Hyponatraemia-related death after paediatric surgery still exists in France

Editor—Guidelines related to maintenance fluids in children have been recently implemented in the UK by the National Patient Safety Agency (NPSA) and a recent editorial commenting on these changes included comparison with the French situation. Although ‘safe’ fluids have been available in France for a while, deaths still occur as a consequence of hyponatraemia as shown by this example extracted from the recent French survey on perioperative deaths.

A 4-yr-old, 15 kg, female child with no medical history (ASA I) was admitted for dental extraction. The anaesthetist in charge was trained to anaesthetize children. Anaesthesia was started at 10:30 a.m. using sevoflurane and sufentanil and tracheal intubation was performed. A peripheral venous access was maintained with dextrose 5%. At 1:00 p.m., the child vomited swallowed blood, cried, and complained of headache. Because vomiting continued, the nurse decided to continue infusion of dextrose 5% solution to counterbalance the effects of fasting. On the next morning (10:00 a.m. and 1:00 p.m.), the child was again examined by two anaesthetists because of persisting sedation. From surgery to the next day, the child had received 1200 ml of dextrose 5% and had had 12 episodes of vomiting. The child was then admitted to the paediatric medical unit at 2:00 p.m. and the child was said to be comatose (Glasgow coma score 6). A blood sample revealed hyponatraemia at 120 mmol litre⁻¹ and haemoglobin concentration at 10.9 g dl⁻¹. The infusion was modified to dextrose 5% +60 mmol litre⁻¹ of sodium chloride over 24 h. In the evening, respiratory distress, a worsening coma, and unilateral mydriasis led to tracheal intubation and transfer in a paediatric ICU. A cerebral CT scan showed diffuse oedema while echocardiography disclosed a very dilated and hypokinetic left ventricle. The patient died several hours after arrival in the ICU.

Search of contributory factors showed that local guidelines on fluid administration in children did not exist in this institution and prescription rules were not clear. Many decisions were also left to the nurse. Apart from these system failures, it is of note that none of the physicians or nurses involved evoked that symptoms could have been related to hypotonic infusion.

We have found that the rate of deaths totally or partially related to anaesthesia for the age range 0–7 yr is 0.6 per 100 000 anaesthetics (95% CI: 0.12–3.2 per 10 000). This translates into a rate of three deaths each year for this age group in our country, of which one is related to hyponatraemia-induced complications. Although the number of accidents is small, their impact is huge and unacceptable. This cannot be considered as a residual small number because the deficiencies observed are amenable to improvement. A solution containing Ringer’s lactate and dextrose 1% also known as B66 is available in France, but was not used. This solution is now commercially available from several industrial manufacturers which should facilitate implementation of its use of such solutions in the UK. Although this wide availability should have also facilitated its use in France, a number of institutions and physicians are not aware of the risk associated with infusion of hypotonic solutions in children. The prescribing of i.v. fluids should be as rigorous as the prescribing of drugs. This suggests that actions such as the one driven by the NPSA are to be commended and are also necessary in France.

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Nitrous oxide and postoperative cardiovascular morbidity

Editor—I read with interest Professor Myles’ article on the effect of nitrous oxide on plasma homocysteine and folate in patients undergoing major surgery. I would like to query the conclusion with respect to cardiovascular morbidity in this cohort. In the patient characteristic data in
Table 1, the patients are well matched until the medical conditions are considered. However, smokers, patients with coronary artery disease, patients with heart failure, and patients with previous stroke seem to be over-represented in the nitrous oxide group. There is no comment as to whether this reached statistical significance, or could have affected the cardiovascular morbidity experienced.

I look forward to the results coming through from ENIGMA-II to determine the impact of nitrous oxide on patients with pre-existing cardiovascular morbidity, though this will not affect my practice as I ceased to use N₂O many years ago.

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Editor—We thank Dr Connor for his letter. Random allocation to groups should result in comparable baseline characteristics, but it remains possible that small differences can still exist. In our study, there were slightly more patients with some risk factors for coronary artery disease in the nitrous oxide group (as suggested by Dr Connors), yet there were slightly more patients with other risk factors in the control groups (older age, diabetes). These random differences are inevitable, and are unlikely to explain the differences in outcome that we observed.

The prime purpose of presenting baseline characteristics—typically Table 1 of a clinical trial—is to demonstrate comparable groups in all respects except that of the intervention of interest (in our case, exposure to nitrous oxide). Although some choose to compare these factors with hypotheses testing, it is wrong and potentially misleading. If groups are randomly allocated, then significance tests only test the success of the randomization. A significance level of 0.05 merely suggests that one in 20 comparisons will be significant purely by chance. Importantly, there may be a clinically significant difference between the groups which is not detected by significance testing, yet such an imbalance may have an important effect on the outcome of interest. We, also, await the results of ENIGMA-II with interest.

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Paradoxical gas embolism during hysteroscopy

Editor—I read with great interest the report by Rademaker and colleagues1 of paradoxical gas embolism during hysteroscopy. I have two questions I wish to pose, having had a similar case myself.2 First, since this was a hysteroscopy using normal saline as the distension medium (thought to lower the risk compared with CO₂), where did the authors suspect the gas to have originated from? There have been suggestions that inadequate purging of air from the equipment can cause embolism. Did the authors suspect this may have been the case? Secondly, and perhaps more importantly, I question why the authors did not consider hyperbaric oxygen therapy (HBO). Urgent exposure to HBO is the current accepted therapy for cerebral gas embolism.3,4 With a confirmed diagnosis of arterialization of gas via transthoracic echocardiography, it should have been considered. The consequences of arterial embolism, as the authors pointed out, include hemiparesis, blindness, and coma. A previous study5 suggests that the outcome is worst when treatment is delayed (more than 6 h) and arterial gas embolism is worse than venous embolism (recovery 35% vs 67%). Was hyperbaric therapy unavailable or discounted for some reason?

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Editor—In response to the two questions from Dr Sherlock, I have the following comments. First, regarding the possibility of improper purging of air from the hysteroscopic instruments, we used normal saline as a distension medium and carefully purged air from all lines and hysteroscopic instruments. After cervical dilation, the exposure of the open cervix to room air was kept to a minimum leaving the cervix closed using a tenaculum forceps. A continuous out-flow system was used so that the distension medium is refreshed actively and bubbles and debris are flushed away. We considered it unlikely that air entrainment played an important role in our case. For that reason, we think that the development of gas produced by the electrosurgical vaporization of myomas, fibroids, endometrial, uterine tissue, or both is much more important factor. Indeed, Munro and colleagues6 demonstrated in vitro that hysteroscopic electrosurgical vaporization using either monopolar or bipolar diathermia results in the production of significant quantities of the highly soluble gases: hydrogen, carbon monoxide, and carbon dioxide. The second question addressed the use of HBO in the case of arterial embolism. Indeed, HBO is considered the mainstay of therapy in arterialization of gas, especially when cerebral gas embolism is accompanied by neurological deficits. Fortunately, our patient did not develop neurological sequelae. She recovered uneventfully after a period of supportive