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Air embolism

SIR,—The case report by Dr A Ireland and others (13 July, p 106) serves as yet another reminder of the not so rare and very real hazard of iatrogenic gas embolism, for which compression and hyperbaric oxygen at the earliest possible opportunity remain the definitive treatment.

In the case described the suggestion that the venous gas emboli ascended the internal jugular veins into the cerebral sinuses is surely unique and yet unlikely in view of the several well established routes by which venous gas emboli may become arterialised. Although the foramen ovale remains the most probable route, and various authors describe an incidence of patent foramen ovale in between 25% and 32.5% of the population, there are several convincing case histories of fatal accidents involving so called "paradoxical arterialisation" in which there was no postmortem evidence of patent foramen ovale, and migration of venous gas emboli through the pulmonary circulation was considered to be responsible.

Whatever the route for arterialisation of venous gas emboli, there is a constant need to remember that, contrary to popular belief, fairly modest amounts of intravenous gas may have disastrous consequences. This is understandable when the central cardiovascular response to venous gas emboli is characterised by rising pulmonary arterial and right ventricular pressure, thereby facilitating arterialisation via the foramen ovale or through the pulmonary circulation.

In 1980 Peirce made the still valid comment, "The recognition and, especially, treatment of iatrogenic cerebral gas embolism has lagged far behind medical knowledge and is not generally taught."¹ At the very least, all those in hospital practice should be aware of the potential consequences of intravascular gas and know where they can gain access to hyperbaric treatment.

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1 Peirce EC II. Cerebral gas embolism (arterial) with specific reference to iatrogenic accidents. *Hyperbaric Oxygen Review* 1980;13:161-84.

SIR,—I was interested in the article by Dr A Ireland and others (13 July, p 106) that showed a successful and normal outcome after accidental air

embolism treated with hyperbaric oxygen within two hours of initial symptoms. I would like to share with Dr Ireland and others some experience gained with massive air embolism that required immediate and more invasive treatment.

In 1975 while working in a large Third World hospital I saw four cases of massive air embolism. The patients were all receiving fluids by a large peripheral vein cannula for the treatment of blood loss due to gastrointestinal haemorrhage or traumatic arterial injury. The intravenous fluids were contained in glass bottles, and the flow rate was increased by pumping air into the bottle with a filtered surgical bicycle pump. It was therefore not surprising that when the fluid ran out undetected air under high pressure gained access to the superior vena cava and right side of the heart. Obviously, the longer the condition remained undetected the larger the air embolism. Consequently, these patients were found collapsed with signs of right heart chamber air embolism, unconscious, severely hypotensive (blood pressure <50 mm Hg), with tachycardia and tachypnoea, and with a loud precordial millwheel murmur.

The rapid measures of preventing further entry of air into the vein by the head down left lateral position and oxygen failed to bring an improvement in blood pressure and conscious level (as did the head down position with the patient almost vertical) so right ventricular aspiration with a cardiac needle was performed by the xiphisternal route. Large amounts of frothy air were aspirated with rapid improvement in blood pressure to near normal levels, return of consciousness, reduction in tachycardia and tachypnoea, and virtual resolution of the millwheel murmur in all cases except one. This patient was thought to have had the largest embolus and died before cardiac aspiration could be performed. The three other patients were subsequently treated with 100% oxygen, nursed in the head down left lateral position, and recovered completely with no detectable neurological deficit. An early necropsy of the fourth patient showed large amounts of air in the superior vena cava, inferior vena cava, right heart chambers, and pulmonary arteries. Interestingly, a small amount of air was also found in the aorta, and in the absence of septal defects this finding perhaps adds weight to the possibility of air passing from right to left through intrapulmonary shunts or small bubbles coalescing after leaving the pulmonary circulation.

It can be seen from this experience that right heart aspiration was life saving. Thus I would suggest that if the simple postural measures for treating right heart air embolism fail to produce an improvement in cardiac output, by removing the block to pulmonary outflow, then right ventricular aspiration should be performed. Aspiration may be facilitated if the central venous line is still in situ and also during cardiac surgery on bypass, when it may be possible to reverse the bypass pump and remove the air.

If, however, after the return of cardiac output residual neurological signs are detected then at that stage the patient should be considered for hyperbaric oxygen treatment, for as Dr Ireland and others stated a successful and normal outcome is possible even if treatment is started several hours after the onset of symptoms.

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SIR,—The report by Dr A Ireland and others (13 July, p 106) is a timely reminder of the many hazards that may be associated with the use of central venous catheters. Although they call for

this type of catheter to be discontinued, we are not told which type of catheter should be used.

As far as I am aware none of the current generally available central venous catheters requires the introducer to be left in situ. The failure to remove such an introducer can lead to several serious complications. Not only does it leave the patient at risk of air embolus if the catheter falls out but there is also the risk of rapid blood loss. In addition, the space between the introducer and catheter provides a route by which infection can easily enter the blood stream. The retention of the introducer may also make it difficult to ensure adequate fixation of the central catheter, and this may have allowed the patient in question to remove the line herself.

The reported complication rates associated with central venous cannulation vary from 0% in experienced hands¹ to 30%.² Many of these complications can be avoided by careful attention to technique and adequate training. Unfortunately, many central catheters, not all of which are necessary, are inserted by inexperienced staff, often under difficult conditions and without supervision. Some handbooks give guidance on how to carry out this technique,^{3,4} but there is no substitute for closely supervised training, not only in the performance of this technique but also in the indications for its use.

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Prevention of hazardous drinking

SIR,—In their leading article (22 June, p 1849) Drs R D Johnson and R Williams stated that, "exactly what constitutes hazardous drinking has now been agreed. . . ." We were surprised to read this as, so far as we are aware, there is no consensus regarding this, and in fact our paper on safe limits of drinking published in the same issue of the *BMJ* emphasised this point particularly.¹

We agreed that it is of vital importance to detect hazardous drinking early, and clearly it is important to be able to give advice on safe upper limits of alcohol consumption, but at the present time the quantitative nature of this advice varies considerably. As we pointed out in our original paper, the Royal College of Psychiatrists recommends an upper limit of 56 units per week irrespective of sex and the Health Education Council advises a consumption of under 21 units a week for men and 14 for women.² The consumption figures quoted in Drs Johnson and Williams's article as being hazardous are levels of over 80 g of alcohol per day for men and 40 g for women, which correspond to approximately 70 units per week and 35 units per week, respectively.

The survey that we carried out on safe upper limits of consumption among 70 researchers on alcohol in Britain confirmed this lack of consensus with opinions varying from seven to 52 units per week in men and from under six to 55 units per week in women.² Furthermore, our more recent survey showed a similar variation,¹ although on the whole lower limits were advised than those suggested by the alcohol experts in the previous survey.²