IN-DEPTH REVIEW

Doctors’ health and fitness to practise: performance problems in doctors and cognitive impairments

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**Background**
As a response to concerns over the safety of patient care and quality of care provided by doctors, there has been an increasing interest in identifying the reasons for medical errors.

**Methods**
This paper reviews briefly the common neurocognitive causes for performance problems in doctors and provides an updated account of the current literature. Search on Medline and PsychINFO for English language articles between 1956 and September 2006 was performed, as well as a manual search by the authors for other relevant articles.

**Results**
Neuropsychiatric and neuropsychological assessment is increasingly accepted as an accurate evaluation tool to clarify the performance problems in doctors. Furthermore, it seems that neurocognitive difficulties are commonly found to be the cause for such problems.

**Conclusions**
The performance problems in doctors need to be acknowledged ‘better too soon than too late’. Neuropsychiatric and neuropsychological assessment helps to create an accurate treatment and rehabilitation plan for the specific functional tasks of the particular doctor’s duties.

**Key words**
Cognitive functions; doctors; frontal-executive functions; impaired performance; memory; neurocognitive; neuropsychiatry; neuropsychology

Introduction

The impact of litigation, increasing demands from the public and the National Health Service (NHS) ‘blame culture’ are placing great pressure on doctors. Job satisfaction is affected and doctors are increasingly vulnerable to pressure and perhaps to mental and physical ill-health. However, doctors in general tend to minimize their own health problems, do not take time off work, have a poor understanding and distrust of occupational health services and tend to self-diagnose and self-prescribe [1–4]. As services are reactive in nature, they tend to become involved only after performance problems have arisen, sometimes after a major medical error has emerged.

Recently, the chief medical adviser for the UK, Sir Liam Donaldson, published the results of the first comprehensive review of the regulation of the medical profession conducted in >30 years [6]. ‘Good Doctors, Safer Patients’ was commissioned following the publication of the Shipman Inquiry. The principal shift in fitness to practise cases has proposed a move from the criminal standard of proof to the civil standard. In addition, Professor Donaldson proposed renewed focus on the assessment, rehabilitation and supervision of doctors with performance problems. He did not specify the actual assessment procedure but rather referred to the assessment protocol in the National Clinical Assessment Service (NCAS) [see also 5]. He also proposed that doctors approaching retirement should be invited to a review with their General Medical Council (GMC) affiliate to assess whether a further 5-year period of re-licensing is appropriate.

In an attempt to elucidate causes for performance problems, great interest has emerged in assessing doctors’ cognitive performance as part of the fitness to practise proceedings and safety measures. This has particularly been the case since an Australian study found that cognitive impairment in doctors is responsible for 63% of all adverse medical events and that most were determined to be preventable [7]. Furthermore, a recent study looking at the neuropsychological profile of 200 doctors involved in litigation suggested that neuropsychological assessment could establish the very precise nature of their relative impairments. This was thought to be helpful in developing personal educational programmes and in providing professional support and help [8]. Turnbull et al. [9], in their study of 27 physicians, raised the issue of whether a neuropsychological screening procedure for
underperforming doctors should be developed although there is no standard battery of tests available for any particular condition. Trunkey and Botney [10], in a review article examining the different ways of assessing competency in surgeons and airline pilots, firmly recommended neuropsychometric testing as part of a reliable assessment for competency. Finally, a retrospective review of 148 doctors with performance problems demonstrated relative deficits on tests in sequencing, attention, logical analysis, eye–hand co-ordination, as well as in verbal and non-verbal learning [11]. The deficits were concluded to be sufficient to explain their performance difficulties.

The aim of this paper is to give a brief description of neuropsychiatric and neuropsychological assessment procedures illustrated by short descriptions of doctors with performance problems.

Neuropsychiatric and neuropsychological assessments

Neuropsychiatric assessment focuses on both psychiatric and neurological aspects of history and examination, with particular attention given to the interplay between brain disorder and mental symptomatology [12,13].

Neuropsychological assessment focuses on the analysis and quantification of cognitive impairments. It can help in a number of areas, for example in localizing brain lesions, differentiating between organic and psychiatric disorders, assessing functional abilities and planning rehabilitation. Current intelligence quotient (IQ) can be compared with predicted pre-morbid IQ to estimate the level of overall intellectual decline. Because IQ tests involve multiple skills subserved by different parts of the brain, further testing of specific domains is carried out.

Memory tests assess the range of memory sub-skills subserved by different brain regions, such as verbal and visual, short and long-term, recall and recognition memory. Further tests are used to assess language and visuospatial skills as well as frontal–executive skills (e.g. verbal fluency, planning and response inhibition). Test results are compared to normative data allowing for chronological age; however, it is important to take into account a patient’s educational and cultural background, physical and psychological state at the time of the assessment and concurrent use of medication or other substances.

Remediability and intervention

If there is a suspicion that cognitive impairment is related to a treatable psychiatric condition, such as depression, appropriate treatment should be accompanied by continued assessment of cognitive function. Alcohol problems can be much more difficult to manage because of the risk of relapse, although a crisis at work may be the precipitant to successful abstinence. Again, once the doctor has become abstinent, it will be appropriate to reassess cognitive function.

Similarly, medical conditions such as multiple sclerosis or space-occupying lesions need to be managed appropriately: more problematic may be the decision about a doctor’s future career once interventions have been completed. Neuropsychological test results may be helpful in this respect. Although drug treatments such as cholinesterase inhibitors are available for some dementias, their benefits are limited and, once a doctor has reached this stage, she/he is extremely unlikely to be fit to practise. In cerebrovascular disorders, the most important aspect of management consists of minimizing the risk of further vascular episodes. Again, the neuropsychological assessment is critical: a doctor may be able to practise after a localized cerebrovascular episode, but more generalized vascular changes may make her/him unfit to practise.

Referral to a rehabilitation unit may well be appropriate. Psychological strategies for coping with memory disorders include the use of external aids, such as diaries, pager systems, etc., or ‘internal’ strategies such as the use of mnemonics [14,15]. These may be helpful to a practising doctor with relatively minor cognitive impairments [16].

Examples of neurological disorders affecting performance at work

Dementia

Like everyone else, medical practitioners are subject to cognitive decline with ageing, but the extent has not been quantified. One extensive computerized neuropsychiatric assessment study compared 1002 physicians with 382 controls of 25–75 years of age [17]. The study showed progressive decline with age in both physicians and controls and a more rapid decline after age 65 years. The most dramatic declines occurred in verbal memory, reasoning and visuospatial ability. Furthermore, the results indicated that the ability to learn, store and retrieve information declined more rapidly in physicians than in same-age peers in other professions.

The disease may have many causes but the types of functional impairments are closely related to the areas of the brain affected, e.g. in Alzheimer’s dementia, the medial temporal region is affected early with later involvement of the parietal and temporal association areas, frontal cortex and brain stem nuclei; in frontotemporal dementia, cortical atrophy occurs in the temporal and/or prefrontal cortex with the parietal and occipital lobes being spared.

Alzheimer’s disease

Alzheimer’s disease is the most common cause of dementia. According to a recent population-based twin study, the extent of hereditability for the sporadic disease is estimated to be almost 80% [18]. Until now, it has been
considered that ‘early’ onset, before age 65 years, and ‘late’ onset cases are similar enough to qualify as a single disease. However, this view is still debated [19]. Depression or physical complaints such as headaches may well be one of the presenting symptoms [see 20].

The following case illustrates that presentation and diagnosis are not always straightforward.

A 50-year-old academic was referred by his general practitioner (GP) due to performance problems at work. The academic dated his difficulties to 8 months previously when he had been mugged with loss of consciousness and suffered up to 6 h retrograde amnesia and 3 h post-traumatic amnesia. During two subsequent assessments in accident and emergency, he had been judged as suffering from confusion due to alcohol, although he admitted only to moderate consumption. He took 5 months off work, during which time he suffered from visual and auditory hallucinations; but subsequently returned to work, although with reduced responsibilities. During the assessment, he presented with psychomotor slowing and nominal dysphasia. Neuropsychological assessment revealed a clear drop in current compared to estimated pre-morbid IQ, most marked across performance domains. He showed impairments in visual and verbal recall, semantic memory and frontal-executive functions (verbal fluency and card sorting). In view of some inconsistencies in his presentation, two tests of malingering were administered (Test of Memory Malingering and Modified Card Sorting Test)—he passed both. Neurological examination, electroencephalography (EEG), lumbar puncture and extensive blood screen were normal. Magnetic resonance imaging (MRI) brain scan showed some evidence of small vessel disease but not sufficient enough to explain his cognitive impairment. Finally, brain positron emission tomography scan showed clear evidence of parietotemporal atrophy consistent with a diagnosis of early onset Alzheimer’s dementia. The academic was commenced on cholinesterase inhibitors and was recommended to opt for early retirement.

Frontotemporal dementia

Frontotemporal dementia is the second most common cause of early onset dementia, according to some reports. It is a complex degenerative dementing syndrome whose broad phenotype often overlaps with that of other neurodegenerative disorders. It constitutes a clinically heterogeneous group [21]. Three variants have been identified: (i) frontal lobe degeneration in which social behaviour and personality changes predominate; (ii) semantic dementia in which naming, word finding and verbal comprehension impairments predominate in the context of fluent speech, associated with left temporal degeneration, with relative sparing of the hippocampi and (iii) progressive non-fluent aphasia, in which speech becomes very sparse, with phonological and syntactic errors, associated with left perisylvian atrophy.

In the frontal variant of frontotemporal dementia, there are early changes in personality and behaviour and a decline in expressive speech. Behaviour is often disinhibited, tactless and childish leading into conflicts at home and in the workplace. Appearance deteriorates with incontinence occurring quite early in the process. There may be over-eating and excessive alcohol use. Lack of insight is usually greater than in Alzheimer’s disease [but see also 22].

Vascular dementia

Vascular dementia is not a single disease; it is a group of syndromes relating to different vascular mechanisms. It is currently widely believed that a continuum exists between the classically described dementias of Alzheimer’s disease and vascular dementia, indicating that these two diseases may possibly reflect one underlying dementia syndrome. Credence is given to this view by increasing evidence that both diseases share common vascular risk factors; hence, research into vascular cognitive impairment becomes crucially important [23,24]. Vascular disease produces either focal or diffuse effects on the brain. Hypertension is the most common cause of diffuse disease, and in many patients, both focal and diffuse diseases are observed together.

A 56-year-old clinical consultant was referred by the NCAS for advice on ability to practise. For the past 4 years, there had been a concern over performance, in particular diagnostic and prescribing competency, standard of continuous professional development and quality of record keeping. There had also been a concern over drinking during work hours. The medical history included a stroke at the age of 51, with dysarthria and fatigue as well as treated hypertension and hyperlipidaemia. In addition, there was a life long history of intermittent heavy alcohol use as well as a diagnosis of bipolar affective disorder. The patient had been treated with mood stabilizers, antidepressants and antipsychotics. The patient presented with no current affective or psychotic symptoms, but was mildly disinhibited in manner. On neuropsychological assessment, there was a significant drop in current compared to estimated pre-morbid IQ, largely in performance domains. There were deficits in processing speed, visual recall and recognition memory, as well as in frontal-executive functions (verbal fluency and response inhibition). In view of strong cerebrovascular risk factors, it was felt that vascular dementia was the most likely diagnosis, although alcohol and high doses of psychotropic medication might also affect cognitive functions. An MRI brain scan confirmed confluent white matter changes as well as small vessel disease. It was advised that frontal-executive deficits would affect ability to plan, judge and inhibit behaviour appropriately, and medical retirement was recommended. Management involved attention to controlling vascular risk factors, addressing alcohol misuse and reviewing psychotropic medication.
Alcoholic brain damage

The medical profession is at high risk of alcohol abuse [25]. It has also been suggested that heavy alcohol use increases as physicians age [26]. Even a moderate intake can impair memory, attention and other cognitive skills, including visuospatial processing, performance of complex tasks and decision making [but see also 27,28]. Alcohol and its metabolites are toxic to the brain and long-term high intake can lead to cerebral atrophy, enlarged ventricles and particular damage to dorsolateral frontal and parietal regions. Also, there is increasing evidence that people who use a moderate to severe amount of alcohol have higher risk of both cerebro- and cardiovascular problems. Binge drinkers seem to be at less risk of cognitive effects than daily heavy drinkers. Abstinence leads to improvement which neuropsychological research has proven to gradually continue over 5 or more years [29,30].

Transient global amnesia

Transient global amnesia (TGA) is a temporary version of amnestic syndrome, occurring in the middle aged or elderly, more commonly in men. It is thought to be due to transient dysfunction in limbic-hippocampal circuits lasting from several hours to ~24 h. It is characterized by repetitive questioning, and often confusion, but without loss of personal identity. It can be accompanied by headache, nausea or photophobia and can be preceded by physical or emotional stressors [31,32]. In two-thirds of patients, the aetiology is unknown; some (25% in some studies) have a history of migraine and ~7% subsequently develop epilepsy. No association has been found with cerebrovascular disease. TGA must be differentiated from psychogenic amnesia (in which there is invariably a loss of personal identity), complex partial seizures and transient ischaemic attacks (in which there is usually alteration of consciousness) [29].

A 45-year-old physician was referred to the service by his GP. He had been the subject of a complaint of indecent assault by a female patient 2 years previously, was facing a criminal charge and was under review by the GMC. His main complaint was of memory difficulties to the past 2 years, but his partner dated them to 16 years previously when the physician had suffered a subarachnoid haemorrhage (SAH) from which he had made an excellent recovery. The situation was complicated by moderate alcohol intake. The physician complained of low mood with early morning waking, diminished appetite, lack of energy and enjoyment, as well as fleeting thoughts of suicide. Neuropsychological testing gave an estimated pre-morbid IQ of 117. He scored well on verbal and visual recall memory and adequately on verbal and visual recognition memory. Tests of frontal-executive function were variable, but of note was a low score on response inhibition. Overall, it was thought that the problem with response inhibition might be related to the previous SAH and might be pertinent to the alleged charges, but that the memory complaints, in the presence of satisfactory memory test scores, are related to his depression—although the use of alcohol complicated the interpretation. He had previously tried an antidepressant and was not keen to resume this. Repeat MRI scan showed further vascular damage particularly in his frontal lobe as well as cerebellar changes in line with his high alcohol intake. Planned management included

likely and it was recommended that there was no reason why the patient should not continue to practise, provided there were no reports of any further episodes.

Examples of functional disorders affecting performance at work

Depression

Depression appears to be a more common problem in doctors than in other professionals, particularly affecting female doctors. Identified risk factors in physicians include sleep deprivation, alcohol use/abuse and poor job satisfaction [33]. The typical cognitive symptoms of depression are poor decision making and planning, memory and concentration problems, inattentiveness, irritability, interpersonal difficulties and in general slowing down [34]. Recent studies have established an association between depression and deficits in episodic memory [35] which may be associated with functional and structural changes particularly in the hippocampus and prefrontal cortex [36,37]. However, the difficulty often lies in deciding whether the patient suffers from primary depression or another underlying condition, such as dementia or brain trauma, to which depression is a response.

A 60-year-old clinical consultant was referred by the local NHS organization following concerns over their fitness to practise. Seven months previously they had suffered an episode of amnesia lasting ~12 h with extensive retrograde memory loss, which subsequently resolved. Accompanying symptoms included severe occipital headache, photophobia and nausea. Shortly prior to the major episode, a diagnosis of chronic idiopathic demyelinating polyneuropathy had been made and treatment with intravenous immunoglobulins commenced. The patient was admitted to hospital where neuropsychological testing following recovery revealed no deficits. EEG and MRI brain were normal. Seven months post-amnesia, a complete recovery was made apart from a brief amnesic gap for arrival to hospital and a short period thereafter. Repeat neuropsychological testing revealed no deficits. The presentation was consistent with TGA, recurrence was unlikely and it was recommended that there was no reason why the patient should not continue to practise, provided there were no reports of any further episodes.
a further discussion of antidepressant treatment and/or psychological input and addressing alcohol misuse.

Post-traumatic stress disorder

Post-traumatic stress disorder (PTSD) is not uncommon; an estimated 3–58% of people subject to a traumatic event develop the disorder. PTSD has a higher prevalence among people who feel helpless or who have additional life stressors [38]. However, even under the most traumatic conditions some individuals will never develop PTSD, and most who develop PTSD will show a remission within several months or years after the event. The symptoms of PTSD are divided into three clusters: intrusive or re-experiencing, avoidance and hyperarousal. PTSD and memory impairment are strongly connected, and PTSD appears to particularly affect executive functions and episodic memory [39]. However, a recent study suggests that exposure to a traumatic event can lead to executive problems regardless of a diagnosis of chronic PTSD [40]. Several studies have shown that PTSD is more common in house officers and emergency doctors as well as in doctors whose work routinely deals with death [41–43].

A 27-year-old doctor was referred for urgent assessment by the occupational health department due to recent difficulties in learning, memory and performance at work. She was a newly qualified house officer with busy on-call duties, and she had been off sick for a number of weeks. During assessment she complained of poor sleep with nightmares, phobic avoidance of going out leading to isolation and constant feelings of guilt and self-loathing. It emerged that she also suffered from flashbacks of a physical attack that had occurred several months before the onset of symptoms, which had left her shaken but not injured. Detailed examination showed no evidence of neurological abnormality, but she scored 25 on the Beck Depression Inventory signifying moderate to severe depression. She described fleeting thoughts of suicide. She was commenced on antidepressant therapy and offered cognitive behaviour therapy. Over the course of the next few months, her symptoms diminished, she was able to return to work and was looking forward to resuming her studies in the next academic year. She never required formal testing as her cognitive problems resolved following treatment of the emotional disorder.

Conclusions

Doctors presenting with performance problems may require a specialist assessment. Such assessments are complex and multidimensional as medical competence requires intact functioning of all cortical domains. In general, assessments need to be carried out by relevant experts, as many doctors and even occupational psychologists are not usually trained in assessing the complex interplay between higher cortical functions and behaviour. No quick questionnaire is available to perform this task, and it is very unlikely that it would be possible to construct such a test. The application of neuropsychiatry/neuropsychology to the routine assessment for doctors with complex performance problems may be the way forward to safeguard patient care.

Conflict of interest

None declared.

References