are triggered by less intense stimuli, but produce more intense responses than is the case with adults. Although their initial studies were based on intellectually impaired children, the authors argued that there was no reason why these same behaviour patterns should not indicate brain damage in children of normal intelligence.

Research findings like these were appealing at the time, because they provided a biological explanation for children's behaviour problems and educational failure. They also excited paediatricians and child psychiatrists who, when trying to help such children, had grown disillusioned and dissatisfied with the lack of success of treatments based largely on psychoanalytic theories. Although welcomed by many, the conclusions by Strauss and his colleagues were also met with scepticism by a number of people. The main criticisms related to the lack of clarity of the criteria by which organic brain disorder was defined. Also, the educational programmes that had been guided by the research frequently turned out to be ineffective (Cruickshank et al., 1961), and the attempts to replicate the psychometric results proved unsuccessful in most instances (Weatherwax and Benoit, 1962).

At the same time as the theories put forward by Strauss and his coworkers were struggling to gain wider acceptance, the validity of the concept of minimal brain damage was strengthened by a substantial body of fetal and animal research. Epidemiological studies demonstrated a strong association between maternal and fetal factors and subsequent behaviour problems in children (Lilienfeld et al., 1955; Pasamanick et al., 1956b), and results of animal studies supported a relationship between disordered behaviour and minor degrees of brain damage (Cromwell et al., 1963). Empirical evidence for a significant link between histories of anoxia at birth and later disorders of development appeared particularly suggestive (Graham et al., 1957), as did the systematic clinical observations of the behaviour of children suffering from epilepsy (Ounsted, 1955), and of other forms of organic brain disorder (Ingram, 1956).

The term 'minimal brain damage syndrome' was a diagnosis also widely used by Soviet and east European clinicians and researchers from the 1950s, at least until the end of the 1970s. Particularly, a constellation of several (even dozens of) symptoms, such as dyslexia, dysgraphia, dyscalculia, attention problems, overall cognitive difficulties and aggression, in addition to motor restlessness, merited the diagnosis in the Soviet practice (Badalyan et al., 1978;
Isaev and Kagan, 1978; Kovalev, 1979). In the former DDR, hyperkinesis was viewed as one aspect of encephalopathy (organic brain syndrome), not indicating any specific illness (Göllnitz, 1981). Rather, a combination of biologically and neurologically tangible symptoms, together with the child’s interactions with his or her environment, formed the syndrome (Laehr, 1975). Also, the term ‘uninhibited child with choreatic symptoms’ was used (Lemmke, 1953).

The continuum of reproductive casualty
The introduction of the concept of a continuum of damage, with a corresponding continuum of medical, behavioural and educational consequences, by Pasamanick and his colleagues presented a theoretical step forward (Knobloch et al., 1956; Pasamanick et al., 1956a,b). The argument was based on the observation that the cause of perinatal death in babies whose mothers had had severe complications of pregnancy, and/or where the pregnancy ended prematurely, was usually due to brain injury. Therefore, amongst the group of infants who did not die, some would be severely injured and develop a variety of disorders. Depending on the severity and location of the injury, the disorders ranged from cerebral palsy through conduct disorders and learning difficulties to mild behaviour problems (Pasamanick and Knobloch, 1961). These findings stemmed from a study of children referred to the Division of Special Services of the Baltimore Department of Education, with nonreferred children from the same classrooms being used as controls. The referred children were found to be three times more likely than the controls to have a history of perinatal complications such as anoxia or premature birth. This was particularly true of those children who presented with hyperactive-type behaviours. In a subsequent prospective study, involving 500 children who had been born prematurely, the incidence of such developmental abnormalities was found to increase with decreasing birth weight (Knobloch and Pasamanick, 1966). Although the findings were mainly interpreted as lending support for the role of gestational and perinatal factors in the later development of hyperactivity, an association between socioeconomic status, race and complications of pregnancy was also recorded. There was a higher incidence of perinatal events in lower socioeconomic and nonwhite populations. This led the authors to conclude that the perinatal complications and adverse environmental factors most likely stemmed from the same social disadvantage.

Minimal dysfunction replaces minimal damage
While Pasamanick and his colleagues were promoting their research as providing evidence for a link between minimal brain damage and hyperactivity,
several authors criticized the idea, increasingly doubting the concept of brain damage as the sole cause of children's hyperactive behaviour. Birch (1964), Rapin (1964) and Herbert (1964) all questioned the assumption that, if brain damage caused behaviour problems, then all children with behaviour problems must have brain damage – even though there might be no physical evidence to support the presence of damage.

Coinciding with this, the Oxford International Study Group of Child Neurology (MacKeith and Bax, 1963) also contended that brain damage should not be inferred from behaviour alone, and recommended that the term 'minimal brain damage' be replaced by 'minimal brain dysfunction' (MBD). It also advocated attempts to reclassify the heterogeneous group of children, subsumed under this label, into a number of more homogeneous subgroups. In one of the longest-established London medical schools, the professor of psychiatry (Pond, 1967, p. 127), specializing in problems of behavioural disturbance and brain damage, also adopted a very sceptical view, and stated: 'There are . . . no absolutely unequivocal clinical signs, physiological tests or psychological tests, that prove a relationship between brain damage and any particular aspect of disturbed behaviour'.

In the USA, a national task force formulated an official definition of the disorder (Clements, 1966):

The term minimal brain dysfunction syndrome refers . . . to children of near average, average, or above average general intelligence with certain learning or behavioral disabilities ranging from mild to severe, which are associated with deviations of function of the central nervous system. These deviations may manifest themselves by various combinations of impairment in perception, conceptualisation, language, memory, and control of attention, impulse, or motor function . . . during the school years, a variety of learning disabilities is the most prominent manifestation (quotation by Ross and Ross, 1982).

The definition was welcomed by those who believed hyperactivity to be an unequivocal diagnostic sign of brain damage (Wender, 1971), but criticized by many (Routh and Roberts, 1972; Rutter, 1977), Gomez (1967), the head of child neurology at the Mayo Clinic, expressed his views on the topic without undue reservation: To him the term 'minimal brain dysfunction' stood for 'maximal neurologic confusion'. Although the term 'MBD' was useful in emphasizing the role of organic factors in the causation of hyperactivity, and thus providing something of a challenge to the psychoanalytic views of the time, proposing that the disorder was due to poor parenting, it was eventually recognized as being overinclusive. 'MBD' was subsequently replaced by more specific terms relating to particular behavioural and developmental disorders, such as...
dyslexia, learning disabilities and language disorders. These terms were based on observed disabilities rather than hypothesized underlying mechanisms.

Emergence of behavioural definitions

Three decades elapsed between Still's (1902) description of children with hyperactive behaviour unrelated to either demonstrable brain damage or a history of suggestive damage and the first comprehensive discussion of hyperactive children. In 1935, Childers noted that only a small proportion of cases with hyperactivity seemed aetologically related to substantiated or inferred brain damage. His deliberation on children with no evidence of brain damage is notable for the differentiation made between the hyperactive child and the brain-damaged child.

Hyperkinetic behaviour syndrome

During the late 1950s and early 1960s, dissatisfaction with the term 'MBD' was growing. The influential publications by Laufer and his associates (Laufer and Denhoff, 1957; Laufer et al., 1957), which introduced the concepts and terms of 'hyperkinetic behaviour syndrome' and 'hyperkinetic impulse disorder', signalled the onset of final acceptance of the construct of hyperactivity as we know it now.

Chess (1960) also emphasized the importance of excessive motor activity as a defining feature of the disorder, and that this should be observed rather than taken on as an anecdotal account from the parents. In addition, she moved away from attributing the disorder to poor parenting or brain damage. Her findings were based on a group of 36 children, termed hyperactive, in a whole sample of 881 children attending a private practice. The features, documented in detail, resemble the present criteria of attention deficit hyperactivity disorder, including sex ratio and age of onset. Aggression and impulsivity were seen as associated characteristics. Chess (1960) proposed that in most cases the disorder was due to 'physiologic hyperactivity', but also noted that hyperactivity could be associated with mental retardation, schizophrenia or organic brain damage. A treatment approach incorporating behaviour modification, medication, special educational provision and psychotherapy was recommended.

The changes in the concept and terminology served as a stimulus for a series of important empirical investigations (Smith, 1962; Werry et al., 1964; Huesey, 1967) and descriptive clinical studies (Bakwin and Bakwin, 1966; Stewart et al., 1966; Werry, 1968). By the late 1960s, the concept of hyperactivity was firmly established in the literature.
First systematic tools for assessment

In the late 1960s, Conners and his team developed parent and teacher rating scales for the assessment of symptoms of hyperactivity (Conners, 1969, 1970). At that time, these questionnaire-based instruments presented an invaluable step forward in allowing for a standardized measurement of children's behaviour with a particular emphasis on hyperactivity. Although initially devised for the measurement of change in behaviour during drug treatment, the scales have also been successfully employed in epidemiological research.

Psychophysiology and the importance of arousal level

Several psychological models have attributed hyperactive behaviour to an abnormality in the level of arousal in the central nervous system. However, the direction of postulated abnormality has been a matter of considerable dispute. The models viewing the deficit as one of overarousal have interpreted hyperactivity as a behavioural manifestation of an overaroused, or highly aroused central nervous system (Kahn and Cohen, 1934; Strauss and Lehtinen, 1947; Laufer et al., 1957; Freibergs and Douglas, 1969). The underarousal models, on the other hand, have conceptualized the condition as a compensatory response by a suboptimally aroused individual through increased sensory input (Bradley, 1937; Werry and Sprague, 1970).

The psychophysiology of hyperactivity was extensively investigated during the 1970s. Several studies were published during this time examining a variety of physiological aspects, such as EEG, galvanic skin response, averaged evoked responses, etc. (Satterfield and Dawson, 1971; Satterfield et al., 1972, 1974; Satterfield, 1973). Many of these also had considerable methodological problems and were highly criticized (Hastings and Barkley, 1978). One of the weaknesses of the psychophysiological research was that the studies were often based on outdated theories of the 1950s suggesting, for example, that hyperactivity was due to cortical overarousal. If anything, the evidence generated suggested that hyperactive children had a slower than normal response to stimulation, thus detracting from theories of overstimulated cortices in these children.

Deficits of attention and motivation

The 1970s saw a proliferation of research into hyperactivity and a fast-growing volume of literature on various aspects of the disorder. Particularly in the USA, features such as short attention span, impulsivity and distractibility, previously considered to be associated characteristics, were now included in the defining
symptoms. At the same time, the popularity of the concept of MBD was declining due to the lack of scientific evidence in its favour.

One of the turning points in the history of hyperactivity was when, in the early 1970s, Douglas and her team at McGill University suggested that motor overactivity was not the core symptom in the syndrome of hyperkinetics, but rather deficits in the ability to sustain attention and control impulsive responding were more important. Douglas also argued that these were the areas in which stimulant medication was most effective (Douglas, 1972).

Employing a series of measures to assess the behavioural and cognitive aspects of the disorder, the McGill team demonstrated that hyperactive children experienced more problems with sustained attention, particularly in situations where there were distractions, but that they were not generally more learning- or reading-impaired, or more distractible than normal children. However, in situations where the children were given continuous and immediate reinforcement, the hyperactive ones could perform at near normal and even normal levels of attention (Freibergs and Douglas, 1969). Another important observation was that, while the excessive motor restlessness usually declined by the early teens, the difficulties in sustained attention and impulse control often persisted into adolescence.

One of the doubtless merits of the systematic and detailed work of Douglas and her colleagues was that it helped to establish a tradition of high-quality research in the study of cognitive aspects of hyperactivity. Another notable feature of her contribution has been the way it was guided by theory, which can be tested and revised when necessary; these are characteristics so often found wanting in the burgeoning hyperactivity research.

The work of Douglas and her team has definitely been influential in shaping the study of hyperactivity. It was probably the main reason for the American Psychiatric Association (1980) altering the diagnostic terminology in DSM-III to attention deficit disorder ± hyperactivity, thus focusing on the attentional aspects of the disorder, rather than on the motor overactivity. Although the theory of Douglas has subsequently been questioned in part, it nevertheless provided the impetus for a new generation of high-quality research in the various aspects of hyperactivity, including the individual processes of attention, motivation and inhibitory control. It has undoubtedly facilitated the development of links between the study of attention as a behavioural manifestation of central processing of information and the neuroanatomical and neurotransmitter processes, providing the focus for the more recent research in the field of hyperactivity and attention problems.
The role of the environment

In the 1970s the trend against drugging children with behaviour disorders was accompanied by a number of alternative theories explaining the causation of hyperactivity. One such explanation, gaining longlasting popularity, was that of food allergy. Feingold (1975) suggested that children demonstrated hyperactive behaviour as a result of an allergic or toxic reaction to food substances, especially food additives. This theory caught the imagination of both professionals and lay people. Several studies were set up to investigate the concept; the more rigorous of these have found no or minimal effect of food substances on the behaviour of children.

Poor parenting as a causative factor in hyperactivity was put forward by both psychoanalysts (Bettelheim, 1973) and behaviourists (Willis and Lovaas, 1977). Psychoanalysis suggested that an excessively negative reaction from an intolerant mother to a child who demonstrates a negative or hyperactive temperament would lead to the clinical presentation of hyperactivity. According to behaviourists, noncompliant and hyperactive behaviour developed as a result of poor conditioning to stimulus control by parental commands and instructions. However, in the search for psychosocial causes, the finding which was of possibly greatest potential importance was that by Tizard and Hodges (1978), demonstrating an association between institutional upbringing and hyperactive behaviour. It suggested that a lack of continuity in parenting may impair the development of the normal modulation of activity and attention. The importance of disrupted core relationships for the development of hyperactive behaviour pattern was also highlighted by the observational study of Routh (1980), and the theoretical model of 'learned ineffectiveness' by Glow and Glow (1979). Other attempts by researchers and clinicians to come to terms with the new, rapidly spreading childhood disease were to seek explanations in wider social ecology (Gadow and Loney, 1981; Gittelman, 1981; Whalen and Henker, 1980a). Thus, causes were looked for in the various social problems of urban living, deteriorating schools and ever-expanding traffic, disposing to lead poisoning.

The 1970s also saw the beginning of a vigorous sociological and medicolegal debate about the implications of the increasingly popular diagnosis of hyperactivity. Block (1977), for example, suggested that technological development, leading to rapid cultural change, was responsible for children becoming hyperactive. The resulting increased excitement and environmental stimulation could in 'pre-disposed' children cause hyperactive behaviour of clinical severity. Conrad (1976), on the other hand, argued that the expansion of the clinical diagnosis stemmed at least partly from the existence of powerful drug
treatments for children's disruptive behaviours. The drugs, of course, had existed long before then, but their availability was convenient when physicians became increasingly confronted with referrals of children with puzzling troublesome behaviours, especially from schools. The issue was also seized by authors of more popular texts. In the USA, for example, journalists Schrag and Divoky (1975) published a book about the 'myth' of childhood hyperactivity and in the UK Steven Box (1977), in a widely read sociological journal, referred to the 'scandalous silence' surrounding the mass labelling and drugging of schoolchildren who failed to conform. Whalen and Henker (1980b) also drew attention to the apparent increased tolerance for diagnostic ambiguity and for a greater tendency to make the diagnosis of hyperactivity once there was evidence of the child's behaviour having improved by the drug treatment. Messinger (1975) went even further by arguing that pure profit-making motives were a major force. As drug companies were benefiting greatly from the sale of stimulants, they were also keen to promote any research likely to increase the detection of hyperactivity.

The issue of a syndrome

The question of whether hyperactivity constitutes a syndrome (O'Malley and Eisenberg, 1973), in the sense of sharing a unitary cluster of symptoms, a common cause in terms of major aetiological factors, a consistent response to treatment, a predictable natural course, and whether or not it differs from other disorders, especially conduct disorder, in terms of these factors, has been a difficult one to settle. At times, the argument has led to considerable polarization of views, with some seeing the whole issue as more of a hindrance to the progress of research (Ross and Ross, 1982), while others have regarded it as an invaluable stimulus for good study (Rutter, 1982).

The interest generated by the syndrome issue has led a small but determined group of investigators to search for empirical support for the disorder. This process has helped to sharpen both the theoretical reasoning behind it (Quay, 1979; Shaffer and Greenhill, 1979; Rie and Rie, 1980; Sandberg, 1981; Rutter, 1982) and, most notably, to improve the search for aetiological processes (Shaffer et al., 1974; Loney et al., 1978, 1981; Sandberg et al., 1978; Schachar et al., 1981; Taylor, 1983). The net result has been an appreciable scientific advancement starting from the early 1970s and which is likely to continue for some time. The scientific and clinical developments of the 1980s and 1990s are the subjects of the individual contributions to both editions of this book, and, therefore, outside the remit of this chapter.
of course, had physicians with puzzling also seized by Schrag and hyperactivity. I referred (dragging of 0b) also drew quity and for a there was ing treatment. sking motives on the sale of to increase the

REFERENCES


27 Historical development


Schachar, R.J. (1986). Hyperkinetic syndrome: historical development of the concept. In The...


Weatherman, J. and Benoit, E.P. (1962). Concrete and abstract thinking in organic and non-
Historical development


Psychiatry in Physical Medicine


Concern about symptoms is a major reason for patients to seek medical help. Many of the somatic symptoms that they present with—such as pain, weakness, and fatigue—remain unexplained by identifiable disease even after extensive medical assessment. Several general terms have been used to describe this problem—somatisation, somatoform, abnormal illness behaviour, medically unexplained symptoms, and functional symptoms. We will use the term functional symptoms, which does not assume psychogenesis but only a disturbance in bodily functioning.

Classification of functional syndromes

Most functional symptoms are transient, but a sizeable minority become persistent. Persistent symptoms are often multiple and disabling and may be described as functional syndromes. Although different medical and psychiatric classifications of functional syndromes exist, these are simply alternative ways of describing the same conditions.

Medical syndromes (such as fibromyalgia and chronic fatigue, chronic pain, and irritable bowel syndromes) highlight patterns of somatic symptoms, often in relation to particular bodily systems. Although they are useful in everyday medical practice, recent studies show there is substantial overlap between them.

Psychiatric syndromes (such as anxiety, depression, and somatoform disorders) highlight psychological processes and the number of somatic symptoms irrespective of the bodily system to which they refer. Depression and anxiety often present with somatic symptoms that may resolve with effective treatment of these disorders. In other cases the appropriate psychiatric diagnostic category is a somatoform disorder.

The existence of parallel classificatory systems is confusing. Both have merits, and both are imperfect. For many functional symptoms, a simple description of the symptom qualified with the descriptors single or multiple and acute or chronic may suffice. When diagnosis of a functional syndrome seems appropriate a combination of medical and psychiatric descriptors conveys the most information, such as “irritable bowel syndrome with anxiety disorder.”

A major obstacle to effective management is patients feeling disbelieved by their doctor. Patients who present with symptoms that are not associated with disease may be thought by some to be “putting it on.” The deliberate manufacture of symptoms or signs, however, is probably rare in ordinary practice.

Epidemiology

Community based studies report annual prevalences of 6-36% for individual troublesome symptoms. In primary care only a small proportion of patients presenting with such symptoms ever receive a specific disease diagnosis. The World Health Organization found functional symptoms to be common and disabling in primary care patients in all countries and cultures studied. Up to half of these patients remain disabled by their symptoms a year after presentation, the outcome being worse for those referred to secondary and tertiary care. The clinical and public health importance of functional symptoms has been greatly underestimated.
Causal factors

The cause of functional symptoms and syndromes is not fully understood, and it is therefore best to remain neutral regarding aetiological theories. In practice, functional symptoms are often attributed to single cause, which may be pathological (such as “a virus”) or psychological (such as “stress”). This simplistic and dualistic approach is unhelpful both in explaining the cause to a patient and in planning treatment. The available evidence suggests that biological, psychological, interpersonal, and healthcare factors are all potentially important.

The dualistic, single factor view has tended to emphasise psychological over biological factors, as exemplified by the commonly used term “somatisation.” However, recent evidence suggests that biological factors (especially reversible functional disturbance of the nervous system) are relevant to many functional syndromes, as they are to depression and anxiety disorders. A pragmatic doctor therefore asks not whether symptoms are “physical” or “mental” but whether they are fixed or are reversible by appropriate intervention.

The role of interpersonal factors in general, and of doctors and the health system in particular, in exacerbating functional symptoms has received less attention than it deserves. Raising fears of disease, performing unnecessary investigations and treatments, and encouraging disability are probably common adverse effects of medical consultations. However, denying the reality of patients’ symptoms may damage the doctor-patient relationship and drive patients from evidence based care into the arms of the unhelpful, unscientific, and unscrupulous.

Aetiological factors can also be usefully divided into the stage of illness at which they have their effect. That is, they may be predisposing, precipitating, or perpetuating. Predisposing and precipitating factors are useful in producing a fuller understanding of why a patient has the symptom, while perpetuating factors are the most important for treatment.

Precipitating factors—Symptoms may arise from an increased awareness of physiological changes associated with stress, depression, anxiety and sometimes disease and injury. They become important to the patients when they are severe and when they are associated with fears of, or belief in, disease.

Predisposing factors increase the chance that such symptoms will become important. Some people are probably biologically and psychologically predisposed to develop symptoms. Fear of disease may result from previous experience—for example, a middle aged man with a family history of heart disease is likely to become concerned about chest pain.

Perpetuating factors are those that make it more likely that symptoms and associated disability persists. Patients’ understandable attempts to alleviate their symptoms may paradoxically exacerbate them. For example, excessive rest to reduce pain or fatigue may contribute to disability in the longer term. Doctors may also contribute to this by failing to address patients’ concern or unwittingly increasing fear of disease (such as by excessive investigation). The provision of disability benefits can also be a financial disincentive for some patients to return to jobs they dislike, and the process of litigation may maintain a focus on disability rather than recovery.

Detection and diagnosis

Almost any symptom can occur in the absence of disease, but some, such as fatigue and subjective bloating, are more likely to be functional than others. Surprisingly, the more somatic symptoms a person has, the less likely it is that these symptoms reflect the presence of disease and the more likely there is associated depression and anxiety.
Patients with functional symptoms can be detected by maintaining an awareness of the problem when seeing new patients and by the use of somatic symptom questionnaires (large numbers of symptoms are more likely to be functional).

Management
Although it is essential to consider disease as the cause of the patient's symptoms an approach exclusively devoted to this can lead to difficulties if none is found. Making explicit from the start the possibility that the symptoms may turn out to be functional keeps the option of a wider discussion open. Even if more specialist treatment is needed, then the problem has, from the outset, been framed in a way that enables psychological treatment to be presented as part of continuing medical care rather than as an unacceptable and dismissive alternative. In this way it is possible to avoid an anxious disabled patient being treated by a bewildered frustrated doctor.

Investigation
An appropriate physical examination and necessary medically indicated investigation are clearly essential. Thereafter, before further investigation is done, the potential adverse psychological effect on the patient should be balanced against the likelihood and value of new information that may be obtained.

Reassurance and explanation
Most patients are reassured by being told that the symptoms they have are common and rarely associated with disease and that their doctor is familiar with them. This is especially so if accompanied by the promise of further review should the symptoms persist.

Reassurance needs to be used carefully, however. It is essential to elicit patients' specific concerns about their symptoms and to target reassurance appropriately. The simple repetition of bland reassurance that fails to address patients' fears is ineffective. If patients have severe anxiety about disease (hypochondriasis) repeated reassurance is not only ineffective but may even perpetuate the problem.

A positive explanation for symptoms is usually more helpful that a simple statement that there is no disease. Most patients will accept explanations that include psychological and social factors as well as physiological ones as long as the reality of symptom is accepted. The explanation can usefully show the link between these factors—for example, how anxiety can lead to physiological changes in the autonomic nervous system that cause somatic symptoms, which, if regarded as further evidence of disease, lead to more anxiety.

Further non-specialist treatment
A minority of patients need more than simple reassurance and explanation. Treatment should address patients' illness fears and beliefs, reduce anxiety and depression, and encourage a gradual return to normal activities.

There is good evidence that antidepressants often help, even when there are no clear symptoms of depression. Practical advice is needed, especially on coping effectively with symptoms and gradually returning to normal activity and work. Other useful interventions include help in dealing with major personal, family, or social difficulties and involving a close relative in management. Other members of the primary care or hospital team may be able to offer help with treatment, follow up, and practical help.

Referral for specialist treatment
There is always a temptation to refer difficult patients to another doctor. However, this can result in greater long term difficulties

Principles of assessment
- Identify patients' concerns and beliefs
- Review history of functional symptoms
- Explicitly consider both disease and functional diagnoses
- Appropriate medical assessment with explanation of findings
- Ask questions about patients' reaction to and coping with symptoms
- Use screening questions for psychiatric and social problems
- Consider interviewing relatives

Principles of treatment
- Explain that the symptoms are real and familiar to doctor
- Provide a positive explanation, including how behavioural, psychological, and emotional factors may exacerbate physiologically based somatic symptoms
- Offer opportunity for discussion of patient's and family's worries
- Give practical advice on coping with symptoms and encourage return to normal activity and work
- Identify and treat depression and anxiety disorders
- Discuss and agree a treatment plan
- Follow up and review

Non-specialist specific treatments
- Provide information and advice
- Agree a simple behavioural plan with patient and family
- Give advice about anxiety management
- Encourage use of diaries
- Advise about graded increase in activities
- Prescribe antidepressant drug
- Explain use of appropriate self help programmes

Specialist treatments
- Full and comprehensive assessment and explanation based on specialist assessment
- Cognitive behaviour therapy
- Supervised programme of graded increase in activity
- Antidepressants when these were previously not accepted or ineffective
- Illness specific interventions (such as rehabilitation programme for chronic pain)
if not carefully planned. When there is a good reason for further medical or psychiatric referral, then a clear explanation to the patient of the reason and an appropriately worded referral letter are essential.

Psychiatric treatments that may be required include more complex antidepressant drug regimens and specialist psychological interventions. Cognitive behaviour therapy has been shown to be effective in randomised controlled trials for a variety of functional syndromes (such as non-cardiac chest pain, irritable bowel, chronic pain, and chronic fatigue) and for patients with hypochondriasis.

Functional symptoms accompanying disease

Functional symptoms are also common in those who also have major disease. For example, after a heart attack or cardiac surgery, minor muscular chest aches and pains may be misinterpreted as evidence of angina, leading to unnecessary worry and disability. Explanation and advice, perhaps in the context of a cardiac rehabilitation programme, may make a substantial contribution to patients’ quality of life.

Conclusion

An understanding of the interaction of biological, psychological, interpersonal, and medical factors in the predisposition, precipitation, and perpetuation of functional somatic symptoms allows convincing explanations to provided for patients and effective treatment to be planned.

Important components of general management include effective initial reassurance, a positive explanation, and practical advice. It is also important to identify early those who are not responding and who require additional specific interventions. The difficulty that health systems have in effectively dealing with symptoms that are not attributable to disease reflects both intellectual and structural shortcomings in current care. The most salient of these is the continuing influence of mind-body dualism on our education and provision of care. In the longer term, scientific developments will break down this distinction. For the time being, it places primary care in a pivotal role in ensuring appropriate care for these patients.

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The ABC of psychological medicine is edited by Richard Mayou; Michael Sharpe, reader in psychological medicine, University of Edinburgh; and Alan Carson, consultant neuropsychiatrist, NHS Lothian, and honorary senior lecturer, University of Edinburgh. The series will be published as a book in winter 2002.

Evidence based summary points

- Functional somatic symptoms are common in primary care in all countries and cultures
- Cognitive behaviour therapies are of general applicability
- Antidepressants are of value whether or not patient is depressed


Further reading


the palms, the soles, and the axillae; thermal sweating is most evident on the forehead, the neck, the trunk, and the dorsum of the hands and the forearms. The sensitivity of the emotional rashes, blisters, and infections; therefore, hyperhidrosis may underlie several other dermatological conditions that are not primarily related to emotions. Basically, hyperhidrosis may be viewed as an anxiety phenomenon mediated by the autonomic nervous system; it must be differentiated from drug-induced states of hyperhidrosis.

**References**


### 28.3 Consultation-Liaison Psychiatry

In consultation-liaison (C-L) psychiatry, a rapidly growing area of expertise and an expanding field of concentration, psychiatrists serve as consultants to medical colleagues (either another psychiatrist or, more commonly, a nonpsychiatric physician) or to other mental health professionals (psychologist, social worker, or psychiatric nurse). In addition, C-L psychiatrists consult regarding patients in medical or surgical settings and provide follow-up psychiatric treatment as needed. C-L psychiatry is associated with all the diagnostic, therapeutic, research, and teaching services that psychiatrists perform in the general hospital and serves as a bridge between psychiatry and other specialties.

In the medical wards of the hospital, C-L psychiatrists must play many roles: skillful and brief interviewer, good psychiatrist and psychotherapist, teacher, and knowledgeable physician who understands the medical aspects of the case. The C-L psychiatrist must be viewed as a part of the medical team who makes a unique contribution to the patient’s total medical treatment.

### Diagnosis

Knowledge of psychiatric diagnosis is essential to C-L psychiatrists. Both dementia and delirium frequently complicate organic medical illness, especially among hospital patients. Psychoses and other mental disorders often complicate the treatment of medical illness, and deviant illness behavior, such as suicide, is a common problem in patients who are organically ill. C-L psychiatrists must be aware of the many medical illnesses that can have psychiatric symptoms. (A list of such medical problems is presented in Table 28.3-1.) Interviews and serial clinical observations are the C-L psychiatrist’s tools for diagnosis. The purposes of the diagnosis are to identify mental disorders and psychological responses to physical ill-
<table>
<thead>
<tr>
<th>Disease</th>
<th>Common Medical Symptoms</th>
<th>Psychiatric Symptoms and Complaints</th>
<th>Impaired Performance and Behavior</th>
<th>Laboratory Tests and Findings</th>
<th>Diagnostic Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperthyroidism (thyrotoxicosis)</td>
<td>Heat intolerance, Excessive sweating, Diarrhea, Weight loss, Tachycardia, Palpitations, Vomiting</td>
<td>Nervousness, Excitability, Irritability, Pressed speech, Insomnia, May express fear of impending death, Psychosis</td>
<td>Fine tremor, Impaired cognition, Decreased concentration, Hyperactivity, Intrusiveness</td>
<td>Free T4 increased, T3 increased, TSH decreased, T4 uptake decreased, ECG: Tachycardia, Atrial fibrillation, P and T wave changes</td>
<td>Full range of symptoms may not be present</td>
</tr>
<tr>
<td>Hypothyroidism (myxedema)</td>
<td>Cold intolerance, Dry skin, Constipation, Weight gain, Brittle hair, Goiter</td>
<td>Lethargy, Depressed affect, Personality change, Manic-like psychosis, Paraesthesia, Hallucinations</td>
<td>Muscle weakness, Decreased concentration, Psychomotor slowing, Apathy, Unusual sensitivity to barbiturates</td>
<td>TSH increased, TSH low if pituitary disease, Free T4 decreased, ECG: Bradycardia</td>
<td>More common in women, Associated with lithium carbonate therapy, Rule out pituitary disease, hypothalamic disease, major depressive disorder, bipolar I disorder</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>Sweating, Drowsiness, Stupor, Coma, Tachycardia</td>
<td>Anxiety, Confusion, Agitation</td>
<td>Tremor, Restlessness, Seizures</td>
<td>Hypoglycemia Tachycardia</td>
<td>Excess insulin often complicated by exercise, alcohol, decreased food intake</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>Polyuria, Anorexia, Nausea, Vomiting, Dehydration, Abdominal complaints</td>
<td>Anxiety, Agitation, Delirium</td>
<td>Acetone breath, Seizures</td>
<td>Hyperglycemia Serum ketones Urine ketones Anion gap acidosis</td>
<td>Almost always associated with brittle diabetes in young juvenile diabetics and elderly non-insulin-dependent diabetics, Rule out depressive disorders, anxiety disorders</td>
</tr>
<tr>
<td>Brain neoplasms</td>
<td>Headache, Vomiting, Papilledema, Focal findings on neurology examination</td>
<td>Personality changes</td>
<td>Lumbar puncture: increased CSF pressure, skull X-ray, CT scan, EEG</td>
<td></td>
<td>40-50% gliomas most common in 40-50-year age group, Corebellar tumors most common in children</td>
</tr>
<tr>
<td>Frontal lobe tumor</td>
<td></td>
<td>Mood changes, Irritability, Facelessness, Impaired judgment, Impaired memory, Delirium</td>
<td>Seizures Loss of speech Loss of smell</td>
<td>Angiogram: space-occupying lesion</td>
<td>Rule out intracranial abscess, aneurysm, subdural hematoma, seizure disorder, cerebrovascular disease, reactive depression, mania, schizophrenia, disorder, dementia</td>
</tr>
<tr>
<td>Parietal lobe tumor</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occipital lobe tumor</td>
<td>Headache, Papilledema, Homonymous hemianopsia</td>
<td>Aura, Visual hallucinations</td>
<td>Seizures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temporal lobe tumor</td>
<td>Contralateral homonymous field cut</td>
<td>Psychomotor seizures Aphasia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebellar tumor</td>
<td>Early evidence of increased intracranial pressure</td>
<td>Disturbed equilibrium Disturbed coordination</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head trauma</td>
<td>History or evidence of head trauma Headache Dizziness Bleeding from ear Altered level of consciousness Loss of consciousness Focal neurological findings</td>
<td>Confusion Personality changes Memory impairment</td>
<td>Seizures Paralysis</td>
<td>Lumbar puncture, skull X-rays, CT scan show evidence of bleeding, increased intracranial pressure Cerebral angiogram EEG</td>
<td>History of blow to head or bleeding confirms cause of ALS, Rule out cerebrovascular disease, seizure disorder, alcohol dependence, diabetes mellitus, hepatic encephalopathy, depression, dementia</td>
</tr>
</tbody>
</table>

(continued)
## Table 28.3-1 (continued)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Common Medical Symptoms</th>
<th>Psychiatric Symptoms and Complaints</th>
<th>Impaired Performance and Behavior</th>
<th>Laboratory Tests and Findings</th>
<th>Diagnostic Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIDS</td>
<td>Fever</td>
<td>Progressive dementia</td>
<td>Impaired memory</td>
<td>HIV testing CT, MRI, lumbar puncture, CSF, and blood cultures</td>
<td>60% of patients have neuropsychiatric symptoms; always consider in high-risk populations and young patients with signs of dementia</td>
</tr>
<tr>
<td></td>
<td>Weight loss</td>
<td>Personality changes</td>
<td>Decreased concentration</td>
<td>Seizures</td>
<td>Rule out other infections, brain neoplasms, dementia, depression, schizophreniform disorder</td>
</tr>
<tr>
<td></td>
<td>Ataxia</td>
<td>Depression</td>
<td></td>
<td></td>
<td>Suicidal behavior is a symptom of underlying psychiatric illness</td>
</tr>
<tr>
<td></td>
<td>Incontinence</td>
<td>Loss of libido</td>
<td></td>
<td></td>
<td>Knowledge of risk factors is helpful but not a substitute for good clinical judgment</td>
</tr>
<tr>
<td></td>
<td>Focal findings on neurological examination</td>
<td>Psychosis</td>
<td></td>
<td></td>
<td>Prediction is best done through assessment of current risk projected into the immediate future</td>
</tr>
<tr>
<td>Injuries requiring ambulatory surgical evaluation and treatment (for example, wrist slashing)</td>
<td>Alcohol abuse and other substance abuse</td>
<td>90% have major psychiatric disease</td>
<td>Frequent accidents</td>
<td></td>
<td>Caused by excessive free water for level of total body Na⁺</td>
</tr>
<tr>
<td></td>
<td>Recent surgery</td>
<td>History of prior suicide attempts</td>
<td>Repeated emergency room visits</td>
<td></td>
<td>Often abnormal SIADH</td>
</tr>
<tr>
<td></td>
<td>Chronic pain</td>
<td>Depressed mood</td>
<td>Eager to leave emergency room</td>
<td></td>
<td>May be psychogenic</td>
</tr>
<tr>
<td></td>
<td>Chronic illness</td>
<td>Postpartum psychosis in women</td>
<td>before full evaluation</td>
<td></td>
<td>Rule out nephrotic syndrome, liver disease, congestive heart failure, schizophreniform disorder, schizotypal personality disorder</td>
</tr>
<tr>
<td></td>
<td>Terminal illness</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyponatremia</td>
<td>Excessive thirst</td>
<td>Confusion</td>
<td>Seizures</td>
<td>Decreased serum Na⁺</td>
<td>Caused by excessive free water for level of total body Na⁺</td>
</tr>
<tr>
<td></td>
<td>Polydipsia</td>
<td>Lethargy</td>
<td>Speech abnormalities</td>
<td>Serum Na⁺ and osmolalities to document syndrome of inappropriate secretion of antidiuretic hormone (SIADH)</td>
<td>Often abnormal SIADH</td>
</tr>
<tr>
<td></td>
<td>Stupor</td>
<td>Personality changes</td>
<td></td>
<td></td>
<td>May be psychogenic</td>
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<tr>
<td></td>
<td>Coma</td>
<td></td>
<td></td>
<td></td>
<td>Rule out nephrotic syndrome, liver disease, congestive heart failure, schizophreniform disorder, schizotypal personality disorder</td>
</tr>
<tr>
<td>Pancreatic carcinoma</td>
<td>Weight loss</td>
<td>Depression</td>
<td>Apathy</td>
<td>Elevated amylase</td>
<td>Always consider in depressed middle-aged patients</td>
</tr>
<tr>
<td></td>
<td>Abdominal pain</td>
<td>Lethargy</td>
<td>Decreased energy</td>
<td></td>
<td>Rule out other GI illness, major depressive disorder</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anxiety</td>
<td></td>
<td></td>
<td>Must distinguish other causes—for example, cancer from exogenous steroid excess</td>
</tr>
<tr>
<td>Cushing’s syndrome</td>
<td>Central obesity</td>
<td>Depression</td>
<td>Disturbed sleep</td>
<td>Elevated blood pressure</td>
<td>Suicide rate in untreated cases is about 10%</td>
</tr>
<tr>
<td></td>
<td>Purple striae</td>
<td>Insomnia</td>
<td>Decreased energy</td>
<td>Poor glucose tolerance</td>
<td>Rule out major depressive disorder, bipolar I disorder</td>
</tr>
<tr>
<td></td>
<td>Easy bruising</td>
<td>Emotional liability</td>
<td>Agitation</td>
<td>Dexamethasone-suppression test (may be falsely positive)</td>
<td>May be primary (Addison’s disease) or secondary</td>
</tr>
<tr>
<td></td>
<td>Osteoporosis</td>
<td>Suicidality</td>
<td>Difficulty in concentrating</td>
<td></td>
<td>Rule out eating disorders, mood disorders</td>
</tr>
<tr>
<td></td>
<td>Proximal muscle weakness</td>
<td>Euphoria</td>
<td></td>
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<td></td>
<td>Hirsutism</td>
<td>Mania</td>
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<td></td>
<td></td>
<td>Psychosis</td>
<td></td>
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<tr>
<td>Adrenocortical insufficiency</td>
<td>Nausea</td>
<td>Lethargy</td>
<td>Fatigue</td>
<td>Decreased blood pressure</td>
<td></td>
</tr>
<tr>
<td>(Addison’s disease)</td>
<td>Vomiting</td>
<td>Depression</td>
<td></td>
<td>Increased Na⁺</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anorexia</td>
<td>Psychois</td>
<td></td>
<td>Eosinophilia</td>
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<td></td>
<td>Stupor</td>
<td>Ddelirium</td>
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<td></td>
<td>Coma</td>
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<tr>
<td>Seizure disorder</td>
<td>Hyperpigmentation</td>
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<td></td>
<td>Sensory distortions</td>
<td>Confusion</td>
<td>Violence</td>
<td>EEG, including NP leads</td>
<td>Consider complex partial seizures in all dissociative states</td>
</tr>
<tr>
<td></td>
<td>Aura</td>
<td>Psychosis</td>
<td>Motor automatisms</td>
<td></td>
<td>Rule out postictal states, catatonic schizophrenia</td>
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<tr>
<td></td>
<td></td>
<td>Dissociative states</td>
<td>Belligerence</td>
<td></td>
<td>Causes hypercalcemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Catatonic-like state</td>
<td>Bizarre behavior</td>
<td></td>
<td>Rule out major depressive disorder, schizoaffective disorder</td>
</tr>
<tr>
<td>Hyperparathyroidism</td>
<td>Constipation</td>
<td>Depression</td>
<td>Increased Ca²⁺</td>
<td></td>
<td>Causes hypocalcemia</td>
</tr>
<tr>
<td></td>
<td>Polydipsia</td>
<td>Paranoia</td>
<td>PTH variable</td>
<td></td>
<td>Rule out anxiety disorders, mood disorders</td>
</tr>
<tr>
<td></td>
<td>Nausea</td>
<td>Confusion</td>
<td>ECG: shortened QT interval</td>
<td></td>
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<tr>
<td>Hypoparathyroidism</td>
<td>Headache</td>
<td>Anxiety</td>
<td>Impaired memory</td>
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<tr>
<td></td>
<td>Paresthesias</td>
<td>Agitation</td>
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<td></td>
<td>Tetany</td>
<td>Depression</td>
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<td></td>
<td>Carpopedal spasm</td>
<td>Confusion</td>
<td></td>
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<tr>
<td></td>
<td>Laryngeal spasm</td>
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<tr>
<td></td>
<td>Abdominal pain</td>
<td></td>
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</tbody>
</table>
### Table 28.3-1 (continued)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Common Medical Symptoms</th>
<th>Psychiatric Symptoms and Complaints</th>
<th>Impaired Performance and Behavior</th>
<th>Laboratory Tests and Findings</th>
<th>Diagnostic Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic lupus erythematosus</td>
<td>Fever, Photosensitivity, Butterfly rash, Joint pains, Headache</td>
<td>Depression, Mood disturbances, Psychosis, Delusions, Hallucinations</td>
<td>Fatigue</td>
<td>Positive ANA, Positive lupus erythematosus test, Anemia, Thrombocytopenia, Chest X-ray: pleural effusion, pericarditis</td>
<td>Multisystemic autoimmune disease most frequent in women, Psychiatric symptoms are present in 50% of cases, Steroid treatment can cause psychiatric symptoms, Rule out depressive disorders, paranoid psychosis, psychiatric mood disorder</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>Sudden transient motor and sensory disturbances, Impaired vision, Diffuse neurological signs with remissions and exacerbations</td>
<td>Anxiety, Euphoria, Mania</td>
<td>Slurred speech, Incontinence</td>
<td>CSF may show increased gamma globulin, CT: degenerative patches in brain and spinal cord</td>
<td>Onset usually in young adults, Rule out tertiary syphilis, other degenerative diseases, hysteria, mania (late)</td>
</tr>
<tr>
<td>Acute intermittent porphyria</td>
<td>Abdominal pain, Fever, Nausea, Vomiting, Constipation, Peripheral neuropathy, Paralysis</td>
<td>Acute depression, Agitation, Paranoia, Visual hallucinations</td>
<td>Restlessness, Diaphoresis, Weakness</td>
<td>Abnormal liver function test results, Abnormal albumin</td>
<td>Autosomal dominant, More common in women in the 20–40 age group, May be precipitated by a variety of drugs, Rule out acute abdominal disease, acute psychiatric episode, schizophreniform disorder, major depressive disorder</td>
</tr>
<tr>
<td>Hepatic encephalopathy</td>
<td>Asthenia, Hyperreflexia, Spider angioma, Palmar erythema, Ecchymoses, Liver enlargement and atrophy</td>
<td>Euphoria, Disinhibition, Psychosis, Depression</td>
<td>Restlessness, Decreased activities of daily living (ADL), Impaired cognition, Impaired concentration, Ataxia, Dysarthria, Remain at great risk for suicide</td>
<td>Abnormal liver function test results, Abnormal albumin</td>
<td>May be acute or chronic depending on cause, Rule out substance intoxication, mania, depressive disorder, dementia</td>
</tr>
<tr>
<td>Injuries requiring inpatient surgical evaluation and treatment (for example, suicide attempts, self-mutilation)</td>
<td>Alcohol abuse and other substance abuse, Serious injury, Major blood loss, Damage to genitals, eyes, face, etc.</td>
<td>99% have severe psychiatric disease associated with psychosis, psychotic depression, impaired mental status secondary to substance intoxication, Bizarre, inappropriate affect</td>
<td></td>
<td></td>
<td>Must assess and treat the underlying psychiatric condition on a priority basis, Maintain a high index of suspicion for suicide risk</td>
</tr>
<tr>
<td>Pheochromocytoma</td>
<td>Paroxysmal hypertension, Headache</td>
<td>Anxiety, Apprehension, Feeling of impending doom</td>
<td>Panic, Diaphoresis, Tremor</td>
<td>Hypertension, Elevated VMA in 24-hr. urine, Tachycardia</td>
<td>Adrenal medulla secreting catecholamines, Rule out anxiety disorders</td>
</tr>
<tr>
<td>Wilson's disease</td>
<td>Kayser-Fleischer corneal ring, Hepatitis-like picture</td>
<td>Mood disturbances, Delusions, Hallucinations</td>
<td>Choreoathetoid movements, Gait disturbance, Clumsiness, Rigidity</td>
<td>Decreased serum ceruloplasmin, Increased copper in urine</td>
<td>Hepatolenticular degeneration, Autosomal recessive disorder of copper metabolism, Often presents in adolescence, early adulthood, Rule out extrapyramidal reactions, schizophreniform disorder, mood disorders, Autosomal dominant, Rule out mood disorders, mania, schizophrenia</td>
</tr>
<tr>
<td>Huntington's disease</td>
<td>Family history</td>
<td>Depression, Euphoria</td>
<td>R rigidity, Choreoathetoid movements</td>
<td>Low thiamine level</td>
<td>Most common in alcoholic persons, Rule out hypomania, depressive disorder, dementia</td>
</tr>
<tr>
<td>Vitamin deficiencies</td>
<td>Neuropathy, Cardiomyopathy, Wernicke-Korsakoff syndrome, Nystagmus, Headache, Amnesia</td>
<td>Confusion, Confabulation</td>
<td>General malaise, Inability to sustain a conversation, Poor concentration</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(continued)
Table 28.3-1 (continued)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Common Medical Symptoms</th>
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<th>Impaired Performance and Behavior</th>
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<th>Diagnostic Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin deficiencies—continued</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nicotinamide</td>
<td>Diarrhea, Stocking-glove dermatitis</td>
<td>Confusion, Irritability, Insomnia, Depression, Psychosis, Dementia</td>
<td>Memory disturbances</td>
<td></td>
<td>Rule out mood disorders, mania, schizophrenia, mood disorder, dementia</td>
</tr>
<tr>
<td>Pyridoxine</td>
<td>Paller, Dizziness, Peripheral neuropathy, Dorsal column signs</td>
<td>Irritability, Inattentiveness, Psychosis, Dementia</td>
<td>Fatigue, Ataxia</td>
<td>Low B12 level, Schilling test, Megaloblastic anemia</td>
<td>Rule out pernicious anemia, mania, mood disorders</td>
</tr>
<tr>
<td>Vitamin B12</td>
<td>Skull lesions, Leukopenia, Periostitis, Arthritis, Respiratory distress, Progressive cardiovascular distress</td>
<td>Personality changes, Irritability, Confusion, Psychosis</td>
<td>Irresponsible behavior, Decreased attention to activities of daily living (ADL)</td>
<td>VDRL, Treponema antibody, CSF abnormal</td>
<td>Rule out neoplasias, meningitis, demencia, psychiatric mood disorder, schizophrenia</td>
</tr>
</tbody>
</table>

ness, identify patients' personality features, and identify patients' characteristic coping techniques to recommend the most appropriate therapeutic intervention for patients' needs.

TREATMENT

C-L psychiatrists' principal contribution to medical treatment is a comprehensive analysis of a patient's response to illness, psychological and social resources, coping style, and psychiatric illness, if any. This assessment is the basis of the patient treatment plan. In discussing the plan, C-L psychiatrists provide their patient assessment to nonpsychiatric health professionals. Psychiatrists' recommendations should be clear, concrete guidelines for action. A C-L psychiatrist may recommend a specific therapy, suggest areas for further medical inquiry, inform doctors and nurses of their roles in the patient's psychosocial care, recommend a transfer to a psychiatric facility for long-term psychiatric treatment, or suggest or undertake brief psychotherapy with the patient on the medical ward.

C-L psychiatrists must deal with a broad range of problems. Studies show that up to 65 percent of medical inpatients have psychiatric disorders, the most common symptoms being anxiety, depression, and disorientation. Treatment problems account for 50 percent of the consultation requests made of C-L psychiatrists. (Table 28.3-2 covers the most common C-L problems.)

SPECIAL SETTINGS

Intensive Care Units

The central psychological aspect of patients in intensive care units (ICUs) is that they are suffering life-threatening illnesses with psychological responses that are predictable and that, if untreated, may threaten life or recovery. Coronary and medical ICU staff members see patients' reactions to acute unexpected illnesses. Patients first show fear and anxiety, followed by the psychological behaviors associated with denial, such as acting out, signing out, hostility, and excessive dependence. Staff members working in burn units encounter patients going through the problems of acute unexpected illness and, later, depression, grief, and dissociation related to pain and disfigurement. Staff members in surgical ICUs see patients recovering from major surgery with the expected disorientation of delirium, depression, and adjustment reactions to surgery. Treatment of the psychological problems in ICUs requires close attention to diagnostic possibilities and details of the environment, as well as careful team communication. Clinicians are clearly helped by familiarity with patients' premorbid character, because the reactions to disease and illness are influenced by previous conditioning. The most common initial reactions to medical disasters include shock, fear, and anxiety. In many patients these reactions respond to treatment by the care team, especially succinct, authoritative, and consistent reassurance. When these measures are insufficient, benzodiazepines—preferably the short-acting forms—should be considered and used cautiously. When fear leads to panic or psychotic loss of control, fast-acting antipsychotics—for example, haloperidol (Haldol)—should be used.

Denial and associated behaviors of acting out, hostility, dependence, and demanding behavior must be dealt with individually on the basis of knowledge of patients and the reasons for their reactions. Several general points are pertinent. Direct communication with patients, which allows but does not force a discussion of feelings, often eliminates disruptive behaviors without dealing with them directly. Allowing patients as much mastery as they want and can handle is the most reassuring approach.

Reason for ICU

Suicide attempt

Depression

Agitation

Hallucinations

Sleep disturbance

No organ failure

Disorientation

Noncompliance

Consent to treatment
## Table 28.3-2
### Common Consultation-Liaison Problems

<table>
<thead>
<tr>
<th>Reason for Consultation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suicide attempt or threat</td>
<td>High-risk factors are men over 45, no social support, alcohol dependence, previous attempt, incapacitating medical illness with pain, and suicidal ideation. If risk is present, transfer to psychiatric unit or start 24-hour nursing care.</td>
</tr>
<tr>
<td>Depression</td>
<td>Suicidal risks must be assessed in every depressed patient (see above); presence of cognitive defects in depression may cause diagnostic dilemma with dementia; check for history of substance abuse or depressant drugs (eg, reserpine, propranolol); use antidepressants cautiously in cardiac patients because of conduction side effects, orthostatic hypotension.</td>
</tr>
<tr>
<td>Agitation</td>
<td>Often related to cognitive disorder, withdrawal from drugs, (eg, opioids, alcohol, sedative-hypnotics); haloperidol most useful drug for excessive agitation; use physical restraints with great caution; examine for command hallucinations or paranoid ideation to which patient is responding in agitated manner; rule out toxic reaction to medication.</td>
</tr>
<tr>
<td>Hallucinations</td>
<td>Most common cause in hospital is delirium tremens; onset three to four days after hospitalization. In intensive care units, check for sensory isolation; rule out brief psychotic disorder, schizophrenia, cognitive disorder. Treat with antipsychotic medication.</td>
</tr>
<tr>
<td>Sleep disorder</td>
<td>Common cause is pain; early morning awakening associated with depression; difficulty in falling asleep associated with anxiety. Use antianxiety or antidepressant agent, depending on cause. Those drugs have no analgesic effect, so prescribe adequate painkillers. Rule out early substance withdrawal.</td>
</tr>
<tr>
<td>No organic basis for symptoms</td>
<td>Rule out conversion disorder, somatization disorder, factitious disorder, and malingering; glove and stocking anesthesia with autonomic nervous system symptoms seen in conversion disorder; multiple body complaints seen in somatization disorder; wish to be hospitalized seen in factitious disorder; obvious secondary gain in malingering (eg, compensation case).</td>
</tr>
<tr>
<td>Disorientation</td>
<td>Delirium versus dementia; review metabolic status, neurological findings, substance history. Prescribe small dose of antipsychotics for major agitation; benzodiazepines may worsen condition and cause sundowner syndrome (ataxia, confusion); modify environment so patient does not experience sensory deprivation.</td>
</tr>
<tr>
<td>Noncompliance or refusal to consent to procedure</td>
<td>Explore relationship of patient and treating doctor; negative transference is most common cause of noncompliance; fears of medication or of procedure require education and reassurance. Refusal to give consent is issue of judgment; if impaired, patient can be declared incompetent, but only by a judge; cognitive disorder is main cause of impaired judgment in hospitalized patients.</td>
</tr>
</tbody>
</table>

approach. Permitting patients to make small choices restores some sense of control over the self and the future, gives them a symbolic sense of progress, and calms them far beyond the meaning of the specific choices. For example, allowing patients to control pain medications, the level of lighting, or the place where they sit reassures, and relaxes them. Whether the disruptive behavior is hostility, dependence, or panic, allowing some behavior to be shown while setting limits of their extremes reassures patients. Thus, an independent patient can be allowed to move around but not too far; a dependent patient can make a limited number of interactions, such as using the call button; and a hostile patient can be permitted some disagreement and ventilation but be limited in disruptive acts.

All ICUs deal mainly with anxiety, depression, and delirium. ICUs also impose extraordinarily high stress, on staff and patients, related to the intensity of the problems. Patients and staff members alike frequently observe cardiac arrests, deaths, and medical disasters, which leave all automatically aroused and psychologically defensive. ICU nurses and their patients experience particularly high levels of anxiety and depression. As a result, nurse burnout and high turnover rates are very common.

Much attention is paid to the problem of stress among ICU staff, especially in nursing literature. Much less attention is given to the house staff, especially those on the surgical services. All people in ICUs must to be able to deal directly with their feelings about their extraordinary experiences and difficult emotional and physical circumstances. Regular support groups in which people can discuss their feelings are important to the ICU staff and the house staff. Such support groups protect staff members from the otherwise predictable psychiatric morbidity that some may experience, and protect their patients from the loss of concentration, decreased energy, and psychomotor-retarded communications that some staff members otherwise exhibit.

### Hemodialysis Units

Hemodialysis units present a paradigm of complex modern medical treatment settings. Patients are coping with lifelong, debilitating, and limiting disease; they are totally dependent on a multiplex group of caretakers for access to a machine controlling their well-being. Dialysis is scheduled 3 times a week and takes 4 to 6 hours; thus, it disrupts patients' previous living routines.

In this context, patients first and foremost fight the disease. Invariably, however, they also have to come to terms with a level of dependence on others, a dependence probably not experienced since childhood. Predictably, patients entering dialysis struggle for their independence; regress to childhood.
<table>
<thead>
<tr>
<th>Organ</th>
<th>Biological Factors</th>
<th>Psychological Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kidney</td>
<td>50% to 90% success rate; may not be done if patient is over age 55; increasing use of cadaver kidneys, rather than those from living donors</td>
<td>Living donors must be emotionally stable; parents are best donors, siblings may be ambivalent; donors are subject to depression. Patients who panic before surgery may have poor prognoses; altered body image with fear of organ rejection is common. Group therapy for patients is helpful.</td>
</tr>
<tr>
<td>Bone marrow</td>
<td>Used in aplastic anemias and immune system disease</td>
<td>Patients are usually ill and must deal with death and dying; compliance is important. The procedure is commonly done in children who present problems of prolonged dependence; siblings are often donors and may be angry or ambivalent about procedure.</td>
</tr>
<tr>
<td>Heart</td>
<td>End-stage coronary artery disease and cardiomyopathy</td>
<td>Donor is legally dead; relatives of the deceased may refuse permission or be ambivalent. No fall-back position is available if the organ is rejected; kidney rejection patient can go on hemodialysis. Some patients seek transplantation hoping to die. Postcardiotomy delirium is seen in 25% of patients.</td>
</tr>
<tr>
<td>Breast</td>
<td>Radical mastectomy versus lumpectomy</td>
<td>Reconstruction of breast at time of surgery leads to postoperative adaptation; veteran patients are used to counsel new patients; lumpectomy patients are more open about surgery and sex than are mastectomy patients; group support is helpful.</td>
</tr>
<tr>
<td>Uterus</td>
<td>Hysterectomy performed on 10% of women over 20</td>
<td>Fear of loss of sexual attractiveness with sexual dysfunction may occur in a small percentage of women; loss of child-bearing capacity is upsetting.</td>
</tr>
<tr>
<td>Brain</td>
<td>Anatomical location of lesion determines behavioral change</td>
<td>Environmental dependence syndrome in frontal lobe tumors is characterized by inability to show initiative; memory disturbances are involved in periventricular surgery; hallucinations are involved in parieto-occipital area.</td>
</tr>
<tr>
<td>Prostate</td>
<td>Cancer surgery has more negative psychobiological effects and is more technically difficult than is surgery for benign hypertrophy</td>
<td>Sexual dysfunction is common except in transurethral prostatectomy. Perineal prostatectomy produces the absence of emission, ejaculation, and erection; penile implant may be of use.</td>
</tr>
<tr>
<td>Colon and rectum</td>
<td>Colostomy and ostomy are common outcomes, especially for cancer</td>
<td>One third of patients with colostomies feel worse about themselves than before bowel surgery; shame and self-consciousness about the stoma can be alleviated by self-help groups that deal with those issues.</td>
</tr>
<tr>
<td>Limbs</td>
<td>Amputation performed for massive injury, diabetes, or cancer</td>
<td>Phantom-limb phenomenon occurs in 98% of cases; the experience may last for years; sometimes the sensation is painful, and neuroma at the stump should be ruled out; the condition has no known cause or treatment; it may stop spontaneously.</td>
</tr>
</tbody>
</table>

states; show denial by acting out against doctor's orders, by breaking their diet, or by missing sessions; show anger directed against staff members; bargain and plead or become infantilized and obsequious; but most often are accepting and courageous. The determinants of patients' responses to entering dialysis include personality styles and previous experiences with this or another chronic illness. Patients who have had time to react and adapt to their chronic renal failure face less new psychological work of adaptation than do those with recent renal failure and machine dependence.

Although little has been written about social factors, the effect of cultural factors in reaction to dialysis and the management of the dialysis unit are known to be important. Units run with a firm hand, that are consistent in dealing with patients, have clear contingencies for behavioral failures, and have adequate psychological support for staff members tend to produce the best results.

Complications of dialysis treatment can include psychiatric problems such as depression, and suicide is not rare. Sexual problems can be neurogenic, psychogenic, or related to gonadal dysfunction and testicular atrophy. Dialysis dementia is a rare condition that evidences loss of memory, disorientation, dystonias, and seizures. The disorder occurs in patients who have been receiving dialysis treatment for many years. The cause is unknown.

The psychological treatment of dialysis patients falls into two areas. First, careful preparation before dialysis, including the work of adaptation to chronic illness, is important, especially in dealing with denial and unrealistic expectations. All predialysis patients should have a psychosocial evaluation.
Second, once in a dialysis program, patients need periodic specific inquiries about adaptation that do not encourage dependence or the sick role. Staff members should be sensitive to the likelihood of depression and sexual problems. Group sessions function well for support, and patient self-help groups restore a useful social network, self-esteem, and self-mastery. When needed, tricyclic drugs or phenothiazines can be used for dialysis patients. Psychiatric care is most effective when brief and problem-oriented.

The use of home dialysis units has improved treatment attitude. Compared with hospital-treated patients, home-treated patients are better able to integrate the treatment into their daily lives and feel more autonomous and less dependent on others for their care.

Surgical Units

Some surgeons believe that patients who expect to die during surgery will do so. This belief now seems less superstitious than it once did. Chase Patterson Kimball and others have studied the premorbid psychological adjustment of patients scheduled for surgery and have shown that those who show evident depression or anxiety and deny it have a higher risk for morbidity and mortality than do those who, given similar depression or anxiety, can express it. Even better results occur in those with a positive attitude toward impending surgery. The factors that contribute to an improved outcome for surgery are informed consent, the education of patients so that they know what to expect, what tubes and gadgets will be in place, and how to cope with the anticipated pain. In cases in which the patients will not be able to talk or see, it is helpful to explain before surgery what they can do to compensate for these losses. If postoperative states such as confusion, delirium, and pain can be predicted, they should be discussed with patients in advance to avoid their experiencing them as unwarranted or as signs of danger. Constructive family support members can help both before and after surgery. Table 28.3–3 lists various surgical conditions with which C-L psychiatrists must deal.

References


Cohen-Cole SA, Pincus HA: Stoudemire A, Fiester S, Houpt JL: Recent research on the premorbid psychological adjustment of patients scheduled for surgery and have shown that those who show evident depression or anxiety and deny it have a higher risk for morbidity and mortality than do those who, given similar depression or anxiety, can express it. Even better results occur in those with a positive attitude toward impending surgery. The factors that contribute to an improved outcome for surgery are informed consent, the education of patients so that they know what to expect, what tubes and gadgets will be in place, and how to cope with the anticipated pain. In cases in which the patients will not be able to talk or see, it is helpful to explain before surgery what they can do to compensate for these losses. If postoperative states such as confusion, delirium, and pain can be predicted, they should be discussed with patients in advance to avoid their experiencing them as unwarranted or as signs of danger. Constructive family support members can help both before and after surgery. Table 28.3–3 lists various surgical conditions with which C-L psychiatrists must deal.

The concept of combined psychotherapeutic and medical treatment—that is, the approach that emphasizes the interrelation of mind and body in the genesis of symptom and disorder—calls for a greatly expanded sharing of responsibility among various professions. From a multicausal point of view, every disease can be considered to be caused by or to be associated with emotional factors. The evaluation of all these factors is usually carried out by the primary care physician, who may need the participation of the psychiatrist to explain fully the psychological factors.

Hostility, rage, guilt, depression, and anxiety in varying proportions are at the root of most psychosomatic disorders. Psychosomatic medicine is principally concerned with illnesses that present primarily somatic manifestations. The pre-
senting complaint is usually physical; patients rarely complain of their anxiety or depression or tension but, rather, of their vomiting or diarrhea or anorexia.

**TYPES OF PATIENTS**

A special evaluation of the psychological and somatic factors of three major groups of medical patients is required.

**Psychosomatic Illness Group**

Patients in the psychosomatic illness group have classic psychosomatic disorders, such as peptic ulcer and ulcerative colitis. In these disease processes clinicians cannot posit a strictly psychogenic explanation; the particular set of emotional factors found, for example, in the typical ulcer cases may also appear in patients with no history of ulcer. There are changes in the autonomic nervous system, however, that cause pathophysiology to occur (for example, vasospasm causing muscle pain).

**Psychiatric Group**

Patients in the psychiatric group experience physical disturbances caused by psychological rather than physical illness. As mentioned above, when the illness is real, the disability involves the autonomic nervous system, which causes pathophysiological changes. There are no pathophysiological signs in hypochondria and delusional preoccupation with physical functioning. Patients in this group suffer primarily from a psychological disturbance that requires psychiatric treatment, but auxiliary medical therapy may be necessary. Patients with conversion disorders (for example, paralysis) show no objective changes consistent with a known disease. In conversion disorder the voluntary nervous system is involved, not the autonomic nervous system.

**Reactive Group**

Patients in the reactive group have actual organic disorders, but they also suffer from an associated psychological disturbance. For example, a patient with heart disease or one with renal disease who requires dialysis may have anxiety and depression in response to the life-threatening condition. This anxiety, in turn, may produce physical manifestations that complicate the somatic situation.

**COMBINED TREATMENT**

The combined treatment approach, in which a psychiatrist handles the psychiatric aspects of the case and internist or other specialist treats the somatic aspects, requires the closest collaboration between the two physicians. The purpose of the medical therapy is to build up the patient's physical state so that the patient can successfully participate in psychotherapy for a total cure.

Disorders such as bronchial asthma, in which psychosocial processes play a distinct role in the development and course, may respond well to the combined treatment approach. Although the asthmatic attacks themselves may be treated successfully by the internist, psychiatric treatment can be useful in the short run by helping to alleviate the anxiety associated with the attacks and in the long run by helping to uncover the causes of the interdependence involved in the disorder.

In an acute somatic illness, such as an acute attack of ulcerative colitis, medical therapy is the primary form of treatment; at this stage, psychotherapy, with its long-range goals, consists of reassurance and support. As the pendulum of disease activity shifts and the illness becomes chronic, psychotherapy assumes the primary role, and medical therapy takes the less active position.

Sometimes, reassurance is all that is needed in the treatment of psychosomatic syndromes. Patients must participate in the process of improving their life situations. The symptoms themselves may be treated by the internist, if necessary. Usually, the psychiatrist can help patients focus on their feelings about the symptoms and gain understanding of the unconscious processes involved with symptom improvement. If patients are handled insensitively or if their illnesses are regarded unsympathetically, the results can be grave.

**Indications for Combined Treatment**

If during an initial attack of a psychosomatic disorder patients respond to active medical therapy in association with the superficial support, ventilation, reassurance, and environmental manipulation provided by an internist, additional psychotherapy by a psychiatrist may not be required. Psychosomatic illness that is chronic or does not respond to medical treatment should receive psychosomatic evaluation by a psychiatrist, and combined therapy as indicated.

**Goal of Treatment**

It is useful to set up a tentative, flexible spectrum of therapeutic goals in the treatment of psychosomatic disorders. The desired end is a cure, which means resolution of any structural impairment and reorganization of the personality so that needs and tensions no longer produce pathophysiological results. Treatment should aim at a mature general life adjustment, increased capacity for physical and occupational activity, amelioration of the progression of the disease, reversal of the pathology, avoidance of complications of the basic disease process, decreased use of secondary gain associated with the illness, and increased capacity to adjust to the presence of the disease.

**PSYCHIATRIC ASPECTS**

Treatment of psychosomatic disorders from a psychiatric viewpoint is a difficult task. Psychiatrists must focus therapy on understanding the motivations and mechanisms of disturbed functioning and helping patients realize the nature of their illness and the implications of its costly adaptive patterns. These insights should produce changed and healthier patterns of behavior.

Psychotherapy based on analytic principles is effective in treating psychosomatic disorders mainly in terms of the patients' experiences in the treatment, particularly their relationships with the therapist. Patients with psychosomatic disorders
are usually even more reluctant to deal with their emotional problems than are patients with other psychiatric problems. Psychosomatic patients try to avoid responsibility for their illness by isolating the diseased organ and presenting it to the doctor for diagnosis and cure. They may be satisfying an infantile need to be cared for passively, while denying that they are adults, with all the attendant stresses and conflicts.

Resistance to Entering Psychotherapy

When psychosomatic patients first become ill, they are usually convinced that the illness is purely organic in origin. They reject psychotherapy as treatment for their sickness; in fact, the very idea of emotional illness may be repugnant because of personal prejudices about psychiatry.

In the initial phase, physical treatment and psychotherapeutic procedures must be combined subtly. In this stage, treatment by a psychologically oriented physician who is sensitive to unconscious and transference phenomena can be therapeutic.

Development of Relationship and Transference

Psychotherapy with psychosomatic patients must often proceed more slowly and cautiously than with other psychiatric patients. Positive transference should be developed gradually, and psychiatrists must be supportive and reassuring during the acute illness. As disorders become chronic, a psychiatrist may make exploratory interpretations, but a strong patient-physician relationship must precede any such exploration. As psychosomatic patients are dependent, this characteristic can be used supportively and interpretatively at crucial periods in the treatment. Much hostility surfaces during therapy—first in the form of overt ventilation and then in the framework of the transference. Therapists must encourage free and appropriate expression of patients’ hostility.

Interpretation

Therapists must pay particular attention to current problems in patients’ immediate life situation and must deal with patients’ reaction to the therapist and to treatment. Therapists should increasingly emphasize evaluation of patients’ characterological difficulties and habitual reactions, particularly actions to themselves (self-esteem, guilt) and reactions to his or her environment (dependence, submission, need for affection). Psychiatrists should also analyze patients’ anxieties and coping mechanisms for stress situations, such as requests for complete care, the need to always be right, lack of self-assertion, and suppression of all forbidden impulses.

Some psychoanalytic investigators have reported dramatic results when unconscious material was interpreted as a drastic measure during an acute illness. Although most Freudian psychoanalysts seem to think that genetic material must eventually be interpreted for a complete cure, new approaches have shown that adequate results can be obtained when psychotherapy is limited to the analysis of characterological and ego defenses associated with disturbed interpersonal relationships.

Patients with psychosomatic disorders are often involved in a repetitious pattern of stress in their interpersonal relationships. Because such patients are usually unaware of the pattern, it is helpful to show them that it is not accidental but is determined by factors of which they are unaware. It is essential to show patients how to change the disturbing pattern and act in a new and healthier manner.

Psychosomatic patients tend to drive toward psychologically regressed mental and physical behavior and usually regress to a traumatic or highly conflictual period. By reenacting certain specific attitudes of childhood or infancy, they are attempting to master the anxiety and illness first manifested during these earlier stages.

In the treatment of psychosomatic disorders, the key concept is flexibility in technique. Because of patients’ lack of motivation and poor physical condition, it may be necessary to make frequent changes in the psychotherapeutic approach.

Resistance During Therapy

Because patients with psychosomatic disorders often strongly resist entering psychotherapy, resistance frequently continues unabated during therapy. Many patients’ motivations for entering treatment are so poor that they frequently drop out of therapy for minor reasons.

Interruption of Psychotherapy for a Medical Emergency

During a course of psychotherapy, a patient with a psychosomatic disorder may require medical or surgical treatment for the organic disorder. The psychiatrist should cooperate closely with the surgeon or medical personnel and should maintain contact with the patient—in person or by telephone—during the emergency. Such interest offers valuable emotional support in a time of crisis.

If a patient is hospitalized, the psychiatrist should help other hospital personnel recognize and learn to tolerate the frequently difficult and provocative behavior of some psychosomatic patients. The preparation can be of use to the patients as well; when they see their demands being met considerably, they may be less inclined to view their world as hostile and formidable.

Danger of Psychosis

There are no simple relations between psychosomatic disorders and psychoses. Some people in whom physiological and psychological processes are poorly integrated manifest both psychosomatic disorders and psychoses. In other people, the ego integration is such that stress produces a breakdown of bodily function rather than a psychotic maladjustment. Some nonpsychotic psychosomatic patients can become psychotic or exhibit psychotic symptoms as a result of too active an interpretation and with the removal of defensive elements in the personality structure.

MEDICAL ASPECTS

Internists’ treatment of psychosomatic disorders should follow the established rules for medical management. Generally,
internists should spend as much time as possible with a patient and listen sympathetically to the many complaints; they must be reassuring and supportive. Before performing a physically manipulative procedure—particularly if it is painful, such as a colonoscopy—the internist should explain to a patient just what to expect. The explanation allays the patient’s anxiety, makes the patient more cooperative, and actually facilitates the examination.

Patients’ attitudes toward taking drugs may also affect the outcome of the psychosomatic treatment. For example, patients with diabetes who do not accept their illness and who have self-destructive impulses of which they are unaware may purposely not control their diet and, as a result, end up in a hyperglycemic coma. Some cardiac patients refuse to curtail their physical activity after a myocardial infarction because of a reluctance to admit weakness or because of a fear that they will somehow be considered unsuccessful. Others use their illness as a welcome punishment for guilt or as a way to avoid responsibility. Therapy in such cases must strive to help patients minimize their fears and focus on self-care and reestablishment of a healthy body image.

**ACCEPTANCE OF PSYCHOMEDICAL TREATMENT**

An advantage of the collaborative approach is that patients benefit from the efforts of specialists trained in various medical disciplines, each working in the area in which they are best equipped to function. Some physicians, however, have resisted a psychiatric approach because of inadequate psychiatric training in medical school, unfamiliarity with the specialized language of psychiatry, and a general prejudice based on the cost of psychotherapy and the alleged unscientific and subjective aspects of psychiatry.

**OTHER TYPES OF THERAPY**

Other types of treatment have been introduced for psychosomatic disorders. The first category includes psychotherapies based on psychological insight and change, such as group and family psychotherapy; the second category is composed of behavior therapy techniques based on pavlovian principles of learning new behavior, such as biofeedback and relaxation therapy.

**Group Psychotherapy and Family Therapy**

Because of the psychopathological significance of the mother-child relationship in developing psychosomatic disorders, modification of this relationship has been suggested as a likely focus of emphasis in the psychotherapy of psychosomatic disorders. Toksoy Byram Karasu wrote that the group approach should also offer greater interpersonal contact and provide increased ego support for the weak egos of psychosomatic patients who fear the threat of isolation and parental separation. Family therapy offers hope of a change in the relationship between the family and the child. Both therapies have had excellent initial clinical results.

The long-term evaluation of the results of the various psychotherapies, individual and group, for psychosomatic disorders remains to be carried out. After an exhaustive study of psychosomatic psychotherapeutic treatment, Karasu concluded that some patients with medical disorders may respond positively to psychological treatment, either physically or psychologically. Some medical disorders seem more amenable to psychotherapy than others, and some therapeutic modalities appear to be more effective than others. Some people may be more responsive to psychotherapy than others, especially in relation to the nature of their psychopathology rather than their physical pathology.

**Behavior Therapy**

**Biofeedback.** The application of biofeedback techniques to patients with hypertension, cardiac arrhythmias, epilepsy, and tension headaches has been successful in many, but not all, instances. Some patients do not respond.

**Relaxation Techniques.** The treatment of hypertension may include the use of the relaxation techniques. Positive results have been published about the treatment of alcohol and other substance abuse by using transcendental meditation. Workers have also used meditation in the treatment of headaches.

**REFERENCES**


Mental Health & Related Acts


2. Various Mental Health Review Tribunal forms. – not provided

3. Protected Estates Act 1983. – not provided

4. Guardianship Act 1987. – not provided

5. Mental Health Act 2007. – not provided

What is the role of a LEGAL REPRESENTATIVE before the MENTAL HEALTH REVIEW TRIBUNAL?

By MARIA BISOJNI

A solicitor who represents a client suffering from a mental illness in decisions about their treatment or financial affairs has 'a delicate duty'.

Recent statistics suggest that about one in every five Australians will at some stage in their lives suffer from a mental illness. The consequences of a diagnosis of mental illness can often be immense. At one end of the spectrum, involuntary commitment, due to mental illness, is the second major form of incarceration after imprisonment in Australia. At the other end of the spectrum, people may have transitory periods of illness and require little or no continuing support or monitoring. Effective and proper legal representation of people with a mental illness is thus of key importance.

Decisions for involuntary commitment, treatment and community care are made initially by Magistrates and subsequently by the Mental Health Review Tribunal. The first step following the scheduling of a patient who is suffering a mental illness is to bring the patient before a Magistrate who may make an initial order for involuntary hospitalisation, compulsory treatment in the community, financial management under the Protected Estates Act 1983, or discharge.

The Tribunal's functions

The Mental Health Review Tribunal is an independent Tribunal established under the NSW Mental Health Act 1990. It sits as a three-member panel consisting of a lawyer, psychiatrist and a person with other suitable qualifications or experience, such as a psychologist, social worker or consumer representative. The multidisciplinary nature of the tribunal reflects the different skills and experience needed to make informed decisions, with each member to be a 'highly informed multidisciplinarian'.

The Tribunal's main functions are to consider applications concerning:

- patients (including long-term hospitalised patients) who have been detained involuntarily in a psychiatric hospital to establish whether the patient should be further detained (including for electroconvulsive therapy (ECT) and surgery);
- patients who have been discharged from hospital and who receive compulsory care and treatment in the community;
- forensic patients, namely those charged with offences but who are found not guilty on the basis of mental illness, unfit to be tried or who have developed a mental illness while in prison; and
- financial management orders under the Protected Estates Act 1983 for persons found to be incapable of managing their financial affairs.

Most hearings are concerned with the primary question of whether the person is a mentally ill person, a definition that has two limbs:

- Are there symptoms of mental illness (e.g., serious thought disorder, delusions, hallucinations, severe disturbance of mood, sustained irrational behaviour, etc)?
- Does the person pose a real risk of serious harm to themselves or others?

In deciding the issue of mental illness, the Tribunal is entitled to take into account the continuing condition of the patient, including any likely deterioration in the patient's condition and the likely effects of a deterioration. The Tribunal must also determine whether care and treatment can be provided in a less restrictive setting.

Rules concerning legal representation

There is little guidance provided by the Mental Health Act as to the role of a legal representative before the Mental Health Tribunal, other than some basic provisions as to the right of representation. The Act provides that:

- The fact that a person is suffering from a mental illness is presumed to be no impediment to their legal representation.
- A forensic patient (a person charged with criminal offence where there is an issue of mental illness) with a matter before the Tribunal must, unless the forensic patient decides otherwise, be represented by a barrister or solicitor, or with the Tribunal's approval, another person of his or her choice. The Act does not indicate what factors should be taken into account in determining whether a forensic patient has made an informed decision to refuse representation. Argument could arise as to whether a person suffering from a disability had made an informed or rational decision as to representation.
- Non-forensic patients (civil review
patients) or persons detained in hospital may be represented by a barrister or solicitor or with the approval of the Tribunal, by another person of his or her choice." The President of the Tribunal or his or her delegate is also given a broad discretion under the Act to appoint a person to assist the Tribunal in respect of any matter before it.

While the Act provides no guidance as to the particular role of representatives, the objects of the Act state that it is to facilitate the care, treatment and control of persons who are mentally ill or mentally disordered, while protecting the civil rights of those persons, to give an opportunity for those persons to have access to appropriate care. Patients are to receive the best possible care and treatment in the least restrictive environment, and restrictions on liberty and other rights, dignity and self respect are to be kept to a minimum necessary in the circumstances. In short, the essential principle underlying the legislation and the Tribunal review is the best interests or welfare of the patient. All professionals involved in the process should bear this principle in mind. While the Act is silent on the matter, it would be expected that these general objects would shape the functions and ethics of legal representatives appearing before the Tribunal.

General duties of representatives

Barristers and solicitors in NSW are governed by a variety of rules and regulations as to their conduct and ethics. The NSW Barristers Rules provide that barristers are to act "honestly, fairly, skilfully, diligently and bravely". A barrister "must seek to advance and protect the client’s best interests to the best of the barrister’s skill and diligence, uninfluenced by the barrister’s personal view of the client or the client’s activities". Further, a barrister must seek to assist the client to understand the issues in the case and the client’s possible rights and obligations. The NSW Law Society Solicitors Rules are in similar terms. Practitioners are to act with "competence, honesty and candour." Practitioners must seek to advance and protect the client’s interests to the best of their skill and diligence, uninfluenced by the practitioner’s personal view of the client or the client’s activities.

Both barristers and solicitors have a fundamental obligation as officers of the court to facilitate the administration of justice. There are no specific rules or regulations dealing with the conduct of matters concerning people with a mental illness. Broadly speaking, the Australian legal system is adversarial in the sense that it is the parties and not the judge or other decision-maker who has primary responsibility for defining the issues in dispute and for investigating or advancing their case. However, the term ‘adversarial’ is also used to connote the culture or attitude of the parties and their legal representatives. Sporting or military epithets and metaphors are often used to describe these contests and, in its most dramatic form, lawyers are depicted as gladiators involved in a confrontational battle from which there is only one winner. This depiction is clearly an extreme one as there are many lawyers who encourage and facilitate settlement and compromise, and most civil disputes are in fact settled.

The Tribunal’s decision-making model

It is clear that proceedings before the Tribunal are not intended to reflect this adversarial model but rather an inquisitorial one. Proceedings are to be conducted with as little technicality and formality, and as much expedition as the Act and proper consideration of the matters permit. The Tribunal is not bound by the rules of evidence and may inform itself in any manner as it thinks appropriate.

In a leading authority on the interpretation of the Mental Health Act 1990, Harry v The Mental Health Review Tribunal (1994) 33 NSWLR 315, 335, Mahoney JA, in the NSW Court of Appeal commented that the duty of an advocate appearing before the Mental Health Review Tribunal was not “to get his client off”. Instead the duty was a “delicate one” of ensuring that the client was afforded all protections available but not to negate the application of beneficial powers for mere technical deficiencies.

The role of the advocate is made still more complex by the differing approaches of professionals working in the system. The medical profession tends to pursue clinical and therapeutic interests while advocates, and the Tribunal itself, as it must under the legislation, focus on the legislative and legal requirements for detention. These approaches reflect distinctly medical and legal models, with the former identifying medical discretion as promoting therapeutic success, while the legal model establishes external checks and balances, quasi-judicial review and limited medical discretion on the basis of protecting an individual’s civil liberties.

A related concern sometimes expressed by medical professionals is that courts, and even tribunals, are adversarial, which may be especially damaging to the therapeutic relationship between the doctor and patient. The legal model has been dramatically, or perhaps melodramatically, described by Appelbaum as an invitation for patients to die with their rights on.” It has been said that the Tribunal has an adversarial structure because hearings are based on confrontation between parties, with the role of prosecutor played by the doctor responsible for the medical care of the patient appearing before the Tribunal.

On the other hand, lawyers sometimes complain that the discourse of such tribunals is dominated by technical medical terminology so that the medical model determines the framework and a patient’s civil liberties may be compromised.

It seems clear that the current model established under the Mental Health Act is designed to balance the potentially competing views of rights-based and therapeutic models. Mental health law and practice develops through the interaction of law and psychiatry. While discourse on the role of the Tribunal and the relative merits of a legal or medical approach must be usefully debated, such discussion should not obscure the basic requirement of the process which is identifying and satisfying as far as possible the interests and needs of those with mental illness.

The primary duties

A primary obligation of a legal advocate in any matter is to obey their client’s instructions and this duty is not varied because of a client’s mental illness. The legal representative is to advocate the client’s wishes and not those of doctors, relatives or friends. Such persons may provide valuable information and support but their wishes and perspectives should not be superimposed over the client’s wishes and interests. In addition, legal professional privilege extends to the lawyer-client relationship in the normal way. However, obtaining comprehensible or coherent instructions from clients suffering from a mental illness may be difficult.

Hence it has been suggested that solicitors appearing for clients with an intellectual...
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role to provide legal advice to the patient and this may involve indicating that clinicians are of the view that a further period of hospitalisation or community care is in the client’s best interests. However, if the client disagrees with this view the lawyer should proceed to advocate the client’s case.

Practical tips on representation and case preparation

There are a number of practical hints on how to represent people, including the following:

- Clients should be treated with respect and understanding and without condescension.
- At least basic knowledge of the relevant legislation, the operation of the Tribunal and relevant medical terminology is desirable. There are a number of sources of information including the Tribunal itself and the Mental Health Advocacy Service.

In relation to case preparation the following points may be of use:

- All relevant material must be identified and considered, including the hospital file. For example, under s.276 of the Mental Health Act, a representative is entitled to inspect medical records in the possession of any person relating to the patient. Moreover, the Tribunal also has a power to summon a person to attend as a witness or to produce documents (s.278).
- Interviewing a client is normally necessary and there should be no reliance placed on written instructions without an interview.
- It may be wise to confirm instructions with the client shortly before any hearing.

The following issues need to be investigated:

- The client’s level of insight in relation to the alleged illness.
- What preparations, if any, has the client made for eventual discharge and what preparations could be made (e.g., place of residence, training and employment, medical treatment).
- What is the level of contact and support with family, friends and within the community?
- Is the client willing to continue with certain treatment after discharge?
- What evidence is necessary and what are the best avenues for its presentation? For example, independent medical evidence necessary or a report from a social worker about the options for the person living within the community?

Conduct of the hearing

The usual procedure of the Tribunal is that the members explain who they are and the purpose of the proceedings. Normally the hospital witnesses, such as the treating doctor and social worker, give evidence first. Such witnesses may be cross-examined by the patient’s advocate. The patient’s advocate may call witnesses including the patient. Members of the Tribunal may ask questions and seek clarification. The patient’s advocate should test the evidence of doctors and others but with the best interests of the client operating as a guiding principle. For example, a variant of ‘plea bargaining’ about the length of hospitalisation is clearly inappropriate as issues as to hospitalisation should be determined by tested and considered medical opinion. Submissions regarding the length of stay in hospital should be grounded on medical evidence as to when the person is likely to respond to treatment, relying on the principle of the provision of care in the least restrictive environment.

In circumstances where the patient is unable to understand the nature of the proceedings it may be appropriate for the advocate to assist the Tribunal to undertake its inquiry and in some circumstances to adopt a position that amounts to an amicus curiae.

After the hearing, the lawyer should explain the order and its consequences to the patient and possibly family and friends.

A dynamic role

There is no static representative model for lawyers in mental health proceedings. Instead, the relationship is dynamic, with a constant need to assess and advocate a client’s rights within the broader context of the objectives of care and welfare. Lawyers should not take the rigours or subtleties involved in performing this ‘delicate duty’ for granted. Lawyers will generally not have the qualifications, training or experience to make informed judgments about appropriate treatment for people who are thought to be suffering from a mental illness. It is thus incumbent upon advocates to assess the level of their knowledge and experience and where appropriate to seek assistance so that they may properly represent their client’s interests. Legal representatives should feel comfortable with seeking information or advice from their peers, their professional bodies and from the Tribunal. Such efforts will improve the quality of representation and the quality and efficiency of the decision-making process.

ENDNOTES: Extensive endnotes for this article are available on the Journal’s website www.lawsociety.com.au.
First steps in new approach to MENTAL HEALTH LAW

By MARIA BISOGNI

Legislation which received bipartisan support recognises for the first time the vital role of carers, and enshrines their rights as part of a new era in mental health law reform.

WITH THE INCIDENCE OF mental illness rising sharply in the Western world, and estimates that the disorder affects one in five Australians, there is considerable public interest in the new Mental Health Act 2007 which came into effect on 16 November 2007. The Act was preceded by a review of the Mental Health Act 1990 which commenced in February 2004 and was in response to the Report of the NSW Parliamentary Select Committee on Mental Health in December 2002. Two discussion papers — “Carers and Information Sharing”, and “The Operation of the Mental Health Act” were widely disseminated for comment and consultation from carers, patients, clinicians and other key stakeholders.

The Mental Health Review Tribunal whose essential role in its civil jurisdiction is to determine whether persons under the Act require involuntary detention for their treatment, care and control or involuntary treatment in the community was a key participant in the reform process and made a number of recommendations for changes to the old Act. Introducing the Mental Health Bill on 9 May 2007, Mr Paul Lynch, the Minister for Local Government, Minister for Aboriginal affairs, and the Minister Assisting the Minister for Health restated the government's commitment to mental health services, citing the legislation as “one of the keystones to supporting ongoing improvement and reform of these services”. The new Act re-enacts much of the old Act but with a number of key reforms in relation to the role of primary carers; first-ever patient's rights clause; flexibility in relation to Community Treatment Order applications and their duration; and arrangements for the transport of mentally ill persons.

ENDNOTES
3. Ibid paras [15]-[19].
5. Ibid para [16].
6. Ibid paras [22]-[23].
7. Ibid paras [21]-[22].
8. Ibid para [25].
9. Ibid para [35].
10. Ibid para [36].
11. Ibid para [57].
12. Ibid paras [46]-[47].
13. Ibid para [48]. The explanation is not clearly developed in the transcript extracted.
17. [2007] NSWCA 290 paras [75]-[74], [76].
18. [2007] NSWCA 290 para [74].
19. [2007] NSWCA 290 para [60].
20. [2007] NSWCA 290 para [67].
22. [2007] NSWCA 290 para [16].
23. [2007] NSWCA 290 paras [46]-[47].
24. [2007] NSWCA 290 para [48].
28. Ibid.


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This article provides an overview of the key changes in the new Act.

Statement of principles for care and treatment

Section 68 sets out a list of patient's and primary caregivers' rights and expands the central underpinning of the 1990 Act that a person with a mental illness should receive the best possible care in the least restrictive environment enabling that care to be given while interfering minimally with their rights, dignity and self-respect.

The following principles of care and treatment have been recognised:

- the right to timely and high quality treatment and care in accordance with professionally accepted standards;
- the designing of care and treatment to assist the person, wherever possible, to live, work and participate in the community;
- the prescription of medication is to meet the health needs of the person and should be given only for therapeutic or diagnostic needs and not as punishment or for the convenience of others;
- information about treatment, treatment alternatives and the effects of treatment;
- the involvement of patients in the development of their treatment and ongoing care plans; and
- the right of carers to be kept informed and to be given effect (see s.68).

Primary carers

There are an estimated 750,000 carers in NSW alone with an estimated 10 per cent of those caring for persons with a mental illness. The repeated theme from carers in response to the discussion papers was the need to seek their formal involvement in treatment decisions and the sharing of information. Some carers had felt excluded from such decisions, while mental health workers clearly felt constrained to deny access to information to carers because of limitations of the 1990 Act compounded by privacy legislation.

The following submission to the 2002 NSW Senate Inquiry by a parents and carers mental health group encapsulated a common complaint: “It is our view that the Mental Health Act does not provide for adequate input by carers/relatives/significant others. It is realised that confidentiality is extremely important. However, each family involved with mental health services in this area, who are members of the parents and carers group, has experienced frustrating and dangerous situations caring for their family, due to the restrictions placed on them by the problems associated with confidentiality and the refusal by many mental health staff to acknowledge the validity and indeed, the importance of input into that person's care, appropriate intervention and ongoing treatment plan.”

Consider the plight of a young carer: “I have been helping to care for since I was 13 ... when I was 15, my brother moved out and I became the main carer for Mum. I was like a parent to her. “It's only over the last year that I've got the doctors and nurses to talk to me, but I've had to push them to do it. I guess they thought I was just a child, but I have been the main carer for Mum for quite a while now.”

The legislation introduces the concept of primary carer for the first time. This will be a welcome development for carers, many of who have frequently complained that they were often not notified of a loved one’s admission or discharge from hospital, despite their providing vital assistance and support. Allowing them into the loop is intended to promote improved outcomes for patients.

A patient of a mental health facility (both inpatient and community) may nominate a person as their primary carer with the latter having rights in relation to access to information, notice of a person's admission or discharge from hospital, including any unlawful absences, and involvement in the patient's discharge planning. Section 71 sets out a prescribed hierarchy of primary carers starting with a guardian, followed by a parent if the patient is under 18 years of age. If there is neither a guardian nor a parent, a person may nominate a primary carer. If no nomination is made, primary carers are appointed from a hierarchical default list, commencing with the spouse or the person at risk of harm, or that the patient will be a welcome development for carers, (s.69).

The legislation’s intent is to ensure that persons subject to the Act will always have their relevant interested others notified of significant events in relation to their hospitalisation and discharge planning.

A patient making the nomination of a primary carer has the right to vary, revoke or exclude persons from any nomination, although a person under the age of 18 is unable to exclude a parent. All nominations, variations or revocations must be in writing. Nominations remain in force for a period of 12 months unless revoked.

Once a nomination is made it must be acted upon by an authorised medical officer or the director of the community treatment unless there is a reasonable belief that to do so would put the patient or nominated person at risk of harm, or that the patient making the nomination, variation or revocation was at the time incapable of doing so.

Section 75 provides that all reasonably practicable steps must be taken to notify the primary carer of a patient's initial detention within 24 hours of the admission.

Section 76 sets out a list of other notifiable events:

- a patient's absence from a facility without permission or failure to return from leave;
- the proposed transfer of a patient to another facility or mental health facility;
- a patient's discharge from a mental health facility;
- re-classification as a voluntary patient;
- a proposed application to the Mental Health Review Tribunal for an ECT inquiry or a determination as to whether a patient can give informed consent;
- a surgical operation;
- an application to the tribunal or Director-General for consent to a surgical operation or special medical treatment.

A primary carer, as well as the patient, must be consulted in relation to the person's discharge planning and subsequent treatment, including the provision of appropriate information as to follow-up care (s.67).

A primary carer may request consent to a surgical operation on behalf of the person, subject to a CTO. The details of such applications must be given directly to the affected person and all those subject to a CTO (see s.75 and s.57(9)).

Community treatment orders

Community Treatment Orders have been reformed to allow them to be made in relation to persons residing in the community, without the need for an inpatient admission. The maximum duration of orders has been extended from six to 12 months. There appears to be some statutory recognition that 12-month orders will be the exception rather than the rule, as orders of this duration confer an automatic right of appeal against the length of the order (see s.67).

The range of potential applicants for community treatment orders has expanded to include the primary carer and a medical practitioner who is familiar with the clinical history of the person. As was the case under the 1990 Act, applications may still be made by the authorised medical officer of a hospital and the Director of Community Treatment.

This reform recognises that the model of care in the community has changed and that many individuals suffering a mental illness or disorder prefer their care to be managed by their general practitioner or private psychiatrist.

Treatment may now be given at a specified place, which may include agencies other than a community mental health facility. A restriction of the 1990 Act was the requirement that the person was required to be present either at the home of the affected person or the health care agency to receive treatment and care from the agency. Section 56 now provides that the order can require “the affected person to be present, at the reasonable times and places specified in

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the order to receive the medication and therapy, counselling, management, rehabilitation and other services provided in accordance with the treatment plan.

The new form of order potentially facilitates treatment by other specialist agencies, including NGOs, and supports the legislative aspiration that treatment and care should be of a kind designed to allow persons as far as possible to lead fulfilling lives in their circumstances. The overall responsibility for implementing the order remains with the declared mental health facility. This means that applicants such as the primary carer or medical practitioner will need to have the support of the facility in order to implement the order. The ultimate responsibility for the enforcement of the order includes the issuing of breach notices remains with the facility.

Applicants seeking an order are required to comply with the notice provisions for service of the application on the person with a copy of the proposed treatment plan. This allows the affected person to have advance notice of any care and treatment proposal well before the hearing. In cases where the application is made for someone who is not detained in a mental health facility the application may only be heard after 14 days notice has been given.

Another significant and welcome change is that re-admission to a hospital no longer has the effect of cancelling the CTO. A person subject to an order who is admitted to hospital on a schedule may be discharged on the same CTO as long as it still has time to run, and unless the tribunal otherwise orders (see s.56(4)). This refinement is an important tool in the ongoing care of persons who might be readmitted to hospital on a schedule and who might then be discharged into the community subject to the existing order.

Transport provisions

Under the 1990 Act the authority to transport persons lies with police, medical practitioners and accredited persons.

This has now been expanded to include ambulance officers and members of staff of the NSW Health Service. The use of police is reserved for cases where there is serious concern about the safety of the person or other persons. In line with legislative developments in other Australian states, the responsibility to transport lies at first instance with the NSW Health Service and only in cases where there are safety concerns will police involvement be required.

This development properly recognises that (in the majority of cases) the transport of mentally ill persons is a health and not a criminal justice issue. The 2002 NSW Inquiry into Mental Health Services noted many examples of direct police experience of being used inappropriately in the detention, transportation and supervision of people with a mental disorder. This change goes some way in accommodating these concerns. A police officer making a submission to the Commonwealth Senate Inquiry into Mental Health in 2005 told the inquiry that police were “often required to transport these people but they are not criminals, they are ill”.

This important reform is designed to place the primary responsibility for the transport of persons on NSW Health staff and will allow police resources to be directed to law enforcement demands. From a consumer perspective, the stigma often associated with police involvement should be greatly reduced.

Persons who are authorised to transport to and from a mental health facility may in exercising those functions use reasonable force and may restrain the person in any way that is reasonably necessary in the circumstances (s.81).

Sedation by an authorised person may be used for the purpose of transporting the person if it is necessary to do so to enable the person to be taken safely.

If there is a reasonable suspicion that a person is carrying anything that would present a danger to the person or another person or that it could be used to assist in the person’s escape from their custody, a frisk or ordinary search is permitted (see s.81).

“…where potential applicants for community treatment orders have expanded to include the primary carer and a medical practitioner who is familiar with the clinical history of the person.”

The role of ambulance officers

The 2007 Act provides a new role for ambulance officers to take persons to mental health facilities for treatment. This may occur if in the course of service delivery there are reasonable grounds to believe that the person appears to be mentally ill or mentally disturbed “and that it would be beneficial to the person’s welfare to be dealt with in accordance with this Act” (see s.20). Only ambulance officers authorised by the Director General can exercise this function. It is envisaged that part of their accreditation will involve appropriate training and support.

Conclusion

The new Act has introduced a number of reforms, which have been well received. The most significant aspect of legislative reform relates to the forensic provisions which deal with mental illness acquittals, persons found unfit to be tried and prisoners who are transferred to a hospital for assessment and care. The Mental Health Review Tribunal’s President, the Hon Greg James QC has conducted a wide-ranging review of the forensic provisions in the 1990 Act and the Mental Health (Criminal Procedure) Act shivered to the theater the publication of a consultation paper dealing with the major areas of principle. He has recently reported to government and his proposals for reform are under consideration.

Presently, the forensic provisions of the 1990 Act have been relocated in the Mental Health (Criminal Procedure) Act 1990. It is anticipated that the parliament will consider a Bill embodying a number of reforms in the 2008 parliamentary cycle.

The tribunal welcomes the input of legal practitioners and has published a number of resources on its role and various heads of jurisdiction. Practitioners are also invited to access this information on the tribunal’s website at www.mhrt.nsw.gov.au.

Endnotes

2. Full-day Hansard transcript p.81.
3. Carers NSW contact Mental Health Coordinating Council (MHCC) discussion paper.
6. A close relative or friend is defined as someone who maintains both a close personal relationship with the patient through frequent personal contact and a personal interest in the patient’s welfare. Again this must not be on a commercial basis and would include someone in receipt of a carer’s pension but not any individual attached to an organisation that provides housing or support.
7. See Mental Health Regulation 2000, clause 44.
8. Although in the case of applications from the Director of Community Treatment there is no requirement that the Director be familiar with the person’s history – see Mental Health Regulation 2000, cl.19.
9. NSW Select Committee on Mental Health, pp.236 – 245.
11. See Mr Paul Lynch’s speech moving the Bill on 9 May 2007, Full-day Hansard transcript p.81.
12. The Psychos and Therapeutic Services Act 1966, and the regulations under that Act, regulate the persons who may prescribe and administer drugs (including sedative drugs).