Editorials

Resuscitation fluid controversies – Australian trials offer new insights

Two substantial Australian multi-centre clinical trials[1,2] have recently provided answers to controversial questions about optimal fluid resuscitation strategies. What were the messages for critical care clinicians?

Hypertonic saline (HTS) had been proposed as a better fluid for pre-hospital resuscitation of trauma patients - especially those with traumatic brain injury (TBI).[3] Potential advantages included rapid increase in intravascular volume, relatively small intravenous fluid volumes, and decreased intracranial pressure.[4,5] Sub-groups and meta-analysis of previous trials reported increased survival in TBI patients receiving HTS and HTS-dextran,[6,7] and commercial products including HTS were approved and marketed in many countries.[3,8]

The HTS study[1] was a double blind randomised trial including all 11 Victorian hospitals receiving trauma patients. Over 3 years, 229 patients with blunt injury, traumatic coma and hypotension were randomised to receive 250 mL 7.5% HTS or 250 mL Hartmann's solution in addition to routine paramedic protocols. In both study groups hypotension was corrected on arrival to hospital, and in both groups survival was better than predicted by the usual scoring systems, and also better than patients in the meta-analysis. HTS tended to decrease the first measured ICP (P = 0.08) but did not improve long term neurological function measured 6 months after injury using the extended Glasgow Outcomes Score. The proportion of patients with favourable neurological outcomes was also the same in both groups.

Therefore, despite theoretical advantages of HTS resuscitation in patients with TBI, in a well developed paramedic based trauma system, pre-hospital HTS was not beneficial and should not replace current isotonic crystalloids. However, in many Australian intensive care units, HTS is instead used as a preferred alternative to mannitol in patients with increased ICP. The Victorian trial did not address this question and indeed many of the study patients would also have received HTS in ICU as one component of therapy for intracranial hypertension. Accordingly, it would not seem appropriate for ICU clinicians to change practice concerning HTS osmotherapy on the basis of this trial. A well-constructed future trial of HTS for intracranial hypertension in ICU may be appropriate.

The truly remarkable saline versus albumin fluid evaluation (SAFE) study[2] compared the effect of 4% albumin and 0.9% saline resuscitation on 28-day mortality in nearly 7000 ICU patients. Patients admitted after cardiac surgery, liver transplant and burns were excluded. Mortality after both types of resuscitation was the same (RR 0.99; 95% CI 0.91 - 1.09). Clinician's interpretation of this finding will undoubtedly be influenced by previous convictions. Those who favour theoretical advantages of colloids including albumin will likely be reassured that reports of increased mortality with albumin were not supported. Furthermore, their future choices of colloid type may shift towards albumin, being the only colloid tested in a large definitive trial and found to be safe. In contrast, crystalloid users may likely see no reason to change current practice, and also may be reassured that they use and teach a cheaper and equally effective product. It will be intriguing to see whether the recent international trend to decreased albumin usage after the Cochrane meta-analysis[9,10] is reversed by the new data, or continued due to the cost considerations.

In SAFE, there were also important findings in pre-planned subgroups, and interestingly these were in opposite directions. In trauma patients, there was a strong trend toward increased mortality in patients treated with albumin (RR 1.36; 95%CI 0.99 - 1.86). This finding was in keeping with a meta-analysis, which found that trauma patients receiving colloids had a higher mortality than those receiving crystalloids.[11] In trauma patients in SAFE, the increased mortality was almost entirely found in patients having trauma associated with brain injury (RR 1.62; 95%CI 1.12 - 2.34). Critical care clinicians are now likely to choose crystalloids for resuscitating future trauma patients and especially patients with associated brain injury, until more definitive data are reported. In the meanwhile, detailed re-examination of the SAFE-TBI patients is urgently required and is ongoing. A future randomised trial of albumin versus saline in TBI patients is inviting, but may be unethical if more detailed examination of the SAFE patients supports the present finding as real. On the other hand, in sepsis patients, the trend to increased survival in albumin treated patients (RR 0.87; 95% CI 0.74 - 1.02) is exciting but also requires confirmation, and invites a future randomised trial. This trial would be large, would have similar logistic and funding complexities to the SAFE study, and may be well supported by industry.

It is intriguing to consider that the much-maligned Cochrane albumin meta-analysis[9] may have been partly correct. We know that the quality of albumin production has improved with time, and that current Australian and

New Zealsnd 4% albumin (Albumex® 4; CSL Ltd) is an industry leader and associated with a very low frequency of adverse reactions. The risk-benefit ratio of albumin resuscitation may have shifted over time with the Cochrane paper reporting studies involving much older albumin preparations, and SAFE reporting a study involving the modern Australian and New Zealand product.

Finally, it is critical to recognise that the remarkable success of these two trials was due to the collaboration and hard work of many individuals and groups. In this environment, the future for collaborative multi-centre research in Australian and New Zealand critical care is looking particularly bright.

The views expressed in this editorial are those of the author alone and do not represent the views of the HTS study or SAFE study investigators.

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"…the occasion fleeting; experience fallacious, and judgement difficult."

A patient with chronic heart failure who has a sudden hypotensive or hypoxic episode may develop an acute reversible elevation of plasma alanine amino transferase (ALT), aspartate amino transferase (AST) and lactate dehydrogenase (LDH).[1,2] The disorder is known as 'shock liver' or ischaemic hepatitis and is caused by hepatocellular hypoxia due to an elevated systemic venous pressure (causing hepatic venous congestion) and low cardiac output (causing a reduction in hepatic arterial blood flow).[3] The disturbance only occurs if both hepatic venous congestion and low cardiac output exist, as severe systemic hypotension alone will not lead to ischaemic hepatitis.[4] While the disorder is usually diagnosed from clinical and biochemical features (in the absence of other causes),[5,6] the *sine qua non* of ischaemic hepatitis is centrilobular necrosis in the absence of inflammation.[7-9]

The diagnosis is usually suspected in a patient with chronic heart failure who has a sudden reduction in systemic blood flow or increase in right heart failure due to an acute myocardial infarct, pulmonary embolism, pneumonia or arrhythmia,[1,2] although it has also been reported in cirrhotic patients following haemorrhagic shock[10,11] and has been described in patients with sleep apnoea.[12,13]

The disease is usually limited to a mild elevation of plasma bilirubin, prothrombin time and alkaline phosphatase, and a characteristically greater increase in plasma LDH compared with plasma ALT or AST.[8,14,15] One study found that the mean serum ALT:LDH ratio in patients with ischaemic hepatitis was 0.87, compared with 1.46 in paracetamol hepatitis and 4.65 in viral hepatitis.[14] They concluded that a serum ALT:LDH ratio of < 1.5 differentiated ischaemic hepatitis from paracetamol hepatitis and viral hepatitis, with a sensitivity of 94% and a specificity of 84%.[14] With the correction of cardiac failure, the enzyme changes

usually resolve rapidly (i.e. > 50% decrease within 72 hr).[1,7]

In this issue of the journal, Sommerville, *et al*,[16] describe a case of acute hepatic failure caused by cardiac tamponade complicating a proximal (type A) aortic dissection. The patient presented to the hospital emergency department, hypotensive and complaining of right upper quadrant tenderness. The plasma biochemistry and arterial blood analyses revealed lactic acidosis and elevated plasma hepatic enzymes. The diagnosis of an intra abdominal 'vascular event' with sepsis was made, although the hypotension appeared to be unusually resistant to intravascular fluid. A laparotomy revealed torrential haemorrhage from the surface of the liver. Both the hepatic artery and portal vein were patient.

Subsequently, the diagnosis of an acute proximal dissecting aortic aneurysm with cardiac tamponade was made, albeit too late to be of benefit to the patient, leaving one to wonder as to the presentation and pathophysiology of this case.

While a retrospective analysis of any puzzling case, once the diagnosis has been made, can always boast 20:20 vision, it can sometimes be instructive. One could speculate that the combination of hepatic venous congestion caused by the cardiac tamponade (which may have been exacerbated by the intravenous fluids) combined with the tamponade induced hypotension, could have caused an acute ischaemic hepatitis (the ALT:LDH ratio was never greater than 0.62). The ischaemic hepatitis could also have been, at least in part, responsible for the acute lactic acidosis due to a diminished hepatic metabolism of the lactate generated by shock-induced anaerobic metabolism. A raised serum lactate with signs suggestive of an acute abdomen due to ischaemic hepatitis has been previously described, with the authors highlighting the presence of markedly deranged transaminases allowing the clinician to differentiate this disorder from intestinal ischaemia.[17]

However, the torrential haemorrhage from the surface of the liver would have required not only a coagulopathy (which in this case must have been multifactorial) and severe hepatic congestion, but hepatic capsular damage as well. Could there have been inadvertent damage to the liver surface during the laparotomy?

Sommerville, *et al*, identified three previously reported cases of acute aortic dissection that presented with acute hepatic dysfunction as the predominant feature. All cases exhibited dissection to the bifurcation of the aorta and loss of hepatic artery and portal venous flow due to obstruction of both the celiac and mesenteric arteries.[18-20] In contrast, in the case reported by Sommerville, *et al*, the acute hepatic dysfunction was due predominantly to cardiac tamponade causing hypotension and hepatic venous congestion.

Are there any lessons? In hindsight, hypotension non responsive to intravenous fluids usually requires an early comprehensive assessment of cardiovascular function (e.g. right heart catheter, echocardiography), lactic acidosis in the presence of elevated plasma liver enzymes is more likely to be caused by an hepatic, rather than gut ischaemic, problem and no matter how long one has been practicing as an Intensivist, a typical patient in shock will often be admitted to the ICU who is just not 'typical'.

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Advanced haemodynamic monitoring: getting to the heart of it

During the last twenty years there has been growing, healthy debate questioning the use, and abuse, of the pulmonary artery catheter (PAC) in critically ill patients. For example, in 1987 Gore and co-workers reported a prospective observational study of patients with myocardial infarction, and found an increase in mortality in PAC patients.[1] However, both groups of patients had a similar and high mortality rate from cardiogenic shock. This was followed by large observational studies of critically ill patients that confirmed higher risk for PAC patients,[2,3] despite attempting to control for selection bias. More recent data from prospective, randomised studies of high-risk surgical patients (n = 1994),[4] and patients with shock or ARDS (n = 676)[5] have found no effect on mortality, duration of ventilation and organ failure, although there was an increase in the rate of pulmonary embolism in PAC patients.[4] While these latter two studies are not as large, their prospective, randomised design gives them more authority than earlier data.

Importantly, we need to return to the questions usually asked when a new diagnostic tool is introduced – how will this change management and lead to a better outcome? Well designed studies such as the ARDSnet Pulmonary Artery Catheter (PAC) study[6] which also compares different fluid management strategies will help address this issue.

In this issue of the Journal King and Lim review the oesophageal Doppler monitor,[7] and Ong and colleagues report difficulties with hypothermic calibration of cardiac output determined using the PiCCO® device.[8] It is clear that controversy regarding the PAC has not markedly dampened enthusiasm for advanced haemodynamic monitoring techniques. Indeed, it has often been used as the reason for looking at ways of monitoring cardiovascular function without the risk of complications directly associated with the PAC. While this particular issue has not been directly examined, Sandham and colleagues[4] found a 1.5% vs 0.5% rate of adverse effects such as arterial puncture, pneumothorax and pulmonary haemorrhage, and Richard and coworkers[5] reported a 7.2% incidence in PAC patients. Some of these complications are common to any form of central venous access, but insertion of a PAC is often performed as an additional procedure. Consequently, when choosing an advanced haemodynamic monitoring technique this must be taken into account.

The oesophageal Doppler monitor uses the familiar Doppler shift to yield blood flow velocity. Stroke volume is calculated from the quotient of stroke distance and aortic radius; with the latter also measured with some devices. As flow is measured in the descending aorta a further assumption regarding the proportion of flow to the lower body is often made (typically 70% of total flow). In addition to systemic flow, the corrected flow time and the peak aortic blood flow acceleration can be used as indices of left ventricular preload and contractility respectively. Despite good correlations with thermodilution cardiac output, when subject to the more demanding Bland Altman analysis, the oesophageal Doppler monitor has wide limits of agreement (1.8 L/min).[9] In other words it is probably a good trend monitor, but the absolute measure of flow may be inaccurate. In addition, the non-invasive label is contentious as this monitor is best tolerated in anaesthetised subjects. Nevertheless, this scepticism may also reflect my lack of experience with the technique.

Techniques such as the PiCCO® device may be more familiar. They use transpulmonary thermodilution using a central venous injection of cold injectate and a fibre-optic thermistor, commonly positioned as a femoral or brachial arterial line, to calibrate pulse contour analysis. This allows an accurate and continuous measure of cardiac output. In addition, extravas-

cular lung water and intrathoracic blood volume, a preload index, are measured. Potentially, this is of greater use than the pulmonary artery occlusion pressure, which correlates poorly with preload in the critically ill patient, and the measures are made without some of the risk associated with the PAC. However, the trade off may be vascular compromise as larger catheters are placed in more proximal arteries. In addition, hypoxaemia and extravascular lung water are complexly related, and the lack of measurement of the pulmonary artery pressure itself may be an important impediment in patients with significant pulmonary hypertension complicated by right ventricular failure.

In our critical care unit we tend to use the PAC if complex haemodynamic monitoring is needed, but we are gaining experience with the transpulmonary technique. In a particular patient one method may have advantages over the other, in others echocardiography may be the investigation of choice. However, none of these techniques can replace a careful history and examination, followed by repeated observation and intervention. Indeed, it's worth reflecting on resuscitation goals the next time you are first on the scene. As only the sickest patients appear to obtain benefit from advanced haemodynamic monitoring,[10] make sure there is an important question and potential therapeutic strategy before proceeding.

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Is there an ideal insulin adjustment protocol for the critically ill patient?

The article by Orford and co-workers[1] in this edition of the Journal describes one practical approach to the maintenance of normoglycaemia in critically ill patients. Interest in this topic has been accelerated by the seminal work of van den Berghe and co-workers.[2] Until the van den Berghe publication, the control of blood glucose levels (BGL) in critically ill patients was mostly carried out on an intuitive level, in the belief that normalising BGL, like many other variables in the ICU, was "a good thing". Furthermore, good BGL control avoided such troublesome complications as osmotic diuresis. The intensive care community was now presented with the added incentive of improved outcome in patients with tight BGL control.

Achieving improved outcomes by better BGL control, such as reduced mortality, reduced infection rates and less organ failure[2] is very satisfying. However, the thinking practitioner will require answers to at least two important questions. What are the risks of tight BGL control and why is high BGL harmful? The main risk, of course, is an increased rate of hypoglycaemia, with possible severe consequences for the brain, with its absolute requirement for glucose as an energy substrate. In the study by Orford *et* al,[1] the rate of hypoglycaemia was only 0.8% (47 episodes of BGL less than 3 mmol/L out of 5603 BGL measurements), with no untoward consequences. Some might argue that the protocol described by the authors required to achieve normoglycaemia and avoid hypoglycaemia is complex and therefore time-consuming.

The answer as to why high BGL is harmful is much more complex. Indeed, mild hyperglycaemia may be beneficial in some instances, as witnessed by the beneficial effects of glucose-insulin-potassium (GIK)

infusions on the heart, post acute myocardial infarction or post-reperfusion.[3] BGL's in this setting may be above the normal range. Much work has been done to elucidate the basis of the adverse consequences of chronic hyperglycaemia. Most of this work relates to the micro-vascular lesion associated with hyperglycaemia. This is postulated to be due to increased intracellular oxidative stress (due to depletion of NADPH and increased production of reactive oxygen species), production of pro-inflammatory mediators (via increased flux in the hexosamine pathway and activation of protein kinase C) and the enhanced production of advanced glycation end products. The micro-vascular injury impacts on many organ systems, including the kidneys, eyes, heart and lungs.

As an example of the complexity of the effects of hyperglycaemia, it has been shown that high BGL's can reduce the production of superoxide by neutrophils,[4] which may explain the known reduced microbial killing effect of neutrophils and macrophages in hyperglycaemic patients. So, at the cellular level, hyperglycaemia may result in blood vessel damage due to excess superoxide, while in the neutrophil the reverse holds (reduced microbial killing due to less superoxide). Hyperglycaemia may also contribute to adverse processes independent of the cellular effects, such as exacerbation of insulin resistance and reduced effectiveness of collectins in the alveolus.

These complex cellular effects, which could be described as part of the "signalling" function of glucose,[5] as opposed to its better know "substrate' function, all take place in the critically ill patient in the environment of stress (usually due to injury or infection). Stress may further complicate the management of hyperglycaemia due to insulin resistance. The "role" of insulin resistance teleologically is difficult to explain, but may be an attempt to shunt glucose substrate to compulsory pathways. An example of this is seen in ischaemic myocardium where lower levels of glucose substrate and higher levels of free fatty acids (due to stress-related lipolysis) exacerbate ischaemic damage, while higher BGL's may be protective. Insulin resistance may be dynamic and high levels of vigilance are required in the management of hyperglycaemia to avoid hypoglycaemic episodes.

Although there is now a firmer scientific basis for tighter BGL control in the ICU, there still needs to be a change in mindset to move BGL control up the list of management priorities. This has been the case recently for several of the more "mundane" therapies, such as enteral nutrition. Management in the ICU has correctly focused on the more "exciting" therapies, such as mechanical ventilation, inotropes and antibiotics. Clearly, attention to feeding and BGL control now need to occupy more of our consciousness. The problem is that good BGL control is time-consuming and labour intensive, requiring frequent BGL measurements and a fairly complex protocol as described by Orford *et al*.[1] One method of achieving normoglycaemia, and alleviating the work involved, is to automate BGL control. A prototype closed loop blood glucose control device has recently been described.[6] However, due to the complex environment in which the device operates and the lack of availability of accurate continuous blood glucose sensors, automatic control of BGL is not yet feasible clinically.

Until such time as automatic BGL control is possible, the use of protocols such as that described by Orford *et al,*[1] represent a viable means of BGL control. This is especially so now that we have good scientific incentive to do so, and even more so, are beginning to understand why a high BGL is harmful.

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MET: the medical emergency team or the medical education team?

Despite recent advances in cardiopulmonary resuscitation and health services management there still exists the problem of significant in hospital morbidity and

mortality secondary to adverse events. The following case report occurred in our institution.

A 47 year old, previously healthy, male underwent a semi-elective thoracotomy for an empyema. The surgical procedure and anaesthetic were uneventful. The patient returned to the ward at 1500 hours with a heart rate of 130 beats per minute (bpm), his observations were otherwise unremarkable. The surgical registrar was concerned about the heart rate and the patient's inability to urinate post operatively. She instructed the intern to insert a urinary catheter, if the patient failed to pass urine by 1800 hours. At 1800 hours the patient had not urinated, the heart rate was 140 bpm. Despite the intern's insistence the patient refused to have a urinary catheter inserted, the patient's condition was otherwise stable. The 'day' intern handed over the patient to the night medical officer at 2200.

The night medical officer was summoned urgently to see the patient at 2330 hours when the patient's blood pressure was 85/60 mmHg. The heart rate was now 150 bpm. The medical officer diagnosed hypovolaemia and administered 2 litres of Hartmann's solution, and ordered a blood transfusion. With this intervention, the blood pressure improved. The patient's vital signs were next recorded at 0230 hours when the blood pressure was observed to be 75/55 mmHg. The medical officer again responded promptly and commenced further intravenous fluids. Again there was a transient improvement in the patient's condition.

At 0400 hours the medical officer was concerned enough to telephone the on-call surgical registrar to explain the patient's condition. The surgical registrar was on-call but rostered off-site. The registrar stated that, he would come in at 0700 hours to review the patient prior to the commencement of his operating list. At 0530 hours the patient had a cardiac arrest. Despite the best efforts of the anaesthetic registrar and the ICU registrar, the patient could not be resuscitated and was declared deceased at 0600 hours. During the entire period of the patients post operative course there was no Medical Emergency Team Call or consultation with the treating surgeon or 'on call' intensive care specialist.

This death occurred in a hospital where the MET had been in operation for more than 4 years and where a fulltime nurse educator was employed to ensure optimal compliance and MET utilisation. The issues that this mans death raised were crystallised in the letter of complaint written to the state coroner by the family who asked:

1 *"Why didn't the resident doctor contact the surgeon who operated on ……, during the night if there were signs of distress, complications or a deterioration in his condition?"*
2 *"Why didn't the resident doctor contact the registrar? Was there a registrar on duty during the night?"*
3. *Why wasn't a medical emergency call put into place after 11.00pm when …… blood pressure fell and remained low? What criteria or symptoms presented by a patient, instigates the medical emergency process…."*

An expert witness appointed by the coroner concluded that: "Another important observation is that the patient fulfilled the criteria for activation of the MET for at least 14 hours. I understand that Dandenong Hospital had a MET at the time of the patient's death and that these MET criteria were widely advertised and known throughout the hospital. I also understand that these criteria were attached to the back of the hospital medical officer's ID card and thus easily available in case of doubt when faced with a sick patient. The timely activation of the hospital MET might have saved the patients life." However, the question remains: why didn't competent and experienced medical and nursing staff involved with this man's care call for expert help?

Some answers came from the staff debriefing. Firstly, because the patient was discharged from recovery with a heart rate of 130 bpm, the junior medical staff assumed that the patient was "okay" from both the consultant surgeon and anaesthetist's point of view. They assumed that if the operating team were unhappy with the patient's condition, the patient would have been transferred postoperatively to the ICU. Yet on review, both the surgeon and anaesthetist were unaware that the patient was discharged from recovery with a heart rate that mandated a MET call. Secondly, the patient looked "okay" despite his elevated heart rate. Indeed, he was sitting up and had a cup of tea whilst talking to his relatives early that evening. Thirdly, the nursing staff were reassured by the fact that the junior medical staff attended promptly to their concerns about the patient and he seemed to be managing the situation appropriately. Finally, while it was a busy night, with the benefit of hindsight everyone involved agreed that they should have put out a MET call.

This case raises questions about the standard of care in our hospitals, not only during the day but more importantly during the night. Can junior medical and nursing staff confidently recognise symptoms and signs of a potential critical illness? Are junior medical staff taught the skills of diagnosis and management of critical illness and are these competencies checked regularly? To what extent should junior staff be left to manage patients in emergency situations? Do hospital administrators have any idea what their staff skills and competencies are and what their workload is? How effective are junior staff in communicating with senior staff? Do we teach doctors to work in teams? Are we expecting too much from the MET's in a hospital with a progressively more complicated case-mix and a

workforce that is inadequately trained and who have little 'on the job' training and support for the required tasks?

The Quality in Australian Health Care Study documented 16.6% of hospital admissions were associated with an adverse event, and that of these 18.5% resulted in permanent disability and death.[1] Further analysis found that cognitive failure was a factor in 57%, and that 10.9% of adverse events comprised of a "failure to attend". Diagnostic and treatment delays were found to contribute to 56.8% and 40.6% respectively of the adverse events. Not surprisingly, events where 'delay' was a contributing factor, permanent disability or death was high (34.8% and 27.7% respectively). The authors concluded that if these delays had not occurred there would have been an 86.5% chance of preventing permanent disability or death, where there was diagnostic delay, and 90.1% chance of preventability, where there was a treatment delay.[2]

Unexpected in-hospital cardiac arrest is associated with a high mortality rate.[3-6] When a cardiac arrest occurs in a general ward area, many hospitals use a "cardiac arrest team" to provide immediate resuscitation. However, this approach has not been associated with an improvement in the mortality rate. Previous studies have suggested that 66 - 88% of in-hospital cardiac arrests are preceded by at least one abnormal clinical feature.[7-10] Traditionally, these observations are reported by nursing staff to junior medical staff, often leading to delays in evaluation and definitive care. There are many studies that also document inadequate experience, skills and knowledge of junior medical staff to manage these situations.[11-13]

To decrease the incidence of unexpected cardiac arrests, the concept of the medical emergency team (MET) has been suggested.[14] The MET consists of experienced clinician's who are paged to respond immediately to patients with predefined abnormal clinical observations and has subsequently been demonstrated to significantly decrease the incidence of cardiac arrests and unplanned intensive care admissions.[15,16] However, even in hospitals with highly responsive MET there is still a significant incidence of unexpected cardiac arrests. This may be due to the low specificity and sensitivity of the MET criteria,[17] reluctance amongst junior nursing and medical staff to breach the usual traditional hierarchal medical referral model of care[15] or simply failure of cognition as described above.[2]

There is little logic to indicate that the cardiac arrest team response to a terminal dying patient is an appropriate intervention and as such it would seem that the MET is a more appropriate response, if only to allow the timely institution of "not for resuscitation" orders, thus avoiding the inhumane and undignified CPR attempts that accompany some deaths. The MERIT study will undoubtedly shed light on this issue. Nevertheless, the expectation that MET can be the "magic bullet" that manages a hospital's acute critically ill patient population, in the absence of other interventions, does not address the real problem. Most hospitals have a hierarchal referral model of care that depends upon an individual's performance, which is often found wanting given the continual technological, economic and case- mix changes that modern hospital practice demands.

The above events and considerations represent "the paradigm" of medical care in modern hospitals. What remains unclear is whether this paradigm, which is essentially 19th century in conception, continues to serve patients well or whether different paradigms of care would achieve better outcomes. Is the "vertical" paradigm of care (e.g. junior nurse to senior nurse to charge nurse to intern to registrar to consultant and back with the possibility of countless miscommunications and misunderstandings) an appropriate approach for the 21st century? Do we need a more horizontal, problem-orientated approach? For example, a patient who has a stroke is managed by the stroke team, a patient who has a tracheostomy is managed by a tracheostomy team, a patient who has an epidural catheter is treated by the pain-management team and if you are acutely ill you are managed by the MET. Should we have "standards of care" (e.g. if you have a fever > 38.5ºC and a white cell count >12.0 x10^9/L should you have blood cultures and antibiotics within 1 hour)? Should we have pathway management nurses and doctors that co-ordinate team integration and activation? Is the patient "ownership" paradigm a paternalistic system that is difficult to audit and unable to deliver superior outcomes? Why do we have coronary care units for patients with an expected mortality of < 10% but do not have an equally well-equipped and staffed post-operative care unit for patients > 70 years who have a >15% mortality?[18]

In the opinion of the authors, the MET system is but "a mote in the eye" of the acute health care system, a minute peripheral challenge to the empire. Much more needs to be done to bring modern hospitals, care models and medical and nursing school training and the whole of the acute health care system kicking and screaming into the 21st century.

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