Letters to the editor

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Delirium: a guide for the general physician

Editor – I offer several comments on the interesting article from Todd and Teale. Circular reasoning about dementia is the rule rather than the exception in the delirium literature. For example, Todd and Teale state that dementia is the most significant risk factor for delirium. In fact, the opposite is equally common: dementia is falsely counted as delirium. This occurs because 1) the clinicians have not spent at least 30–45 minutes with family members and other informants searching the time course of present symptoms and the behavioural antecedents; 2) clinicians have not performed domain-specific cognitive tests such as digit span forward for inattention. For example, an 83-year-old man with dementia developed sudden agitation, physical and verbal aggression. Based on sudden onset of confusion, agitation and aggression, he was mislabelled as delirium. A more thorough history revealed that the aggression came after he was treated like an imbecile in a specialist clinic. Everyone talked over him as if he did not exist. Aggression stopped on arrival in the emergency department and did not recur. It would be highly unusual for delirium to clear in two hours so behavioural and psychological symptoms of dementia (BPSD) were the problem rather than delirium. Fluctuating course with altered level of consciousness occur in diffuse Lewy body dementia or diffuse Lewy body mild cognitive impairment.

The authors do not highlight the drawbacks in the 4AT in dementia. Months of the year backwards (MOYB) is usually normal in mild cognitive impairment but abnormal in moderate or severe dementia. 4AT also yields false positives after sedatives or antipsychotics in the emergency department or ward.

The authors assert that a confused patient has delirium until proven otherwise. This assumption can lead to unnecessary investigations and hospital admission.

In Box 4, the authors suggest arterial blood gases. Arterial puncture is difficult in agitated or suspicious patients so a venous PCO₂ and finger oximetry will be more patient-friendly. Box 4 omits measuring serum drug levels (eg lithium) for those on psychoactive drugs, troponin and occasionally creatinine since symptom onset.

The Central Coast Australia Delirium Intervention Study (CADIS) demonstrated a more specific method to identify delirium by adding three features to the Confusion Assessment Method:

1. meticulous exclusion of BPSD by interviews with family, carers and residential care members
2. not using disorganised thinking, fluctuating cognition or impaired level of consciousness for delirium diagnosis when these may have been caused by antipsychotics or sedatives since symptom onset
3. at least a 25% recent decline in attention of 5- and/or 6-digit span forward using imputed values for baselines.

This CADIS phenotype produced 116 delirious patients with extremely rapid onset and almost as rapid reversal of neurocognitive impairments.

I have recently shown that in 405 medical articles on delirium (ie not intensive care or postoperative) with 789,709 patients, only 2.7% of articles reported mean onset in days and only 6.2% reported the mean or median days to recovery. In other words, the bulk of delirium articles describe an event of unproven times of onset or recovery. The CADIS phenotype deserves further study and analysis.

Conflicts of interest

The author has no conflicts of interest to declare.

Paul Regal
Geriatrician, Senior Lecturer 2007–16, Regal Elderly Medicine, Wyong, Australia

References


The new UK internal medicine curriculum

Editor – I read Professor David Black’s article describing the new UK internal medicine curriculum with interest. It would seem at long last there is a plan to simplify the greatly criticised ‘tick-box approach’ to medical training. This is hardly a new idea and this progress has been greatly hampered and delayed by the Joint Royal Colleges of Physicians Training Board (JRCPTB) themselves.

I, and others, were on the Royal College of Physicians Trainee Committee almost a decade ago telling Professor Black’s predecessor, Bill Burr, that we should be doing far fewer assessments and we should do them better. We also said at the time that the process of linking competencies (one consultant seeing you clerk someone with acute coronary syndrome, doing a case-based discussion and another consultant deeming you therefore ‘competent’) is academically absurd.
I hope Professor Black is genuinely planning to get a grip on this aspect of training. To speak frankly, as it stands the ePortfolio is a crushing piece of bureaucratic nonsense. If you set out to develop an education initiative with the sole aim of making training less enjoyable you would struggle to beat this. There is no doubt in my mind that the ePortfolio and assessments (and by extension JRCPTB) have been a large driver of falling morale among medical trainees. As things stand, there are too many assessments; yes, they are clunky and the IT is abysmal but the real enemy here is the ‘linking’, so while the focus on outcomes is welcomed, I hope this is also tackled. Currently, a trainee has to link evidence to each part of their ePortfolio, then write about why they are competent in that area and then ask their supervisor to countersign that they are. The idea that the linking process will highlight the failing trainee, or even contribute meaningfully to the training of good trainees, is laughable.

While I congratulate Professor Black on trying to improve things and reduce the tick-box culture (that his organisation was instrumental in creating), it might be prudent to consider why they were unresponsive to criticism for so long. Certainly when I argued for simplification of the system in 2008 I was treated as a wayward schoolboy who, perhaps by virtue of not having paid for a masters in medical education, was unenlightened. When future trainees bring up genuine concerns about the next curriculum iteration these can’t be dismissed so easily.

**Conflicts of interest**
The author has no conflicts of interest to declare.

**RODRIC JENKIN**
Consultant in acute and geriatric medicine, Whittington Hospital, London, UK

**Reference**

**The latest national clinical guideline for stroke**

Editor – There is lack of clarity in the latest *National clinical guideline for stroke* on cardiac monitoring if a cardio-embolic stroke is suspected.

It is estimated that between 25 and 60% of atrial fibrillations are paroxysmal (PAF) and approximately one third of the patients affected are asymptomatic.

Thirty percent of all strokes remain without an identifiable cause even after extensive workup. Occult PAF seems to be one of the culprits of ‘cryptogenic’ strokes.

All ischaemic stroke patients should have a standard 12-lead electrocardiogram (ECG), which will detect a new PAF in 2–4% of patients.

Cardiac telemetry for 28–72 hours after index hospital admission has been reported to detect new AF in up to 2.4–18.5% of patients with acute ischemic stroke. This facility, however, is often used to monitor patients receiving systemic thrombolysis.

Serial ECG assessments within the first 72 hours of an acute stroke significantly improve detection of AF. This strategy is particularly useful when cardiac telemetry is not readily available.

Given the asymptomatic nature of PAF, patient-activated cardiac monitoring devices are of low clinical value. Most patients in the UK will receive a single 24/48/72-hour Holter monitor after a cryptogenic stroke as per the current guideline (Fig 1). The diagnosis of PAF is often missed because of low rates of arrhythmia detection by these devices. These patients will thus miss out on anticoagulation, leading to recurrent strokes and a higher mortality.

A 7-day ambulatory ECG monitor detects AF in 5.7% of patients with a normal standard ECG and 24-hour Holter. In our trust, 7-day ambulatory ECG monitors are used instead of 24/48/72-hour Holter to detect occult PAF.

The EMBRACE (Event Monitor Belt for Recording Atrial Fibrillation after a Cerebral Ischemic Event) study demonstrated that the 30-day event-triggered recorder was significantly more effective than 24-hour Holter for identification of AF in patients with recent cryptogenic strokes. My practice is to perform a 32-day ambulatory ECG monitoring if the 7-day ECG does not detect low-burden PAF.

Surface ECG recorders (7- or 32-day ECG monitor) rely on skin contact electrodes. Skin irritation or ‘allergic’ reactions have been the commonest cause of non-compliance in my practice. This makes it difficult for patients to wear these devices for long durations.

AF detection rate in the CRYSTAL AF (Cryptogenic Stroke and Underlying AF) study were 8.9% at 6 months, 12.4% at 12 months and 30% at 3 years in those with cryptographic stroke. In view of this evidence, I refer patients in whom prolonged ECG monitoring has failed to detect PAF, have developed allergic reactions to skin contact electrodes or do not wish to wear devices for 32 days to a cardiology colleague for an insertable loop recorder.

Finally, dual-chamber pacemakers and implantable cardioverter defibrillators are cardiac implantable electronic devices. These in situ intra-cardiac devices can be programmed for continuous ECG monitoring in patients with suspected cardio-embolic stroke to detect asymptomatic atrial tachyarrhythmia.

**Conflicts of interest**
The author has no conflict of interest to declare.

**SHAHID A KAUSAR**
Consultant in stroke, geriatrics and general (internal) medicine, Russells Hall Hospital, Dudley, West Midlands, UK

**References**