limbs can be avoided, if for no other reasons than patient anxiety.

From first principles (but not evidence), I suggest that the following procedures increase the risk of infection, venous thrombosis and lymphoedema in a vulnerable limb (from lowest to highest risk):

- isolated or occasional non invasive blood pressure measurements
- uncomplicated needle venepuncture or arterial blood gas sampling
- short-term venous or arterial cannulation performed aseptically. This risk is likely to depend on the duration of cannulation, risks of contamination and products to be infused (e.g. consider relative risks of intravenous fluids, antibiotics, anaesthetic drugs, and vesicant chemotherapy).
- axillary or subclavian vein access. The insertion site should be at the margin or proximal to lymphatic blockage, but still carries the risk of venous thrombosis
- midline and peripherally-inserted central catheter (PICC) line (both medium to long-term duration), with risk of infection and thrombosis

Wherever possible, I suggest that clinicians discuss these issues with the patient, considering the overall risk-benefit of using an at-risk limb on an individual case-by-case basis. It is unlikely that definitive evidence or studies will clarify this issue in the near future. Interested readers are encouraged to further study the relatively poorly-understood lymphatic system further [2, 3].

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Vascular access, cerebral air embolism and hyperbaric oxygen therapy

Although very comprehensive, the AAGBI safe vascular access guideline fails to mention the important role of hyperbaric oxygen therapy (HBOT) in the management of air embolism complicating vascular access [1].

Depending on the speed and quantity of air/gas entrainment, cardiac arrest may be irreversible. Some patients are resuscitated successfully; others have insidious onset, presenting with air in the coronary or cerebral circulation. The latter present with varied neurological signs, but a `peri-procedural stroke' should trigger suspicion of cerebral air embolism, a life-threatening emergency. Although there are case reports of these being aspirated percutaneously, the most effective treatment is urgent HBOT [2–4]. The outcome is dependent on HBOT within 6–8 h, akin to early thrombolysis in thromboembolic stroke.

Hyperbaric oxygen therapy beyond this ideal window should still be considered and discussed with experts in the field. Good outcomes from delayed HBOT may be more likely related to retrograde cerebral venous air embolism rather than cerebral arterial air embolism. Peripheral venous lines have been described many times as a source of cerebral gas embolism, and the absence of a central line should not exclude the diagnosis. The pathophysiology of gas bubbles in the cerebral vascular system and the mechanism of HBOT has been clearly described [5]. The diagnosis may be difficult, and a high index of suspicion is required in case of a peri-procedural neurological event, including any form of vascular procedure. Sometimes the diagnosis is inevitably associated with delays, but once the diagnosis is made, every minute counts. For that reason, clear guidance is essential.

The quoted incidence of 0.8% of all air embolism events translates into several dozen cases per year of significant cerebral gas embolism, which currently may be missed in the UK [1]. The incidence may be higher [2–4]. Vascular-related air

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embolism has recently been removed from the `Never Event' list in the UK and it will be interesting to see if the rate of reporting will change.

The British Hyperbaric Association has guidance on their website about how to access emergency HBOT in a suitable Category 1 unit (able to deal with critically ill patients) [6].

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Does mannitol contribute to hypotension after parenteral paracetamol administration in critical care?

Kelly et al. compared the haemodynamic effects of parenteral and enteral paracetamol in critically ill patients who might benefit from analgesia or fever relief [1]. Although I agree with the authors that hypotension after paracetamol administration could be related to the sympatholytic effects of analgesia, a further cause might be related to the co-administration of mannitol in the parenteral preparation (Perfalgen, Bristol Myers Squibb Australia, Mulgrave, Vic, Australia).

Mannitol is added to the parenteral preparation as a stabilising agent, 100 ml of 1 g paracetamol containing 5 g mannitol. In a critically-ill, hypovolemic patient who requires vasopressors, this dose is sufficient to cause adverse haemodynamic changes secondary to osmotic diuresis, requiring fluid and drug intervention [2, 3].

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Engineering jargon

While reading Raithatha and Ahmed's letter about a faulty arterial transducer set [1], I was reminded of the statement in *Good Medical Practice* that a doctor `must give patients the information they want or need to know in a way they can understand' [2].

The authors reported the defect to the manufacturer, whose reply opines that "*Trend analysis performed showed that the condition evaluated is in control. According to all exposed above, no further investigation or action is required at this time. . .*". I have no idea what these sentences actually mean. Might I suggest that guidance on using simple language with non-colleagues is added to the next edition of *Good Engineering Practice?*

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