bolus was given on the background of an intense sympathetic drive. Sudden injection of dye in this case could have precipitated neurogenic pulmonary oedema. Alternatively, the pulmonary oedema could have been caused by the release of neurotransmitters, serotonin or histamine (or a combination), as the contrast agent passed through the lungs.

Despite the theoretical advantages of these newer agents, these cases have shown that serious life-threatening events are associated with their use. This reinforces the knowledge that great care must be exercised in the cardiovascular management of such neurosurgical patients undergoing general anaesthesia for cerebral angiography, as their potential for sudden clinical deterioration remains substantial.

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Hyperkalaemia and rapid blood transfusion

I read with interest the case report by Carvalho and Quiney entitled 'near-miss' hyperkalaemic cardiac arrest associated with rapid blood transfusion (*Anaesthesia* 1999; **54**: 1094–6). Although the blood transfused was 17 days old, I would be curious to know the actual potassium content of the blood transfused. I fully agree with him in suggesting saline-washed, red cell concentrates for transfusion in high-risk patients, but this is only possible during elective blood transfusion. During emergency blood transfusion, the use of fresh blood may be safer in high-risk patients.

I would like to report a similar experience with a fatal outcome in a 35-year-old man while receiving rapid blood transfusion during an elective repair of a thoraco-abdominal aortic aneurysm. All his routine pre-operative investigations including echocardiography and electrocardiogram were within normal limits. During the repair of the aneurysm, on releasing the aortic clamp the patient developed severe hypotension with a systolic blood pressure of 40 mmHg. This was treated with rapid transfusion of crystalloid and colloid solutions, 4 units of warmed CPD-A (citrate–phosphate–dextrose–adenine) stored whole blood, 100 ml of 7.5% sodium bicarbonate and 1 g of calcium gluconate. As the hypotension persisted, the aorta was reclamped (supracoeliac) to maintain vital organ perfusion. This was immediately followed by bradycardia, widening of the QRS complexes and ventricular fibrillation. The surgeons started internal cardiac massage, as the ventricular fibrillation was resistant to 3-DC shocks. According to the surgeons the heart was very flabby. In view of the DC shock-resistant ventricular fibrillation and a flabby heart, hyperkalaemia was suspected. Blood transfusion was discontinued and hypovolaemia was treated by infusing crystalloid and colloid solutions only. The serum potassium at this point of time was found to be 7.0 $mmol.l^{-1}$. The transfused blood (unwarmed) was found to be 16 days old with a potassium concentration of 16.6 $mmol.l^{-1}$. In the presence of the aortic clamp, the coronary circulation might have received the majority of the hyperkalaemic, acidotic, citrated blood, resulting in ventricular fibrillation and a flabby heart. This was successfully treated with 100 ml of 50% dextrose and 20 units of soluble insulin infused over 15 min and intravenous furosemide 100 mg; the systolic blood pressure improved to 80 mmHg. An infusion of dopamine 10 $\mu g.kg^{-1}.min^{-1}$ was started to augment the blood pressure and the aortic clamp was successfully released. At this stage the serum potassium was found to be 5.5 $mmol.l^{-1}$, falling to 3.3 $mmol.l^{-1}$ 1 h later. Unfortunately, the patient died of multiple organ failure 3 days after the operation in the intensive care unit.

Following massive blood transfusion of stored blood, complications such as hyperkalaemia and citrate toxicity are well recognised, but fortunately very rare. A high index of suspicion is required, especially during resuscitation of an acutely injured patient in the accident and emergency department, or a patient bleeding in the operating theatre during aortic surgery.

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Use of a pleurogram to identify the position of a misplaced subclavian catheter

We should like to report an interesting and rare complication of subclavian vein catheterisation.

A 65-year-old woman with Hodgkin's lymphoma was admitted to the intensive care unit with septic shock, multiple organ failure, thrombocytopaenia and coagulopathy. Central venous access was required but was unsuccessful by the right internal jugular route. Cannulation of the right subclavian vein was therefore attempted using the Seldinger technique. Despite good back flow of blood on initial location of the vein and easy insertion of the triple-lumen catheter, we were unable to aspirate blood freely from the catheter once inserted. Extravascular placement was suspected but in view of the potential risks of further cannulation in the presence of a coagulopathy and prior technical difficulty, it was decided to perform a chest radiograph prior to removal of the catheter, to be sure that misplacement had occurred. The catheter was also connected to a pressure transducer, the recording from which is shown (Fig. 1)

