

MORTALITY IN OUT-OF-HOURS EMERGENCY MEDICAL ADMISSIONS

I read with great interest the recent paper by F Maggs and M Mallet (Mortality in out-of-hours emergency medical admissions – more than just a weekend effect. *J R Coll Physicians Edinb* 2010; 40:115–8), regarding the mortality among the emergency admissions during out-of-hours periods in an NHS hospital. From time immemorial, UK hospitals have had increased admissions on Mondays and Fridays and only those very ill got admitted out of hours. It is not surprising that the authors' findings correlated well with the previous studies. Of course, with changing working pattern and the current new medical training system, continuity of care has been a major problem, which perhaps could be a contributory factor.

Although NHS hospitals run 24/7, out-of-hours care is done by limited numbers of staff who have been inundated with ever-increasing referrals, which could affect the morale of the team as well as the delivery of effective care.

While the single-point access to emergency admissions remains with secondary care, in my opinion radical changes need to be made with regards to the management of referrals to the admissions unit. While Lord Darzi's plan of polyclinics may be the harbinger of that process, even during the working hours and if possible extended evening hours, medical assessment unit clinics or accident and emergency clinics should be set up. This should expedite the assessment and not fill beds until patients have been reviewed.

This might be difficult to start with, but having worked in that system in the Midlands myself, I do feel that this needs to be pursued and that it would be fit for purpose in the current economical climate.

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Further reading

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- Cei M, Bartolomei C, Mumoli N. In-hospital mortality and morbidity of elderly medical patients can be predicted at admission by the modified early warning score: a prospective study. *Int J Clin Pract* 2009; 63:591–5. doi:10.1111/j.1742-1241.2008.01986.x
- Jones S, Barnett N. Data briefing. Why admission day affects outcomes. *Health Serv J* 2008 Oct 30:17.

Authors' reply

As stated in our paper, the reasons behind increased mortality rates relating to the day of admission are likely to be multifactorial, and Dr Nallasivan considers a few of these potential factors. Our acute medicine clinics for

early assessment and avoidance of unnecessary admissions may indeed be one of the factors contributing to our findings.

Dr F Maggs, Dr M Mallet

WARFARIN OR NOT

Dr Neilson raises the question of whether warfarin should be replaced by dabigatran in the prevention of venous thromboembolism (VTE) and pulmonary embolism (PE) (*J R Coll Physicians Edinb* 40:222–3) and Professor Hyers and colleagues debate the optimal length of therapy (*J R Coll Physicians Edinb* 40:224–8).

Having had a near-fatal PE a year ago, based on breaking an ankle after falling out of a plum tree, I can assure readers that this is not an experience I want to be repeated. Since the difference in bleeding risks between dabigatran and warfarin is minimal and dabigatran is not yet licensed in the UK for the prevention of VTE, I shall certainly continue on warfarin for two and probably more years.

The burden and inconvenience of monitoring the international normalised ratio (INR) can be reduced by the purchase of a simple home kit (Roche, about £400) for which the basic check strips can be supplied through the NHS. I have now used this for nearly a year with confidence and without the need to visit any anticoagulant clinic, although it is reassuring to report by telephone or email the monthly INR results to such a clinic.

Professor Michael Oliver

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Author's reply

I am grateful to Professor Oliver for his comments. I am also sorry that he has suffered a pulmonary embolus which I agree is not to be repeated nor wished on anyone else. However, I think he has misrepresented my points on the modern anticoagulant agents.

Warfarin has only lasted so long as an oral anticoagulant as there were no alternatives as effective. Sadly this efficacy has come at a price. The scope for the occurrence of an adverse event related to warfarin is large because of the biological variation in response to treatment and because of the potentially large number of individuals and structures involved in a patient's care. While this may be reduced by home monitoring, it is still the case that warfarin therapy in itself is associated with significant morbidity and mortality. The risk of haemorrhage on long-term oral anticoagulation ranges between 1% to 15%, increasing with a rising INR. Warfarin in primary care is one of the medicines most commonly associated with fatal medication errors. According to

the National Patient Safety Agency, it was associated with 480 episodes of harm or near-harm between 1990 and 2002 and 92 deaths in the same period.¹

Contrary to Professor Oliver's comments, dabigatran, as an oral direct thrombin inhibitor, is associated with a statistically significant lower risk of haemorrhage compared with warfarin. It also does not require laboratory monitoring. The licensing issues surrounding the newer anticoagulants are ones that are being met as the trials mentioned in my paper produce their findings.

When I qualified as a doctor in the 1980s drugs such as alpha-methyl dopa, heminevrin, paraldehyde and thioridazine were regularly and indiscriminately prescribed. With the introduction of better drugs with fewer side effects their use fell away. Hopefully warfarin will soon join this pharmacological antiques roadshow.

Dr RF Neilson

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OPTIMUM DURATION OF ANTICOAGULATION FOR IDIOPATHIC VENOUS THROMBOEMBOLISM

In the latest current controversy (What is the optimum duration of anticoagulation for the management of patients with idiopathic deep venous thrombosis and pulmonary embolism? *J R Coll Physicians Edinb* 40:224–8) Drs HG Shetty and IA Campbell stated that the presence of thrombophilia could make it easier to decide about long-term anticoagulation. I feel that this statement requires clarification in the light of published evidence and guidelines. The majority of patients presenting with venous thromboembolism will not have a known diagnosis of a thrombophilic trait and the statement therefore raises the question as to whether testing is of clinical utility in this situation.

Current North American¹ and British guidelines² are both in agreement that the presence of hereditary thrombophilia should not influence decisions about the duration of anticoagulation following a first unprovoked venous thromboembolic event. Prospective studies (reviewed in the above guidelines) have indicated that the risk of recurrent venous thromboembolism following a first event is only very modestly increased in patients with *Factor V Leiden* or the *G20210A* prothrombin gene mutation.

In individuals with deficiencies of the natural anticoagulants (protein C, protein S and antithrombin) the risk of recurrence is more difficult to estimate due to the smaller numbers of patients with these disorders. However, prospective data again indicate that the relative risk of recurrence is likely to be <2.0 in those without a strong

family history of thrombosis. Excluding patients with a deficiency of natural anticoagulants, a history of venous thromboembolism in one or more first-degree family members has no effect on the risk of recurrence after a first unprovoked venous thromboembolic event. The implication of these findings has been confirmed by a large study which found that testing for hereditary thrombophilia, after a first venous thromboembolic event, did not reduce rates of recurrence.³ The British guidelines therefore conclude that indiscriminate testing for heritable thrombophilia in unselected patients after a first episode of venous thromboembolism is not recommended.

In contrast, the presence of the acquired thrombophilia of the antiphospholipid syndrome is associated with an increased risk of recurrence after the first venous thromboembolism and long-term anticoagulation should be considered after a first event. Testing for this should therefore be considered in young patients or those with suggestive features.

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WHY DOES A HOSPITAL ADMISSION REMOVE THE RIGHT TO VOTE?

The UK has recently undergone a general election in which the future of the National Health Service (NHS) and social care reform have been key issues. The debates surrounding these issues show how they could potentially become a pivotal component of many future electoral discussions.

Older people constitute a large proportion of the electorate and are frequent users of the health services. According to recent political science studies, age often has an effect on voter turnout rates, with older voters turning out more frequently than younger voters. One such study, undertaken by De Montfort University, projected that over 55s will account for the majority of votes cast in 319 constituencies across Britain in this recent election.¹ This fact is increasingly important as the number of people aged 85 and over is projected to rise from 1.3 million in 2008 to 1.8 million in 2018 (an increase of 34%) and to 3.3 million by 2033, double the number in 2008.² If not managed effectively, this could have a massive impact on the 159,386 beds currently available in the NHS.³ This set

of individuals will therefore require the support of society and, more importantly, the government, to ensure that their social care packages meet their needs. Any policies by the main political parties of the day will require great thought as the correct manifesto pledge could turn this large-scale vote into a winning seat.

The *BMJ* recently published an editorial 'Where now for social care in England?'⁴ that outlined the five potential funding options for a social care system from a 2009 green paper, 'Shaping the future of care together'.⁵ These potential funding mechanisms are a source of major concern for many older voters. Unfortunately, like the people who could not vote on polling night due to various problems, neither could the inpatients on our ward. This lack of vote is astonishing whether it is due to a mix-up at the polling station or simply due to a lack of access. Similar to society enforcing disability on people by not supplying appropriate facilities for less able individuals, people without a vote are disabled in their right to say who is in control of their destiny.

Our reply⁶ to the *BMJ* editorial stimulated some interesting viewpoints regarding the vote of older people in the UK and its impact. We wrote: 'One might ... ask what provisions are placed in position for (older) people in hospital to empower the leader they believe will support their social care needs, when their only voice is taken away: their vote... Although postal voting has recently seen an increase, this has to be arranged in good time prior to the election and a substantial number of voters still prefer to attend the polling station. In addition, many of those admitted to hospital may be caring for their spouses, who now also happen to have had their vote removed as nobody is present to take them to the voting booth.'⁶

With all the information noted above – how can one realistically state that a true reflection is being gained from older voters, when no access is being supplied to a proportion of them to cast their votes? In light of all the pledges made by the government to advance voting reform, an integral section of society appears to have been missed.

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WE 'AIRBRUSH' THE EVALUATION OF JUGULAR VENOUS PRESSURE OUT OF CLINICAL ASSESSMENT OF BLOOD VOLUME STATUS AT OUR PERIL

Among the physical signs listed for the assessment of blood volume status in the diagnostic testing of hyponatraemia (Table 5 in Ball SG. Hyponatraemia. *J R Coll Physicians Edinb* 2010; 40:240–5), the evaluation of jugular venous pressure (JVP) was notable by its absence, notwithstanding its advantages over the evaluation of lying and standing blood pressure in severely ill patients.¹ In the supine, ill patient with hyponatraemia, when the 'jugular veins are flat' (i.e. not distended) this is likely to signify hypovolaemia and to justify a fluid challenge.² In that context, the advantage of evaluating JVP in addition to postural change in blood pressure is that in an ill patient who is 'unable to stand for vital signs' JVP can be evaluated without having to get the patient out of bed, both for the identification of hypovolaemia and for the identification of hypervolaemia.

By contrast, changing the patient from the supine to the sitting position 'instead of standing up the patient' markedly reduces the clinician's ability to detect postural changes induced by blood loss¹ and, by inference, postural change induced by any other mechanism of blood volume depletion. Furthermore, postural hypotension, defined as a >20 mm Hg fall in systolic blood pressure (BP), after assuming the standing position, occurs in up to 10% of normotensive subjects aged <65, and in 11–30% of normotensive subjects beyond the age of 65 with normal blood volume, thereby reducing its likelihood ratio to an amount close to unity.¹ At the other extreme, in the context of fluid overload, 'when performed properly by experienced physicians JVP estimation is fairly accurate', being characterised by positive and negative predictive values of 85% and 62%, respectively.³

The disrepute into which the evaluation of JVP has fallen is, arguably, attributable to the fact that it has now become a skill which is badly taught, hence the proliferation of evaluations where this parameter is documented as 'JVP raised 2 cm'. There are, however, important implications to suboptimal skills in the evaluation of JVP. These include not only the failure to use JVP as a back-up for the evaluation of a postural fall in BP, especially when the patient is too ill to stand up,¹ but also failure to use an elevation of JVP as a back-up for the validation of heart

failure diagnosis when the left-ventricular ejection fraction (LVEF) is intact, and there are caveats to sole reliance on raised levels of brain natriuretic peptide.⁴

Consequently, where the evaluation of JVP is suboptimal, 'its use in multicentre studies [such as those for evaluating treatments for heart failure] is not reliable without adequate audit of clinical expertise in each centre'.³ The latter observation is, arguably, accountable for the paucity of clinical trials (which require multicentre participation) into the treatment of heart failure characterised by intact LVEF because, in the latter context, greater reliance will have to be made on clinical than on laboratory criteria for the validation of heart failure diagnosis.

Raised JVP, however, was documented in only 6.6% and 8.1% of patients allocated to candesartan and placebo, respectively, in the CHARM-Preserved Trial evaluating the role of angiotensin receptor blockade in heart failure patients with LVEF >40%. In that trial, arguably as a result of inadequate audit of clinical expertise in each centre, greater reliance was placed on New York Heart Association status and on the 'history of hospital admission for a cardiac reason' for the purpose of validating the diagnosis of heart failure.⁵

In conclusion, especially in an ill patient who is unable to stand for 'vital signs', an evaluation of JVP should be used to complement the evaluation of the postural changes in BP occasioned by change from the supine to the sitting up position. Where the patient is able to stand, the suboptimal likelihood ratio of postural hypotension for the validation of hypovolaemia can be mitigated by the adjunctive use of postural pulse increment of 30/min or more attributable to hypovolaemia.¹

Finally, skills used for the evaluation of JVP so as to distinguish hypovolaemic hyponatraemia from hypervolaemic hyponatraemia are translatable to the wider arena of validating the diagnosis of heart failure when enrolling patients in multicentre clinical trials of heart failure treatment.

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SOME MUSICAL MEDICAL THOUGHTS

On percussion: Percussion of the chest produces sounds that are traditionally graded as 'resonant' or 'dull'. There is confusion as to what these terms mean. Some think it is volume, whereas others (rightly in my hearing) think it refers to the pitch of the note. The difference between stony dullness and resonant, implying pleural liquid is approximately an octave, whereas the difference between dullness and resonant, usually implying consolidation, is approximately the musical interval of a fourth or a fifth.

On auscultation: Rheumatologists attach significance to joint crepitus, usually detected by palpation during joint movement. It is far easier to listen for crepitus using a stethoscope. I have the impression that the sounds produced by various pathologies might be quite distinct (foreign bodies, osteoarthritis, torn cartilages).

Doctors, when dealing with deaf patients, often shout. On multi-bedded medical wards this makes nonsense of confidentiality. In the absence of hearing aids one can use the stethoscope as an ear trumpet – put the earpieces in the patient's ears and use the stethoscope diaphragm as if it were a microphone.

There are two main descriptions of cardiologists, non-invasive and interventional. There should be a third, essential for those who teach – singing cardiologists who can counter failures to hear sounds at pitches at which we are not expecting to hear them.¹ Few students hear the high-pitched murmur of aortic incompetence, even when told the murmur is high pitched. They need a cardiologist to sing *luptush* at the same pitch as the murmur. Similarly mitral stenosis in sinus rhythm is the opening four notes of Beethoven's *Fifth Symphony* – LUP t t ruv (LUP = the loud mitral first heart sound; the first t = closure of the aortic valve; the second t = the opening snap; ruv = the low-pitched rumbling mid-diastolic murmur with a pre-systolic crescendo if the patient is in sinus rhythm).

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Answers to the cannabis and the lung MCQs on pages 333–4:

1: D. 2: C. 3: A. 4: E. 5: D.