Delirium – how can we tackle the most elusive giant?

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Introduction

One of the biggest social transformations of the 21st century is that of population ageing. It has been predicted that within the next decade the number of adults aged 65 and over will outnumber the number of children under the age of five (1). Moreover, by 2050, the number of adults world-wide aged over 80 will reach 395 million, a four-fold increase since the year 2000 (1). With these changes in demographics come new healthcare challenges. The principal categories of impairment in old age which impact on the physical, mental and social wellbeing of older adults were described by Professor Bernard Isaacs as the “geriatric giants”, namely instability, immobility, incontinence and intellectual impairment (2). It is a common misconception that these conditions are an unavoidable part of ageing. However, they are often in fact non-specific initial presentations of acute illness and they can frequently be improved.

Perhaps the most difficult to measure of these ‘giants’ is confusion. It is estimated that between 25% and 30% of adults over the age of 85 have some degree of cognitive decline (1). Delirium in particular is a common and serious condition, especially among older people and, historically, it has been under-researched, certainly out of proportion to its prevalence and impact on clinical outcomes. The prevalence of delirium varies according to the patient population, method of study and risk factor being investigated. In hospitalised elderly people, rates may be between 14% and 56% (3)(4), with post-operative prevalence in this group reaching as high as 87% (5).

Delirium is sometimes referred to as ‘acute confusional state’ and is characterised by an acute disturbance of consciousness, cognitive function or perception, which has a fluctuating course. It is a medical emergency and its early identification and management as well as identification of the underlying cause is vital to reduce long-term morbidity and mortality.

The purpose of this report is to review the literature on delirium, including the current ideas on the underlying pathophysiology. The review was written following a five-week placement in a geriatric medical unit and includes case studies illustrating the various clinical presentations of delirium. It also includes a discussion of the current protocols used for the identification and management of delirium in hospitalised older adults.

What is delirium and where did the term come from?

Throughout medical literature, including in ancient and medieval times, there are descriptions of the syndrome that we now call delirium, albeit under many different names. There was a general consensus that it was usually caused by a febrile illness and that it denoted a grave clinical situation. The different
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subtypes were also often described, sometimes as forms of the same illness and other times as separate conditions. Delirium remains difficult to define and to study. The word delirium is derived from the Latin delirare, literally translated “off the track or furrow” or figuratively meaning, “to become deranged or mad”. Hippocrates observed a fluctuating, temporary change in mental status with physical illness and used several terms to describe the clinical syndrome we now call delirium (6). These included “phrenitis” (meaning frenzy), referring to a hyperactive form and “lethargus”, referring to a hypoactive form. Hippocrates also stated that phrenitis could change to lethargus without warning. In the first century AD, Celsus coined the word delirium as a medical term, using it interchangeably with phrenitis, to describe mental abnormalities during fever or head trauma.

It was not until the late 20th century that delirium was classified by consensus as a clinical entity, along with other mental disturbances, in the Diagnostic and Statistical Manual of Mental Disorders, third edition (DSM-III). This may have been a result of the demographic change in the population, resulting in more hospitalised patients in their 70s and 80s presenting with a complex interplay of physical and cognitive complaints (6). DSM-III classified delirium as an organic brain syndrome with the essential feature of a “clouded state of consciousness (reduced clarity or awareness of the environment)” with disorders of attention (7). The necessary criteria for delirium have been progressively reformed through DSM-III-R to the most recent version of this manual, the DSM-IV-TR (Table 1), which is now the gold standard for delirium diagnosis (8). Modifications in this version identify advanced age as a prominent risk factor for delirium and emphasize the importance of early recognition and management as well as the presence of hyperactive and hypoactive subtypes of delirium, which will be discussed further below.

Table 1 - DSM-IV-TR diagnostic criteria for delirium

<table>
<thead>
<tr>
<th>Criteria DSMIV- TR</th>
<th>Delirium</th>
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<tr>
<td>Criteria A</td>
<td>Disturbance of consciousness, with reduced ability to focus, sustain or shift attention</td>
</tr>
<tr>
<td>Criteria B</td>
<td>Altered cognition (memory, orientation, language disturbance) or the development of a perceptual disturbance (delusion or hallucination or illusion) that is not better accounted for by preexisting dementia</td>
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<tr>
<td>Criteria C</td>
<td>Disturbance develops over hours or day and tends to fluctuate during the course of the day</td>
</tr>
<tr>
<td>Criteria D</td>
<td>There is evidence of an aetiological cause</td>
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What does delirium look like?

Although by definition delirium has an acute onset and fluctuating course, and always involves attention and awareness deficits, it is otherwise heterogeneous, with the variable presence of numerous neuropsychiatric
symptoms. As a result of the multifactorial nature of delirium it can present very differently from patient to patient. Perhaps the most prominent manifestation of delirium is a disturbance of consciousness. The first part of this refers to the level of arousal, which ranges from fully alert to coma. Consciousness also refers to self-awareness and awareness of the environment but this requires a certain level of alertness (9). In an individual patient with delirium the level of consciousness may fluctuate between the extremes even within a single day.

Inattention is another cardinal feature of delirium and patients tend to be easily distracted by inappropriate stimuli during conversation or clinical interview. Deficits in cognition are another common feature of delirium. Patients' short and long term memory, orientation and understanding may all be affected (10). This may be confounded by inattention as patients may find it difficult to register new information and to remember instructions. Similarly, disorientation to time, place and to the identity of others is also common. These alterations in the thought process can also manifest as language difficulties and this may present as incoherent speech or the inability to find or pronounce words (11).

Disturbances in perception are also observed in patients with delirium and may culminate in psychosis with visual hallucinations, which tend to occur more often at night, and paranoid and persecutory delusions (10). Sleep patterns are also often disorganised and these may range from daytime somnolence to reduced sleep and complete reversal of the day to night sleep-wake cycle (11).

Increased or decreased psychomotor activity is another clinical feature of delirium. Consequently, delirium is classified according to psychomotor subtypes as hyperactive, hypoactive and mixed delirium. Increased agitation, hypervigilance and increased motor activity are seen in hyperactive delirium (Box 1), whereas a reduced level of consciousness, lethargy and inattentiveness are the main presenting factors in hypoactive delirium (Box 2). Many patients fluctuate between these motoric subtypes and are classed as having mixed delirium. Identification of these subtypes is clinically useful as they are each associated with different clinical aetiologies and treatment responses, but this is rarely done in routine clinical practice.
Hyperactive delirium is often associated with alcohol or drug withdrawal and adverse drug effects (12) (13). The hypoactive subtype has been seen more frequently in older adults and in the palliative care setting. It tends to be misdiagnosed, frequently as depression, or dismissed as clinically irrelevant. This form also tends to be associated with metabolic abnormalities such as hepatic encephalopathies and dehydration as well as hypoxia, and is correlated with a higher mortality than the hyperactive form (14) (15). This hypoactive presentation is one of the reasons why, despite its high prevalence in elderly medical patients, delirium is often undiagnosed (16).

Box 1: Case example 1

Mr M is an 83-year-old retired teacher who was admitted to hospital from his home following a fall in his garden. He had cataracts, diabetes and osteoarthritis but was on no regular medication. He underwent successful surgery for a fractured neck of femur. The surgery and post-operative recovery were unremarkable. However, his sleep had been disturbed since the night prior to his surgery. A few days following surgery, Mr M pulled out his intravenous cannula and became agitated and confrontational when nurses tried to replace it. He became paranoid and anxious that the hospital staff wanted him to be “locked up” and believed they wanted to take his organs. He had also been seen by nurses shouting at an empty chair to “go away”. During clinical consultation Mr M was noted to be “picking at the bed sheets” and was easily distracted by activity in the ward.

Prior to hospitalization, Mr M lived independently in the community. Collateral information was obtained from his daughter, who explained that her father experienced a similar episode during knee surgery three years previously. His son confirmed that Mr M had no history of drug or alcohol use.

Characteristics

This patient has several risk factors for delirium, including hip fracture, recent surgery and visual impairment. In addition, his older age and a suspected history of a previous episode of delirium also increase the likelihood of Mr M’s current symptoms being attributable to delirium. This case demonstrates deficits in attention and orientation, an increased sensitivity to the immediate surroundings, with restlessness and risk of aggression. This patient also demonstrates psychotic symptoms, with persecutory delusions and hallucinations. These are all features of hyperactive delirium.
Since the publication of diagnostic criteria, the term “subsyndromal delirium” has been added to the language to describe patients with one or more delirium symptoms that do not meet the full criteria. Subsyndromal delirium is estimated to occur in 21-76% of hospitalised older adults and it is a clinically important condition that is consistently associated with poor outcomes (17). It has been suggested that there is a continuum of acute neurocognitive disorders ranging from no delirium to full delirium, which is not taken into account in the DSM-IV-TR. In this notion, risk factors and symptoms are quantitatively related to adverse outcomes (18).

**What causes delirium?**

The aetiology of delirium is typically multifactorial but it maybe caused by a single factor, such as alcohol or substance abuse. Very generally, the cause of delirium is a medical illness or a result of treatment for a physical illness or accident e.g. medication or a procedure such as surgery (Figure 1).
There is significant evidence to support a number of consistent risk factors for delirium, and its aetiology in older adults involves a combination of precipitating and predisposing factors. Predisposing factors are risk factors that make the patient more susceptible to delirium while precipitating factors are acute factors that trigger delirium (19) (Table 2).

Existing cognitive impairment seems to be a significant risk factor for delirium and can lead to delirium superimposed on dementia (DSD). It is thought that existing dementia makes the brain of these patients vulnerable to the insults of acute physical injury or illness combined with environmental factors leading to delirium (11). Drug effects are another important risk factor for the development of delirium in older adults and medications may be the only precipitant in up to 39% of cases (20). Medications can lead to the development of delirium due to many factors. For example, the presence of poly-pharmacy (i.e. more than three different drugs) is a common problem in older adults and can lead to a combination of pharmacokinetic and pharmacodynamic interactions. The use of psycho-active drugs such as opiates and benzodiazepines and agents with anti-cholinesterase activity are also known to increase the risk of delirium (20).
Table 2 - Predisposing and precipitating biological factors associated with delirium (9) (21) (22) (23)

<table>
<thead>
<tr>
<th>Predisposing factors</th>
<th>Precipitating factors</th>
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<tbody>
<tr>
<td>Older age (65 years and over)</td>
<td>New illness (e.g. infection)</td>
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<tr>
<td>Polypharmacy (i.e. more than three drugs)</td>
<td>Metabolic and electrolyte derangements</td>
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<tr>
<td>Physical frailty</td>
<td>Iatrogenic complications</td>
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<tr>
<td>Co-existing multiple and severe medical conditions (e.g. heart failure)</td>
<td>Primary neurological conditions (e.g. acute stroke)</td>
</tr>
<tr>
<td>Cognitive impairment (e.g. dementia)</td>
<td>Surgery (particularly orthopaedic surgery)</td>
</tr>
<tr>
<td>Visual or hearing impairment</td>
<td>Drugs (particularly psychoactive drugs)</td>
</tr>
<tr>
<td>Depressive illness</td>
<td>Benzodiazepines</td>
</tr>
<tr>
<td>Neurodegenerative disease:</td>
<td>Sedative analgesics</td>
</tr>
<tr>
<td>Alzheimer’s disease</td>
<td>Drugs with anticholinesterase effects</td>
</tr>
<tr>
<td>Parkinson’s disease</td>
<td>Uncontrolled or prolonged pain</td>
</tr>
<tr>
<td>Prion disease</td>
<td>Environmental</td>
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<tr>
<td></td>
<td>Ward moves</td>
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<td></td>
<td>Use of restraints</td>
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<tr>
<td></td>
<td>Absence of clocks etc.</td>
</tr>
<tr>
<td></td>
<td>Dehydration and malnutrition</td>
</tr>
<tr>
<td></td>
<td>Alcohol excess</td>
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</table>

The pathophysiology of delirium is the subject of ongoing research; current knowledge in this area is still limited. However, there are several promising hypotheses. The mechanisms are thought to vary according to different clinical circumstances. A number of neurotransmitters have been implicated in the pathogenesis of the state of delirium, the most prominent being acetylcholine (Ach) and dopamine. These act in opposite ways: Ach reduces whilst dopamine increases neuron excitability. Cholinergic deficit with dopaminergic hyperactivity is thought to be the final common pathway in all delirious states (24).

The most widely accepted theory focuses on the direct deregulation of the neurotransmitter system by anticholinergic and dopaminergic agents. The activity of Ach may be reduced in several ways. Ach synthesis and release may be directly impaired by hypoglycaemia, hypoxia, metabolic disturbances or medications. Certain drugs may also compete with or inhibit Ach from binding to its receptors (25). Excess dopamine, which causes psychosis, is also thought to contribute to the pathogenesis of delirium, both alone and through the induction of a hypocholinergic state (26) (27). The interaction of the cholinergic and dopaminergic systems via the cortex, striatum, and thalamus with other neurotransmitters such as glutamate and GABA (gamma-aminobutyric acid) is also thought to contribute to form a complex acute imbalance (28). There is less certainty as to the significance of other neurotransmitters such as serotonin and noradrenaline. This theory is also supported by the fact that medication with anticholinergic and dopaminergic activity can evoke delirium and also by the fact that dopamine receptor agonists and, to some extent cholinesterase inhibitors, can provide symptomatic relief (22) (20).
There is accumulating evidence that delirium may be triggered by an inflammatory mechanism originating outside the brain (29). This may be the result of an inflammatory disease process or trauma leading to a systemic inflammatory response and increased cytokine production. This in turn leads to widespread microglial activity resulting in an inflammatory reaction in the brain (30). This has damaging effects on neurons and neurotransmitter function. This pathophysiological mechanism may be more significant in patients with existing neurodegenerative disease (31). Another hypothesis is that stress factors and trauma induce a sympathetic response by the body. This may be related to the exaggerated cortisol response to stress, associated with advanced age, dementia and other central nervous system diseases (31).

It is unlikely that these theories are mutually exclusive and a great deal of interaction exists between many of the aforementioned systems. Due to the nature of the multiple factors triggering delirium, the concept of a final common pathway seems to be most probable (Figure 2). Whatever the case there is clearly a wide range of potential targets for drug therapy. This presents the exciting prospect that perhaps in the future it will be possible to tailor delirium treatment to specific presentations based on underlying pathophysiology.

**Figure 2 - Summary of the "final common pathway" hypothesis for delirium pathophysiology**

**How is delirium diagnosed?**

In the absence of a single specific diagnostic test, delirium remains a primarily clinical diagnosis, involving a comprehensive clinical history and observation as well as physical and cognitive examination (11). Despite its high prevalence in elderly patients, delirium goes unrecognised in at least a third of cases (32). This is due to its overlapping symptoms with dementia and depression. This is especially true for older adults as they present more commonly with hypoaactive symptoms rather than the more obvious symptoms of agitation and
hallucinations. There is also a general under-appreciation of the importance of routine formal cognitive assessment. A recent survey indicated that although junior doctors in the UK were aware of the prevalence and significance of delirium, they lacked basic knowledge on the diagnosis and management of the condition (33).

The most recent national guidelines published by the National Institute for Health and Care Excellence (NICE) stipulate that all elderly patients admitted to hospital should be screened for risk factors for developing delirium and should be assessed for cognitive impairment (34). Risk factors specified by NICE include: age over 65, current hip fracture, severe illness and existing cognitive impairment. Cognitive function should be tested in those at risk using a brief test such as the Abbreviated Mental Test (AMT) (35) or Mini Mental State Exam (MMSE) (36). The MMSE and AMT are widely used objective measures of cognitive function in older adults. The MMSE is scored out of 30. The AMT consists of 10 items to assess orientation, memory and concentration. The use of the MMSE has become limited in recent years due to the enforcement of copyright.

If initial testing reveals altered cognitive function, perception or social behaviour then a clinical assessment based on the DSM-IV-TR criteria or using the short Confusion Assessment Method (CAM) (37) is warranted to confirm the diagnosis of delirium. The short CAM includes a diagnostic algorithm (Table 3), based on four key features of delirium and is designed for use by non-specialist staff after some training (38). NICE specifies that a healthcare professional trained in the evaluation of delirium should carry out this assessment. The CAM should be scored immediately following observations made during the interview with the patient and in the brief formal cognitive assessment. In this situation the CAM has excellent sensitivity (86%) and specificity (93%) for identification of delirium (39). It should also be noted that variations of CAM have been developed for use in special clinical situations such as in intensive care (40) and long-term care facilities (41).

Table 3 - The short Confusion Assessment Method (short-CAM) criteria for delirium

| Feature 1 | Sudden onset and fluctuating course |
| Feature 2 | Inattention (for example serial seven test with reduced ability to focus, or shifts in attention) |
| Feature 3 | Disorganized thinking (for example rambling or incoherent speech) |
| Feature 4 | An altered level of consciousness (for example the person is agitated or lethargic) |

For a diagnosis of delirium to be made CAM requires the presence of features 1 and 2 and either 3 or 4.

It is not possible to diagnose delirium based on a single assessment of cognition. In order to take into account the fluctuating course of delirium symptoms, patients should be assessed more than once during the day. Furthermore, delirium is by definition an acute condition and in order to determine whether the patient’s symptoms are new it is important to ascertain
their baseline function. This can be achieved by obtaining records of previous cognitive testing or from collateral information from other sources including family, carers or other healthcare professionals (42).

If delirium is diagnosed a thorough assessment must be made to determine the underlying cause. Therefore, it is important that thorough physical and neurological examinations are used to help identify infection or disease (4). Although brain imaging does not aide the diagnosis of delirium, it can sometimes be useful if there is evidence of an intracranial cause on examination (21). A review of medication and identification of any substance usage, including alcohol and benzodiazepines, is also important to identify potential causes of delirium.

**Delirium and dementia**

Delirium is a medical emergency that needs to be dealt with immediately by a team of appropriate medical and allied health professionals. One of the most common issues with the diagnosis of delirium is differentiating it from dementia. More specifically, simple delirium must be separated from delirium superimposed on dementia (DSD) or whether the symptoms are caused by dementia alone. For example, one of the differentiating features of delirium is the change in consciousness. It is estimated that the rate of DSD in older adults with dementia ranges from 22-89% (43). It seems likely that the majority of patients with dementia will have at least one episode of DSD at some point in their life.

Deterioration in mental status in hospitalised patients should be considered to be delirium unless determined otherwise (16). Careful consideration should be given to the possibility of delirium using an appropriate instrument before embarking on a series of tests for other major neurocognitive disorders. It may be more appropriate to delay extensive cognitive evaluation until 6-8 weeks after the acute episode has resolved. This could help avoid confusing symptoms of delirium for those of dementia. According to DSM-IV-TR, dementia cannot be diagnosed until delirium is ruled out (8). Patients who have a diagnosis of dementia should be regularly monitored for delirium, even if it has been previously ruled out and appropriate steps towards delirium prevention should be taken.

**Why is delirium so important?**

The presence of delirium has a negative impact on clinical outcomes in ill patients, including increased mortality, length of stay, and need for long term care (11). Delirium in older adults has been shown to have a negative impact on prognosis, with a 5-year survival rate in patients with delirium comparable to people with severe dementia, and on average a survival rate 50% lower than those without delirium (44).

Delirium has been shown to increase the incident risk of dementia eight fold and to lead to deterioration in global intellectual function and increased dementia severity (45). This demonstrates that apart from the established
immediate consequences of delirium, there is mounting evidence that even a single episode of delirium can be detrimental to cognitive function in the long-term and may more than double the risk of dementia (46) (47). Patients with pre-existing dementia who subsequently experience delirium during hospitalization have a two-fold increased risk of mortality in the 12 months following discharge (48).

The concept that delirium may not be as transient as once thought, coupled with an ageing population, has the potential for disastrous consequences, with more patients having long-term or permanent cognitive dysfunction, leaving them unable to live and work independently. This in turn would lead to an increased need for long-term care and a higher demand on health and social services.

**How can delirium be managed?**

Perhaps the most important thing about delirium is that it is preventable in an estimated 30–40% of cases (49) (19). Preventive strategies have become increasingly important, as knowledge of the risk factors has increased. A number of interventions have been recommended to reduce modifiable risk factors in individual patients and studies have shown that they can successfully reduce the incidence, severity and duration of delirium (19).

One important example of successful interventions to prevent delirium comes from Yale, the Hospital Elder Life Program (HELP) (50). This initiative, which is now adopted internationally at more than 60 sites, aims to provide standardised interventions for delirium prevention by managing six key risk factors (cognitive impairment, sleep deprivation, immobility, visual and hearing impairment, and dehydration) using multidisciplinary teams. Delirium is best prevented in this way using multicomponent interventions due to the multifactorial nature of its precipitants. Effective strategies for delirium prevention as advocated by NICE are listed in table 4 (34). Although the interventions are individually small, together they lead to significant clinical improvement.
Table 4 - Strategies for the prevention of delirium in hospitalised older adults (34) (51)

| Orienting communication and therapeutic activities | • Regular conversation to reorientate to surroundings and time of day  
| • Provide stimulating activities such as reminiscence sessions and art work  
| • Encourage visits from family and friends  
| • Provide familiar objects or pictures from the patient's home |
|---|---|
| Maintain mobility | • Encourage mobilisation following surgery or range of motion exercises |
| Maintaining nutrition and hydration and prevent constipation | • Encourage patients to eat and drink  
| • Ensure dentures fit properly |
| Pain management | • Regularly assess for and manage any pain |
| Reduce the risk of infection | • Avoid catheterisation unless essential  
| • Follow infection control procedures |
| Reduce sleep disturbance | • Maintain appropriate lighting  
| • Avoid procedures and medication rounds at night |
| Ensure adequate vision and hearing | • Ensure working hearing and visual aids are available to those who need them |
| Prevent hypoxia | • Regularly assess for hypoxia and optimise oxygen therapy if necessary |
| Avoid polypharmacy | • Regularly review medications and discontinue any that are no longer required or could be avoided (e.g. psychoactive drugs) |

The primary goal of delirium treatment should be to promptly identify and treat the underlying cause. Timely management leads to reduced duration and severity of delirium (52). Discussion of possible treatments for medical illnesses that could cause delirium is beyond the scope of this article. The focus of this discussion is on treatment and management of delirium symptoms themselves.

A variety of non-pharmacological measures can be implemented to provide orientation and a comfort while maintaining patient safety. Measures for managing delirium are similar to the preventive strategies. Maintaining a good sensory environment with appropriate lighting and noise levels can reduce confusion. Patients with sensory impairment should be encouraged to wear their glasses and hearing aids. Language barriers should also be addressed through family members or interpreters. It helps to have clocks and calendars visible to improve orientation in time and staff should regularly reorientate patients through clear communication. Continuity of care and avoiding non-clinically indicated ward moves is also recommended. The management of delirium can be challenging on any ward, and the idea of “delirium rooms” has also been advocated. Here, trained staff can maintain a calm comfortable environment and non-pharmacological interventions are standard practice (53).
For hyperactive delirium, the use of physical restraint should be avoided as it puts patients at risk of harm and may worsen agitation (53) (51). Furthermore, pharmacological treatment should be avoided where possible as drugs may lengthen or worsen delirium episodes. Instead, verbal and non-verbal de-escalation techniques should be used to re-direct or calm patients. Off label use of low dose haloperidol or olanzapine are indicated only when a patient is a threat to themselves or to others (54). Treatment should not exceed one week in duration. Neuroleptics should be completely avoided in patients with Parkinson's or Lewy body dementia (lorazepam may be used an alternative in these patients) (23) (34). An increased risk of stroke has been associated with the use of the antipsychotic drugs risperidone and olanzapine in patients with DSD and thus these should be avoided (55) (56).

**Conclusion – what can be improved?**

Lack of understanding of delirium has played a part in hindering education about this syndrome. However, as our knowledge of neuroscience increases, and the underlying pathophysiology of delirium is delineated from that of conditions with similar symptoms, the syndrome will continue to be elucidated. This will hopefully improve communication about the condition and the care of the patients it affects. Moreover, the mechanisms underlying delirium and its link to on-going cognitive impairment must be explained if the care need is to be reduced in the long run.

The unenlightened view that memory loss is a normal part of ageing should be discouraged and it is important to remember that cognitive and behavioural symptoms of delirium can be subtle and difficult to detect. A raised level of awareness of these changes and of the idea of three overlapping syndromes of delirium, dementia and depression could increase the rate of detection. Education is required to discourage the use of the terms “poor historian”, “poorly motivated” or “vague” to describe older patients, as these problems might indicate underlying cognitive impairment or depression (57).

The simple use of the term “confusion” as a diagnosis limits clinical thinking. The idea that cognitive assessment is too complicated or lengthy to be carried out as one of the core assessments at the time of presentation should be overcome. A patient with confusion should receive a formal cognitive assessment just as a patient presenting with cardiac chest pain would be offered an ECG. Perhaps, embedding brief cognitive assessment tests into patients’ notes along with prompts to talk to carers could improve ease and speed of use.

Unfortunately, the UK is yet to see a widespread systematic plan to exploit the full potential of delirium prevention. Healthcare workers and policy planners continue to follow current trends of heart disease and stroke prevention. The lack of attention to delirium is concerning, given the scope of the problem and the associated complications; it is certainly a concern as the ageing population expands and longevity increases.
Rates of non-detection of delirium remain high and the most obvious solution is to improve education at all levels to reflect its clinical impact. In the UK, old age psychiatrists are still considered responsible for diagnosing cognitive dysfunction and excellence in delirium care is not considered among the core skills of a good doctor. However, the changing demographic and public pressures on hospitals mean this is no longer tenable. The profession must adapt and doctors must take the lead in order to tackle this elusive geriatric giant.

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