Letters to the editor

Delays in the referral and acceptance of patients with subarachnoid haemorrhage to specialist care – a call for a national conversation

We would like to highlight a recurring issue in the referral of patients presenting to district general hospitals (DGHs) with subarachnoid haemorrhage (SAH). Guidance from NICE suggests that patients with SAH should have rapid access to appropriate specialist care.[1] In reality, the referral, acceptance and transfer to a specialist centre is often far from rapid.

Many will recognise the scenario of a patient presenting to the emergency department with sudden onset of severe headache along with 'red flag' signs. They are scanned quickly and a diagnosis of SAH is made. On referral to Neuroscience Centre A, the history and scans are relayed to a neurosurgical trainee. Instead of receiving an immediate decision on whether the patient is suitable for acceptance and whether there is a bed available, there is often a time delay in which the patient's history is further relayed to a consultant. If the patient is accepted by the team at Centre A, it is not uncommon to find, after a further time delay, that the intensive care unit is full. The process is then restarted by the DGH with Neuroscience Centre B. After more delays, if it is ascertained that no bed is available at this centre, Neuroscience Centre C must then be contacted. With no system in place to link images to neuroscience centres other than the centre immediately affiliated with the DGH, the process is more difficult. Some time having elapsed, and the patient's condition possibly having deteriorated in this time, a further CT is sometimes requested before acceptance of the patient to Centre C, several hours after presentation of the patient to the DGH.

We propose a number of solutions to the problems outlined, which if agreed on a national level, could help eliminate the unnecessary delay, repetition and procrastination which often occur in the referral of SAH patients. We suggest the adoption of a single point of contact at a national level, either such as that seen with patients who need extra-corporal membrane oxygenation or such as that seen with co-ordinated transfer in UK paediatric services.[2,3] We think consultant-to-consultant level discussion is warranted for the management of these patients. Additionally, national protocols could provide guidance to optimise management prior to transfer and could indicate clearly which patients would not be suitable for transfer. This could even help alleviate some of the demand for specialist beds as some patients could be managed in general intensive care units, as has been suggested for traumatic brain injury patients.[4]

At the very least, we would like to see a national conversation on how best to serve patients with SAH presenting to DGHs, who need urgent access to neurosurgical expertise.

Declaration of Interest

Carl Waldmann is a co-editor JICS.

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Response: 'D minus – not good enough.'

There is no argument with the case presented by Sodha and colleagues. Like so many services in the NHS, neurosurgery has developed in a rather *ad hoc* manner across the country. The latest Department of Health figures indicate that there are 249 specialist neurosciences critical care beds in England comprising 116 level 3 and 133 level 2 beds.[1] The beds are not evenly distributed between units, with the largest having 22 beds and the smallest only 6. A snapshot audit of neurocritical care capacity conducted in February 2006 found 84 patients with a primary neuroscience diagnosis who were managed in a non-neuroscience ICU.[2] Over a year, this would amount to roughly 4,000 patients managed in a district general ICU rather than a specialist unit. Recommendations from the Society of British Neurosurgeons (SBNS) in 2003 were for 10 neurocritical care beds per million population served. In 2009, the SBNS also stated that 'admission to a regional neurosurgical service for life-saving emergency surgery should never be delayed' and that 'lack of critical care beds must not be a reason for refusing admission.'

So which patient with a subarachnoid haemorrhage (SAH)

is a neurosurgical emergency? All, none or some? Outcome is largely determined by age and grade of presentation. Although the elderly generally fair less well, age is no longer a contraindication for definitive treatment. The incidence of rebleeding after initial SAH is between 9-17% of patients, with most cases occurring within six hours of the initial bleed.[3] Mortality in patients who rebleed is up to 80%. Rebleeding is more common in patients in poor clinical condition, with large aneurysms, those with loss of consciousness or sentinel headache. Repair of a ruptured aneurysm by either coil embolisation or microsurgical clipping will significantly reduce the risk of rebleeding.[4] That argues for immediate transfer of all SAH patients for consideration of early angiography and securing of the aneurysm if identified. The resource implications are significant, not just at the neurosurgical centre but also at the DGH who would need to transfer the patient.

Should we transfer all patients or just those with good clinical grades? Some patients with initially poor clinical condition may benefit from emergency neurosurgery;[5] intracerebral extension of a SAH occurs in around 1/3 of cases; patients with large haematomas may require surgical evacuation preferably preceded by occlusion of the aneurysm, or hemicraniectomy to allow expansion of swollen brain tissue. Subdural haematomas occur in 2% of SAH and may require removal if causing mass effect.

Management of patients who arrive in a poor condition with enlargement of the ventricular system on initial CT is controversial. Does neurological impairment result from the initial haemorrhage or the hydrocephalus? Some neurosurgical centres will suggest the patient is observed for 24 hours locally. A proportion of patients will improve spontaneously with the initial episode of unconsciousness ascribed to the ictus. These patients should then be transferred for aneurysm management. Other units advocate early transfer and placement of an external ventricular drain with subsequent observation on the neurointensive care unit for signs of improvement.

NICE head injury guidance and recommendations from the NHS Clinical Advisory Group on regional networks for major trauma are clear that all severe head injuries should be managed in their regional neurosciences centre. The development of hyperacute stroke centres may result in similar recommendations for patients with SAH. In the meantime we are likely to continue with regional and individual variations in care. Neurosurgical centres must be clear about their indications for admission and reasons for refusal. Lack of capacity is no longer acceptable as a reason to delay admission and centres must continue to own patients who are initially admitted into DGHs, without suggesting transfer to other units. Critical care networks should compile records of delayed or refused admissions to regional units. The RAIN study will report in March 2012 on the outcomes and cost-effectiveness of care for traumatic brain injury in neurosciences centres versus DGHs.[6] Perhaps the next study should do the same for SAH?

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Competence of trainee doctors responsible for high dependency patients

We report the results of an audit carried out in the North-West region of England concerning the management of high dependency patients. According to the Department of Health, in January 2011 there were 1,672 level 2 beds available in England. Of these, 501 were in combined general intensive care/high dependency units and 521 were in separate non-specialist high dependency units. The others were mainly in specialist units such as cardiothoracic and liver units.

The experience of the authors is that in the North-Western Deanery region, general high dependency patients in separate units are either managed by the parent team with little critical care team input, or *vice versa*. We have been involved in several incidents where trainees from parent teams have sought help from the critical care team for management of high dependency patients, as the trainees felt they were required to act outside their competence. Therefore, we undertook a survey to assess the competence of non-critical care trainees managing high dependency patients.

The first part of the survey was telephone-based, involving all 20 high dependency units in the region. We found that in 45% (9/20) of high dependency units, patients were managed by the critical care team, who were actively involved in stabilising patients. In the remaining 11 units, parent teams managed patients. For those 11 units we asked to speak to a doctor responsible for looking after patients on the unit. We found that nine (82%) of those doctors did not feel competent to insert arterial lines, seven (64%) did not feel competent to insert central lines and four (36%) did not feel competent to manage vasoactive infusions of drugs.

The preliminary survey was followed with an internet-based

region-wide survey asking similar questions. There were 43 complete responses, with the following representation across specialties: surgery 25 (59%), medicine 9 (20%), obstetrics and gynaecology 5 (13%), orthopaedics 3 (7%) and others 1 (2%). Twenty-two (52%) of respondents said that the parent team had overall responsibility for high dependency patients where they work. Despite this, 35 (81%) replied that the critical care team was responsible for the placement of invasive lines. Twenty-one (49%) respondents felt competent with arterial lines and 44% had received training. Only 11 (26%) felt competent with central lines and 18 (42%) had received training. Only nine (21%) felt competent to manage vasoactive infusions. Worryingly, only nine (21%) felt competent overall to manage high dependency unit patients.

Although there are limitations to our survey, it is clear that in approximately half of the units surveyed, the critical care team did not manage high dependency patients. This raises significant clinical governance issues with regards to the management of these patients by parent teams. We suggest that further work to elucidate the national picture should take place. If the regional picture is replicated, the solution must be either education of parent team trainees or the provision of extra resources to critical care teams to care for all high dependency patients.

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Is ventilator-associated pneumonia (VAP) an appropriate quality indicator?

'Quality means doing it right when no one else is looking' Henry Ford.

It has been estimated that up to 10% of hospital patients are affected by healthcare-associated infections (HAI), and as many as a third of cases are preventable. Ventilator-associated pneumonia (VAP) is the most common HAI in intensive care units (ICUs) with a reported incidence between 9 and 28%.[1,2] Studies have demonstrated that VAP increases morbidity, number of ventilator days and length of both ICU and hospital stay.[3] Moreover, it constitutes a significant economic burden for every patient. Each VAP infection is estimated to increase patient care cost by £6,000-£22,000.[4]

Considering the significant clinical and financial implications, it is unsurprising that NHS targets have increasingly focused on outcome measures related to hospital-acquired infection. Failure of NHS organisations to effectively audit policies and to implement procedures to prevent infection can now result in penalties, suspension and even closure.[5] In the United States, insurance companies regard this apparent failing in patient care as a breach of contract. On this basis, the Centers for Medicare and Medicaid Services have stopped reimbursing hospitals when patients suffer nosocomial infections such as vascular catheter-related bloodstream infections and catheter-associated urinary tract infections. The proposal that VAP is to become the next 'preventable complication' has been raised but so far has been met with disapproval by clinicians.[6] Whether this objection will be sustained is not yet clear. If VAP does ultimately become a marker of failing standards or poor performance, we will no doubt see these same arguments transferred to the UK.

Until an acceptable 'gold standard' is agreed for diagnosing VAP, comparison between units to determine quality and tariff on the basis of VAP incidence cannot be justified. Diagnosing VAP correctly and reliably is challenging, as current definitions lack the necessary diagnostic discrimination. NICE guidance of 2008 refers to VAP as a pneumonia in a 'mechanically ventilated patient that develops 48 hours or more after intubation with an endotracheal or tracheostomy tube that was not present before intubation.'[7] However, many conditions common in ICU patients, such as ARDS, sepsis, cardiac failure and lung atelectasis have similar clinical signs. As a consequence, more than 50% of patients diagnosed with VAP do not have the disease, whereas up to one third of patients with VAP are not actually diagnosed.[8,9] Scoring systems provide a method of pooling data to determine the likelihood of a positive or negative diagnosis. The Clinical Pulmonary Infection Score (CPIS),[10] Centre for Diseases Control and Prevention National Healthcare Safety Network (CDC-NHSN) Definition[11] and the Hospitals in Europe Link for Infection Control through Surveillance (HELICS) criteria[12] are recognised systems. The CPIS, which incorporates only qualitative microbiological analysis, is the most commonly used scoring system in the UK (Wong A, Mathieu S, Williams M; unpublished data). HELICS is widely used in Europe and relies on a combination of clinical, radiological and microbiological criteria. Unfortunately, these scoring systems and definitions lack the necessary sensitivity and specificity required of a 'gold-standard' diagnostic tool. In addition, the subjectivity of individual domains (clinical signs, radiology, microbiology sampling) has meant a considerable variation in interpretation by clinicians and therefore similar discrepancy in the reported incidence rates of VAP.

Even given a reliable, robust and accurate system for recognising VAP, at what point in the continuum between lower respiratory tract colonisation and infection with pathogenic microorganisms does VAP become a distinct entity? Is it truly preventable with current measures? Do the surrogate

markers of quality care for prevention of VAP reliably reflect an improvement in outcome? While there is consensus that VAP has a deleterious effect on morbidity, ventilator days and length of hospitalisation, there is discordance about the magnitude of its effect on mortality.[3,13]

If the difficulties in the prevention and diagnosis of VAP are compared with that of catheter-related blood stream infections, it is perhaps understandable to see the significant contrast in the adoption of these processes to monitor and improve rates in the UK. 'Matching Michigan' provided a simple, standardised means for identifying nosocomial infections. Moreover, by adopting evidence-based methods and empowering a culture change, benefits could be readily recorded and tracked. Success and failure were transparent and the efficacy of local and national changes in practice could be determined. Over 95% of NHS Trusts have been involved with the NPSA's (National Patient Safety Agency) Matching Michigan campaign and quantifying the rates of catheter-related blood stream infection (CRBSI). This is not the case with VAP. We conducted a national survey of all adult general ICUs in the UK and demonstrated that two-thirds of respondents were not collecting VAP data and did not feel that it was an appropriate quality indicator. Although endorsed by the Intensive Care Society (ICS) and publicised at a national meeting, there was a poor response rate of 39% to this survey (Wong A, Mathieu S, Williams M, unpublished data). The reasons for the low response rate are multi-factorial, but the inadequacies of a consensus definition, reliable scoring systems, and consistent evidence of an outcome benefit will almost certainly have contributed. Having initiated the survey to try to determine UK practice with regard to VAP diagnosis, it now seems clear that lack of confidence in any of the currently available methods for diagnosis has resulted in disengagement with its completion.

The information gathered from the survey has resulted in our reflection about VAP assessment and on the processes we used on our ICU. Like many units, we implemented the Department of Health's High Impact Interventions (HII), including number 5 (care bundle for ventilated patients to prevent VAP).[14] Having been repeatedly requested to produce audit data of compliance against care bundles, we considered it more pertinent to see whether this translated into a reduction in the incidence of VAPs, with consequent patient safety and outcome benefits. Even though the diagnosis of VAP remains controversial, it is logical that all ICUs should consider implementing processes to identify outcomes (including VAP, by whatever diagnostic criteria agreed locally), so that evaluation of 'improvement measures,' within care bundles, can be made.

At the Queen Alexandra Hospital, VAP rates have been tracked continuously since October 2009 using CPIS and semi-quantitative non-bronchoscopic lavage (NBL) microbiology sampling.[15] Suspected cases are discussed at a quarterly multidisciplinary meeting involving senior critical care and microbiology representatives, ensuring that the rates of VAP are monitored and that the efficacy of changes in practice are determined. This process was initially started because of a request by Primary Care Trust (PCT) commissioners to provide data and has been continued. Auditing compliance with the HII ventilator care bundle[14] and VAP rates are the only indicators we use to minimise nosocomial pneumonia. The audited VAP rates have consistently compared favourably to published data,[1,2] although this inference should be viewed with caution given the diagnostic controversies.

What if VAP rates were unacceptably high within a unit, or if other units using a different system had a lower incidence? There is a danger that alternative systems are used simply to manipulate data. Alternatively, some centres may make a decision to disengage entirely from the process so the concept of nosocomial pneumonia is driven 'underground.' Quality indicators are becoming increasingly important in commissioning healthcare provision. Trusts are under increasing pressure to demonstrate compliance with a rising number of measures to maintain Care Quality Commission (CQC) targets. Realistically though, in order for performance to be based on outcomes, there has to be a consistency of diagnostic criteria in order for comparison of measurements 'like with like.' Currently this is not practical without a consensus about which system (if any) is appropriate to use for diagnosing VAP.

The Commissioning for Quality and Innovation (CQUIN) payment framework, introduced in April 2009, enables commissioners to withhold a proportion (up to 1.5%) of Trust income for failing to drive improvement against outcomes. With around 400 existing quality indicators, it may only be a matter of time before VAP re-appears as one of them. If this is the case, it is hoped that a robust, accurate and clinician-approved diagnostic tool will be available and that its resultant implementation will translate into improved patient outcome. The incentive for its mandatory introduction should not be to enforce unrealistic targets in order to penalise Trusts.

In conclusion, measuring quality in critical care is fraught with hazards. There is clearly unease among the critical care fraternity about using VAP rates as a quality indicator. In the absence of a confirmed and reliable consensus diagnosis, any related requirement by commissioners should therefore be strongly resisted. Despite this, it would seem reasonable to recommend that local processes be developed to identify VAPs as accurately as possible. It is essential to ensure that measures to reduce this HAI are evaluated and add to the culture of maintaining infection prevention measures.

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TRALI or TACO? A diagnostic dilemma

We wish to report a case of refractory hypoxia after massive transfusion.

A 64-year-old female patient was resuscitated with fluids and blood products following a massive haematemesis. With ongoing resuscitation, she was taken for laparotomy to control bleeding. She was transfused a total of 13 units of blood, 12 units of fresh frozen plasma and 1 unit of cryoprecipitate in a span of five hours. Two hours into the surgery, the patient developed severe hypoxia manifested by a decrease in the SpO_2 to around 75%. Arterial blood gas (ABG) analysis showed pO_2 5.1 kPa, pCO_2 14.5 kPa, pH 7.03, HCO^{3-} 28.8 mmol/L. The patient was being ventilated on a GE Datex Ohmeda Aestiva® 5 ventilator with pressure control ventilation and a positive end-expiratory pressure (PEEP) of 10 cm H_20. Various manoeuvres to resolve the hypoxaemia (eg 100% FiO_2, bronchodilators, recruitment manoeuvres) proved unsuccessful. On-table chest X-ray showed bilateral pulmonary infiltrates. Based on the possibility of transfusion-related acute lung injury (TRALI) or transfusion-associated circulatory overload (TACO), a dose of frusemide 40 mg was given. The patient's oxygen saturations were at or below 75% for nearly an hour, but the patient was stable haemodynamically. The patient was connected to a Dräger Oxylog® 3000 portable ventilator using the BIPAP/ASB setting with PEEP of 15 cm H_20. Within minutes, the patient's oxygen saturation improved dramatically and maintained above 92% thereafter. A further ABG with FiO_2 1.0 showed pO_2 15.9 kPa, pCO_2 8.8 kPa, pH 7.27, HCO^{3-} 30.3 mmol/L. Surgery continued; the patient had a palliative procedure when primary anastomosis proved impossible.

Differential diagnoses of TRALI include TACO, anaphylactic transfusion reaction and transfusion of contaminated blood products.[1] However, laboratory tests for incompatibility of donor units ruled out the possibility of TRALI and favoured a diagnosis of TACO. Positive fluid balance, high volume of transfusion, faster rates of transfusion, large volumes of FFP transfusion, age less than three years or greater than 60 years particularly with baseline cardiovascular dysfunction, are the risk factors for the development of TACO. There is no single diagnostic test that reliably discriminates TRALI from TACO although the clinical features of hypertension, neck vein distension, pulmonary artery occlusion pressure greater than 18 mm Hg, transudate pulmonary oedema fluid, significant response to diuretic and brain natriuretic peptide >1,200 pg/L may favour a diagnosis of TACO as opposed to TRALI.[2,3] Although it is difficult to pinpoint whether the resolution of hypoxia in our case was due to the use of a different ventilatory strategy with a high-powered ventilator, or a significant but slower response to frusemide, the case highlights the diagnostic difficulties faced in such situations and the need to consider TRALI and TACO early in the differential diagnosis of hypoxia, especially after massive transfusion..

Acknowledgement

Written consent was obtained from next of kin.

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Comment:

Transfusion-related acute lung injury (TRALI), is defined as acute dyspnoea with hypoxia and new or worsening pulmonary infiltrates arsing during or within six hours of transfusion of plasma, cellular blood components or immunoglobulins.[1]

The risks of blood transfusion are either intrinsic to the stored blood, such as infection, or related to an immunological reaction, whether it be from ABO or other sources of incompatibility. TRALI is suggested to be an immunological response to blood products and is defined largely by its pattern of presentation and at present confirmed by immunological testing. The situations where it presents are often similar to those where transfusion-associated circulatory overload (TACO) may occur and hence this is the main differential diagnosis. TACO is made a more diverse condition by virtue of the fact that fluid overload may be more likely with cardiac dysfunction or where there may be a predisposition to increased pulmonary capillary permeability, such as is seen in sepsis. It is also by nature, a dynamic process that may be transient in causation but persistent in effect.

Traditionally, the focus of transfusion risks has been with the risk of infection but as that risk diminishes, the relative importance of side effects such as TRALI is rising.[2,3] FFP and platelets carry a far higher risk than red blood cells (by a factor of 6); FFP is associated with about half of all cases and red cells about a third.[1] The time limit for the 'arbitrary' diagnosis of TRALI (**Table 1**) is six hours from transfusion. About 70% of cases were admitted to ICU and half of these were ventilated. There is a quoted mortality of about 20% in those meeting TRALI criteria.[4] TACO, which is a relatively common phenomenon, is rarely reported and curiously seems to have a very limited place in any review of this subject.

TRALI is considered to be an immunologically-mediated reaction and hence constitutes a form of incompatibility between donor and recipient.[5] In most cases of TRALI, HLA (Human Leukocyte Antigen) or neutrophil-specific antibodies are present in the donor plasma and a corresponding antigen is found in the recipient. It is proposed that this antibody-antigen reaction triggers an inflammatory response. HLA antibodies can cause TRALI, but not all cases have white cell antibodies in the donor. This is antibody-negative TRALI and the incidence may be between 11 and 39%.[1,6,7] This may relate to the sensitivity and range of testing, which is improving.[8,9] The second possible mechanism is that the patient has a problem that results in neutrophil migration to the lungs. The storage of blood releases cell components, lipids and lipoproteins, which can promote neutrophil activation as a 'priming agent.' It is these lipid components that, when infused, interact with the lung neutrophils and produce a reaction. This is the 'two hit' mechanism for TRALI.[10-12]

The situation is made more complex by the prevalence of HLA antibodies in blood products. In an assessment of cryoprecipitate, fresh frozen plasma, red cells and apheresis platelets, 22% contained HLA alloantibodies. A total of 18-40% of multiparous females have either granulocyte or HLA antibodies and about 1.7% of transfused males, but also a very small percentage of untransfused males.[13] As the prevalence of cognate antigen in the recipient is also high, the potential for interaction is far higher than any reported rate and so the presence of both antibody in the donor and cognate antigen in the recipient does not necessarily predict a reaction.[14] Nevertheless selective use of male untransfused products should decrease the risk and does appear to have reduced the incidence, as has leucodepletion.[8,5,16] Differentiating TRALI from TACO in many circumstances is likely to remain a diagnostic conundrum.

Probability	Description
Highly likely	No other cause identified for the symptoms and positive serology
Probable	Either positive serology as defined below but with other causes for symptoms also present OR no other causes present, but with either absent or incomplete serology
Possible	Clinical picture compatible with TRALI, no other cause present, but results of patient and donor investigation negative as defined below
Unlikely	Another cause of symptoms present AND results of patient and donor investigation negative

Table 1 Working diagnosis of TRALI.

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Ultrasound Training and the Faculty of Intensive Care Medicine

In the late 1990s, the Association of Cardiothoracic Anaesthetists (ACTA) took the initiative to establish a standard in transoesophageal echocardiography (TOE) for cardiac surgery. Training centres were established and in 2003, having been joined by the British Society of Echocardiography (BSE), an accreditation process started which has been running ever since.[1] Many of those involved worked on intensive care units where we could see the need not only for echocardiography but ultrasound skills in other areas such as insertion of chest drains, intravascular catheters, etc. As such, several lectures at meetings organised by the Intensive Care Society (ICS) gave insight as to the usefulness of ultrasound on intensive care. A recurring question was, 'when will general intensivists have a training standard in ultrasound/echocardiography?' The ICS had no particular enthusiasm to take this on and the Intercollegiate Board of Training in Intensive Care Medicine did not see this as a necessary competency. So the answer I gave at the time was that when the Faculty of Intensive Care Medicine (FICM) arrived, these aspirations would be fulfilled and proper accreditation developed. A year ago at the ICS annual meeting, a group with members from the BSE, ACTA and the ICS met and put together proposals from a variety of sources. What is needed now is the authority given by the FICM.

Thus I was delighted to read Professor Bion's editorial in the October edition of *JICS* stating that they, 'propose ICM as a cross-cutting theme, with standards covering infection prevention, care processes and outcomes.' Further that, 'we have established working groups with all partner specialties to determine the extent of shared competencies between ICM and other programmes.'[2] I hope that this means that echocardiography/ultrasound will become part of ICM training.

The BSE responded and offer two levels of intensive care echo training. One is a 'high' level accreditation similar to the existing transthoracic echo process with some TOE added on. Though laudable and desirable for a few who want to practise echocardiography at a high level, this is not possible or necessary for the majority of ICU doctors. At the other end, the BSE run 'FEEL' (Focused Echocardiography in Emergency Life support) courses, which offer one day of training for echo in the peri-arrest situation. This is not actually enough for many of the management decisions faced on ICU. Walker called on the ICS to fill the gap, but I think it is clearly the role of the FICM.[3]

What is required is an intermediate level of training that all doctors training in ICM must achieve, and a process for training that is accessible to existing practitioners. The UK has been at the forefront of echo training for many specialties but not in ICM.[4] Now is the time to catch up. Already in the country there are many courses with detailed curricula aimed at the 'ICU doctor.'[5] But without authority, they can only offer a certificate of completion. It would not be a huge leap for such courses to tie in with an accreditation process. Having taught on many of these courses for several years, I know that there is real enthusiasm among intensivists to learn these skills to help with patient management on the ICU.

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Response:

We are pleased to respond positively on behalf of the Faculty of Intensive Care Medicine to the letter from Dr Bennett about critical care ultrasound. There can be little doubt that ultrasound imaging will become part of the standard diagnostic armamentarium for intensive care specialists. As with any new technology, the process has started in specialist areas led by enthusiasts, but in time is likely to become a universal skill. The questions we need to answer are: at what level, whether mandatory or optional, and to what effect?

Dr Bennett describes three levels of competence: emergency 'quick scan' ultrasound; intermediate level diagnostic imaging; and advanced imaging including transoesophageal

echocardiography. This seems eminently sensible. However, the FICM cannot mandate training at any of these levels without considering the specific competencies, the time taken to acquire them, availability of training and equipment, and whether there is access to funding in order to attend courses. Our current position is that the existing excellent work done by ACTA, the BSE and the ICS provide an ideal basis on which to build optional special skills modules during Stage 2 of the new ICM training programme (depending on the trainees' ICM programmes). From this position we will build a cadre of trainers and approved courses which will allow us to incorporate ultrasound skills formally within the ICM programme. A parallel process of systematically evaluating the utility of the training would be helpful, in terms of patient outcomes.

In the new year we will bring together the various groups involved in developing ultrasound skills under the remit of the FICM Training Committee. We will also ensure representation from other professional groups with an interest in this area, not least our radiology colleagues. Our plan is to review the current proposals from the various interest groups involved, and to develop a harmonised position which can then be integrated with the new ICM training programme as optional modules. We will also need to ensure independence of the accreditation process from those providing the courses.

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