

# Pulmonary artery catheter versus new technology

In their article, 'Pulmonary artery catheterisation - a problem swept under the carpet?' Steele and Bihari make a strong case against the use of this procedure in intensive care practice.<sup>1</sup> They point out the hazards, the frequent lack of user knowledge and the paucity of evidence of patients benefiting from therapies guided by the information obtained. A basic principle arising from all of this is that if non-invasive or minimally invasive technologies exist which can provide the same or equivalent accurate information as invasive procedures, then the latter should be abandoned. Why then, are pulmonary artery catheters (PAC) still commonly used, especially in the setting of monitoring haemodynamics in the critically ill patient? There are undoubtedly many reasons and issues involved. I will mention a few.

1. As pointed out by Steele and Bihari, the PAC has been around for more than 30 years and the Swan-Ganz catheter (SGC) since the '70s. Frequent and long standing users consider they are familiar with it including the situations where it may deliver false information. Many intensive care units have thermodilution systems integrated into their bedside monitoring systems which analyse, store and display both raw data and derived indices.
2. Many experienced users consider, rightly or wrongly, that they make sensible use of the data in determining patient management. Many have never followed 'supranormal' goal based therapy. Haemodynamic numbers should never be considered in isolation but be evaluated along with other clinical findings. In other words all available information on the individual patient is considered before making a management decision. Hayes *et al*<sup>2</sup> certainly showed that up to 200µg/kg/min of dobutamine to attain an unattainable cardiac index and oxygen delivery level in some patients resulted in casualties in the 'supranormal' arm of their study. To state the obvious, we all know the SGC is not a treatment in itself, it is simply a diagnostic and monitoring tool. Trials to reliably evaluate the SGC will be incredibly hard to design and undertake but are possible as others have pointed out.<sup>3,4</sup>
3. It is only recently (year 2000) that some of the more promising less invasive or non-invasive technologies

have become available in Australia following Therapeutic Goods Administration approval, although they have been available in Europe for many years. Less invasive arterial thermodilution and pulse contour cardiac output (Pulsion Pacific Pty Ltd) and oesophageal doppler monitoring (Cardio Q) are both interesting and exciting innovations. Unfortunately the basic unit and consumables are quite expensive. As with the SGC it is unlikely reliable data will be obtained in all clinical situations.

Thoracic electrical bioimpedance (TEB) methods of measuring cardiac output have been around longer than the SGC thermodilution method. Though some authors have not found TEB a satisfactory method for measuring cardiac output in the critically ill,<sup>5</sup> others have considered it may offer a valuable alternative to the invasive method of measurement.<sup>6,7</sup> Again it is unlikely accurate data will be obtained in all clinical situations by the TEB method<sup>7</sup> though large studies involving a wide range of critically ill patients will be required to evaluate this.

Gastric tonometry has a regional blood flow focus. Although there is evidence that data obtained helps predict outcome, evidence for its value in guiding therapy is less convincing. A recent prospective, randomised, controlled trial concluded that the routine use of treatment titrated against pH<sub>i</sub> in the management of critically ill patients cannot be supported.<sup>8</sup>

4. Expensive and potentially hazardous technologies and procedures are often introduced into medicine without rigorous evaluation being carried out. Once they become 'established practice' it becomes very hard to convince clinicians it is ethical to conduct a trial. This is precisely the situation which led to the premature conclusion of a randomised, controlled trial of right heart catheterisation in critically ill patients.<sup>9</sup> Of course many things we do in day to day practice have never been subject to adequate trials - urethral catheters, nasogastric tubes and central venous lines to name a few.
5. Publications do exist which give some support to the case for the use of the SGC.<sup>10-17</sup>

In Australia we now have the opportunity to explore the non-invasive methods more fully. We certainly should do this. At this stage I do not share Steele and Bihari's view that "the ICU clinician is now able to obtain cardiac output, measures of adequacy of intravascular filling and tissue perfusion in more reliable ways". Many more large, well designed trials will be needed before this will be elucidated. Indeed we should not repeat what has happened with the SGC and wait

until the new technologies become established practice before getting all excited about the amount of money that is being wasted on unproven technologies!

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## Does oxygen debt play a central or peripheral role (or any role) in the pathophysiology of septic shock?

Shock is a clinical syndrome that is diagnosed in the presence of hypotension, oliguria and poor peripheral perfusion.<sup>1</sup> Sepsis is defined as an infection induced 'systemic inflammatory response syndrome', and septic shock is shock induced by sepsis.<sup>2</sup> However, as the physiological focus in shock is often on oxygen delivery, this has led many to the pathway of increasing oxygen delivery as a primary therapeutic approach to the management of septic shock.

In a 'point of view' article, the history of normal and supranormal oxygen delivery therapies in the management of critical illness is retraced by Morgan.<sup>3</sup> He takes us through the various clinical studies until he reaches the statement used by the current proponents of supranormal oxygen delivery - that therapy 'is always too late',<sup>4</sup> when attempting to explain its inability to reduce morbidity and mortality. While he points out the many weaknesses in the two studies that had provoked an editorial recommending the use of *preoperative* supranormal oxygen delivery (using catecholamines),<sup>5</sup> he also expresses justifiable difficulty in reconciling these studies with the studies that demonstrated a reduction in mortality with preoperative beta-adrenergic blocking agents.<sup>6-8</sup> Moreover, the studies reviewed appeared not to have had any of the complications associated with right heart catheterisation that are currently highlighted in the article by Steele and Bihari.<sup>9</sup>

Surgical correction and replacement of blood for haemorrhagic shock, and coronary reperfusion and balloon counterpulsation for cardiogenic shock, may improve peripheral oxygen delivery and mortality. However, septic shock is predominantly an inflammatory disorder that appears to have a more complex pathophysiology than just a reduction in peripheral oxygen delivery or utilisation.

The adverse effects of sepsis result largely from the overwhelming production of inflammatory mediators resulting in widespread tissue injury.<sup>10</sup> However, to date none of the therapies that have been used to modulate the chemical mediators responsible for the organ dysfunction have altered mortality, and treatment to support organ failure has probably contributed largely to the reduction in mortality.<sup>11</sup> While fluids and inotropic agents to increase oxygen delivery are used to manage the haemodynamic failure associated with severe sepsis and septic shock, they are not a panacea, there are numerous clinical instances where haemo-dynamic support does not impede the development of organ failure.

To state the obvious, we have a long way to go before we fully understand the pathophysiology of sepsis. The inflammatory disorder is still best treated by focusing on the management of the infective lesion (e.g. antibiotics, surgical resection and drainage) with organ support used to allow the body time to mount its own attack.<sup>12</sup> Measures to promote supra-normal oxygen delivery have not yet been shown to be beneficial.

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## Defending cerebral perfusion pressure in traumatic brain injury: the goalposts keep moving

Cerebral hypoperfusion, either due to systemic hypotension, intracranial hypertension or both, is associated with adverse outcomes following traumatic brain injury. Consequently, trauma resuscitation and intensive care protocols in head injured patients are directed at the early detection, prevention and treatment of cerebral hypoperfusion with indirect evidence that these strategies improve outcomes.<sup>1</sup> With respect to published guidelines, a cerebral perfusion pressure of at least 70 mmHg is suggested as the minimum pressure where head injured patients are most likely to be 'safe' with respect to cerebral perfusion pressure/cerebral blood flow thresholds.<sup>2</sup> The evidence for this threshold is scant, mainly due to limited human studies where both cerebral blood flow and cerebral perfusion pressure are measured directly at various time intervals following injury. The duration of defending cerebral perfusion pressure at this level remains contentious.

Key work by Bouma *et al* established that cerebral blood flow is significantly reduced in the first 18-24 hours following traumatic brain injury.<sup>3</sup> Recently, important work by Martin *et al* identified varying cerebral blood flow patterns using Xe<sup>133</sup> enhanced computed tomography (CT) and transcranial Doppler over two weeks in a cohort of 313 severely head injured patients.<sup>4</sup> Three distinct patterns of flow were identified at different time intervals. An initial hypoperfusion phase from injury to 18-36 hours that paralleled Bouma's study. This was followed by a hyperaemic phase from 24 hours to 6 days, characterised by increasing cerebral blood flow patterns peaking at 3 days before subsiding towards 'adequate' values (defined as > 30 mL/100g/min). A third phase in a

minority of patients was characterised by a vasospastic profile with relatively low cerebral blood flow measurements and high Doppler frequencies. This was attributed to post traumatic hypometabolism, resetting cerebral autoregulatory thresholds and was a feature in patients with significant traumatic subarachnoid haemorrhage.

Importantly, patients in whom prolonged duration and severity of these abnormal cerebral blood flow patterns were demonstrated had poorer outcomes that correlated with severity of primary and secondary injuries.<sup>5</sup>

These important observations have significant clinical implications. Firstly, head injured patients have abnormal cerebral blood flow patterns over the post-injury period. The assiduous defence of cerebral perfusion pressure in order to improve cerebral blood flow early in the time course following injury (up to 36 hours) is probably valid and a target of 70 mmHg is reasonable. Thereafter, cerebral perfusion pressure targets should be reconsidered if patients exhibit signs of cerebral hyperaemia. The diagnosis of this phase remains problematic, but may be considered using multimodalities such as direct measurements of cerebral blood flow, transcranial Doppler or jugular venous oximetry in conjunction with intracranial pressure monitoring and CT scanning. The impressive, but inconclusive results in patients with intracranial hypertension published by Cruz using 'optimised' hyperventilation directed at high jugular venous saturations<sup>6</sup> and those by the 'Lund' group using vasoconstrictor therapy and lower cerebral perfusion pressure<sup>7</sup> may have a pathophysiological basis if these therapies are primarily directed at patients with significant hyperaemia. Finally, the diagnosis and treatment of late traumatic cerebral vasospasm remains speculative but is an important area of future study.

The optimisation of cerebral blood flow for a given cerebral perfusion pressure remains at a critical point in managing traumatic brain injury. Much progress has been made in identifying the importance of prompt resuscitation and meticulous intensive care to minimise secondary insults. The next challenge is to move from a 'set and forget' paradigm to tailoring treatments that are applicable to the underlying pathophysiology. Although this will require improvements in technologies and assessed in adequately powered controlled trials, it remains a critical step in future management.

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## 'Spot diagnosis' in the ICU patient

In the management of any patient the diagnosis is pivotal. In reaching it, many methods of reasoning are used by the clinician, although one of the most satisfying is the 'spot diagnosis'. It provides a quick, clean and efficient way to reach the diagnosis and provides some intellectual pleasure to the one who has picked the prize. However, one has to be wary of the seductive nature of this approach, as there have been many false starts to the diagnostic (and therapeutic) road by those who jump to conclusions too early.<sup>1,2</sup>

The 'spot diagnosis' is most often performed successfully by the experienced (and cagey) clinician who uses it on a background of some clinical information, and is not usually made without more than one clinical symptom or sign to support it.

With these caveats, the *Journal* has added to its pages a new section 'Investigation vignette'<sup>3</sup> where an instance of an unusual biochemical, radiological haematological, etc. test, has led to the diagnosis in usual and unusual circumstances.

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