Thorascopic sympathectomy

Sir,—I read with interest the review article by Fredman, Olsfanger and Jedeikin on thorascopic sympathectomy in the treatment of palmer hyperhidrosis. The authors reported on the cardiac complications and noted the two reported cases of intraoperative cardiac arrest, the postulated pathophysiology being that preexcitation of the myocardium by sympathetic ablation lowers the threshold for ventricular fibrillation.

I wish to report on a 31-year-old woman undergoing left-sided thorascopic sympathectomy. Induction of anaesthesia was uneventful and a left-sided 37 Ch Mallinckrodt double-lumen tube was inserted without difficulty. Monitoring included electrocardiogram (ECG) with lead 2 selected, non-invasive arterial pressure (AP), pulse oximetry ($Sp_O_2$), and inspiratory and expiratory gas concentrations. The left lung was collapsed and deflated by clamping the endobronchial tube and opening to air. A size 12 mm trocar was introduced into the chest between the third and fourth ribs in the mid-axillary line under direct vision and carbon dioxide 2 litre insufflated with a pressure limit of 5 mm Hg. Although the lung was deflated, it was difficult to see the T4 ganglia and therefore additional carbon dioxide was insufflated, with the same pressure limit. At this point the patient developed sudden onset of ST segment depression on the ECG. This was not accompanied by changes in F or R waves suggesting that this was not solely the result of a change in the cardiac axis as a result of tension pneumothorax. Arterial pressure decreased from 90/60 to 60/40 mm Hg, with no significant change in heart rate. The surgeon was informed of these changes, and immediate release of carbon dioxide insufflation was performed to augment lung collapse and protect the lung during insertion of the endoscopic instruments. As a result the patient developed sudden onset of hypotension and a decrease in myocardial ischaemia. The two reported cases of cardiac arrest occurred during left-sided procedures. It is possible that excess pressure, especially during left-sided procedures, leads to myocardial ischaemia and hence to arrhythmias. Possible mechanisms include: coronary vessel compression as the myocardium is traversed; coronary vessel spasm; reduced coronary flow as a result of torsion of the mediastinum; and reduced coronary flow caused by hypotension. In this case it seems unlikely that hypotension alone was the cause of ischaemic changes as an arterial pressure of 60/40 mm Hg in a healthy, young patient would not usually be expected to be accompanied by ST segment depression. As non-invasive arterial pressure monitoring has the advantage of being time-cycle dependent, it is possible that arterial pressure during the acute event was much lower than actually captured and displayed. Thus one cannot state that “an arterial pressure of 60/40 mm Hg in a healthy, young patient would not usually be expected to be accompanied by ST segment depression.”

When providing anaesthesia for thorascopic sympathectomy the advantages and potential pitfalls of the various techniques should be remembered. When using a tracheal tube, surgical exposure is dependent on continuous insufflation of carbon dioxide into the intrapleural space. To avoid inadvertent tension pneumothorax a pressure-limited, variable flow insufflator is used. In contrast, when using the endobronchial technique, carbon dioxide insufflation is performed to augment lung collapse and protect the lung during insertion of the endoscopic instruments. Therefore, collapse of the entire lung is accomplished and maintained by the endobronchial tube and carbon dioxide insufflation plays no part in providing surgical exposure. As a result, a pressure-limited, variable flow insufflator is not necessarily required. With the endobronchial technique a tension pneumothorax may occur and extreme caution should be exercised if additional gas is insufflated after lung collapse. Finally, irrespective of the technique used, hypoxaemia is a major concern during thorascopic sympathectomy. It is for this reason that we recommend vigilant $Sp_O_2$ monitoring during lung collapse.

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Simulated anaesthetic emergencies

Sir,—We congratulate Drs Byrne and Jones for their study of an anaesthesia simulator as a tool for performance evaluation. We have operated a realistic simulation centre for several years and have been involved extensively in the research of anaesthesia simulation as a potential assessment tool. We would like to offer the following comments.

The purchase of a realistic simulator represents only a fraction of the cost for the construction of a simulation centre. In fact, the operating costs (mostly salaries for staff) outweigh this initial capital cost within 2 yr. Trying to save money by advocating the initial purchase of a less realistic simulation system may therefore confer only a short-lived benefit and limit the option of expanding simulation of realistic environments in the long term. Furthermore, simplified part-trainers appear to offer a less valuable learning experience compared with realistic simulation environments. In order to substantiate claims that subjects use the same mental models as in real life it is necessary to present data to document that participants indeed "suspend disbelief" (e.g., rating scales on realism). Otherwise the correlation of findings to clinical events remains speculative.

Physician assessment in anaesthesia to date remains largely a subjective process. Availability of simulators offers the opportunity to expose different individuals to identical scenarios in a high fidelity environment and compare their performance to a "gold standard" reference group. The development of a new assessment tool, however, requires careful evaluation of its reliability and validity in order to define its value compared with accepted evaluation tools.

Drs Byrne and Jones did not measure the reliability of their proposed evaluation tool and given that they did not define the final end-point of a successful intervention, inter-rater reliability may indeed be poor. They neither defined nor validated their time definitions (e.g. the "gold standard" time until the "gold standard" intervention) and did not provide measurements of a reference group (e.g., additional reference group of certified anaesthetists).

It is of interest that the responses to their simulated emergencies appeared to yield such variable results (e.g. some items such as awareness had consistently good responses whereas others such as asystole had mostly poor responses). Statistical tests to measure internal consistency are available (e.g. Cronbach’s alpha) and are helpful in designing tests with internally consistent items. The clinical implications of untreated awareness are very different from untreated asystole and the different responses could be a result of varying degrees of perceived difficulty. In that case, individual scores should be weighted accordingly when combined into the final score for statistical analysis.

We believe that simulation has the potential to address some of the shortcomings of traditional evaluation tools. However, practical and clinically relevant scoring systems need to be developed and the reliability of these scoring systems demonstrated. A data base of performance measures of various clinical groups (e.g. the "gold standard" vs trainees of various experience) should be generated and demonstrate feasibility and validity of the evaluation tool. Until these studies are complete it is premature to declare that "simulators can be used to assess the ability of anaesthetists to deal with anaesthetic emergencies".

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Sir,—We applaud Drs Byrne and Jones for their study of the performance of anaesthetists at different levels of experience in the management of low-fidelity simulations of critical events. The authors have detected the same characteristic of anaesthetists’ technical responses as were found over the past 8 yr in previous studies using both hands-on simulators and computer-screen simulators. These characteristics are: (i) significant variability in response between individuals and between events; (ii) modest improvement in performance caused by increased anaesthesia experience, especially compared with the most junior personnel; (iii) occurrence of serious, even catastrophic, errors at every level of experience; and (iv) lack of uniform adherence to published guidelines for managing critical events.

These characteristics appear to be reproducible features of anaesthetist performance. That they can be detected with instruments of different fidelity is not surprising (one can see craters on the moon with the naked eye and with a telescope). Furthermore, Byrne and Jones compared their results using the low-fidelity ACCESS simulator with those obtained with early versions of our more hands-on simulator (CASE 1.2/1.3). CASE 1.2/1.3 had greater fidelity than ACCESS because it provided monitoring data via actual clinical monitors, but compared with today’s simulators and simulation environments, CASE 1.2/1.3 was itself a low-fidelity simulator.

There were several methodological problems in this study. The presence of the “tutor” acting as an assistant (while operating the simulator) could have altered the results, especially if the tutor was not blinded to the experience level of the subjects. Also, having a tutor to pass equipment is not routine in most anaesthetic environments. Actions were timed to the nearest 30 s, a rather coarse measure given the rapidity with which some diagnostic and therapeutic manoeuvres can be completed (some can even be conducted in parallel), as demonstrated in previous studies in which the simulations were videotaped. Hypervigilance (expecting problems that are not present) and cavalier behaviour (not responding avidly because the patient is not a real human being) are common findings in simulation studies and can affect performance, but these issues were not mentioned by the authors.

We view with scepticism the contention of the authors that “to measure performance… it is not necessary to construct a ‘high-fidelity’ environment, but merely to provide enough realism to enable those tested to suspend disbelief”. First, the authors have not proved that they accurately tested the true performance of their subjects, only that they identified characteristics of performance similar to those measured previously. Second, there is no proof that “…subjects used the same mental models as they use in real life and results are therefore valid.” No systematic analysis of mental models was conducted. Third, while we agree that suspension of disbelief is necessary when simulating a human being, the authors provided only an anecdote suggesting that one subject suspended disbelief; they provided no data that subjects did so in general. Hence, if the goal was to determine the level of simulator fidelity required to support a given type of performance assessment, this study shed little light on the question. To do so it would be necessary to test the same individuals using comparable scenarios on simulators of differing fidelity.
Moreover, the type of performance assessed in this study, and in previous studies cited, is only a limited portion of clinical skill–technical performance. When simulations are conducted using realistic replications of a clinical environment, complete with actual equipment and supplies, and a full set of clinical personnel such as nurses, surgeons, anaesthetists and technicians, a more complete set of clinical skills can be probed. Over the past 7 yr many investigators have been examining more comprehensive aspects of performance, including crisis management behaviours such as leadership, communication and teamwork. The different techniques to measure these aspects of performance and technical management of complex situations are currently under development. We believe that to address the full complexity of clinical management for research studies or for training it is necessary to recruit clinicians in settings that challenge the full scope of their abilities. However, as for other part-trainer and low-fidelity simulators, ACCESS may be useful for investigating and teaching restricted aspects of patient care.

Finally, many simulation centres have expanded their attention beyond anaesthesia to conduct research and training on comprehensive management issues in a variety of medical domains, including combined team performance in the operating room, delivery suite, emergency department and intensive care unit. Such expansion is desirable not only to maximize the benefit of simulation-based training, but also as a way of sharing expenses. Such expansion is desirable not only to maximize the benefit of simulation-based training, but also as a way of sharing expenses.

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Pigs are not humans

Sir,—It was with considerable interest that we read the article of Lentschener and co-workers on the haemodynamic repercussions of pneumoperitoneum in the pig.1 Although we share the authors’ concern concerning extrapolation of animal data to clinical experience, several points in this study which contradict examples in the literature may have resulted in inappropriate conclusions.

Several studies on the haemodynamic effects of pneumoperitoneum have been carried out in the pig2–4 and have given different results to those of Lentschener and colleagues, being similar to changes observed in humans.5 Most of the studies quoted by Lentschener and colleagues were carried out in the dog6,7 which, on the basis of the authors’ conclusions on the differences between species, are equally difficult to extrapolate. Moreover, in humans, some studies have not always found the changes usually observed during pneumoperitoneum.8,9

Some factors may influence the haemodynamic repercussions of pneumoperitoneum. The most important would appear to be the state of hydration of the patient; hypovolaemia limits the haemodynamic effects of peritoneal insufflation.3,10 This could be the case in the study of Lentschener and colleagues where significant vascular filling was used (Ringer’s lactate 500 ml h–1) for animals weighing approximately 18 kg. The high pulmonary capillary pressure and cardiac flow rate values agree with this hypothesis. Another factor which influences venous return during pneumoperitoneum is the position of the patient11 and in this study it is not clear in what position the animals were placed. The type of anaesthesia can also have considerable influence on the haemodynamic repercussions observed during pneumoperitoneum12 and it would be interesting to know the exact doses of fentanyl and ketamine used during this study. In addition, we are not sure that domestic pigs weighing 40 kg should be considered as adults.

Without wishing to cast doubt on the results, we feel that experimental and anaesthetic conditions can considerably influence the results of experimentation, which implies that the conclusions should be guarded, especially when they contradict those of most of the literature.

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1. Lentschener C, Benhamou D, M’Jahed K, Moutafis M, Fischler M. Increased intraperitoneal pressure up to 15 mm Hg does not reliably induce haemodynamic changes in pigs. British Journal of Anaesthesia 1997; 78: 576–578.
We did not investigate pigs in other positions as we failed to reliably reproduce haemodynamic changes associated with pneumoperitoneum in the supine position.

Bazin and Schoeffler suggested that the way we conducted general anaesthesia may have altered our findings. First, general anaesthesia is induced in pigs with i.m. ketamine in most investigations. I.m. ketamine was used for induction of general anaesthesia in most studies mentioned in Bazin and Schoeffler’s letter (references 1–4, 7, 8). Ketamine stimulates the sympathetic system and is likely to cause increases in arterial pressure and systemic vascular resistance. As increases in arterial pressure and systemic vascular resistance were expected in our investigation but did not occur, we conclude that ketamine had no effect. The method of anaesthesia was discussed extensively in our article and in our opinion we definitively ruled out possible pharmacological contamination of our model.


**Obstruction of the lumen of a plastic 15-mm connector inserted into a cut paediatric tube—Is it possible to avoid this damage?**

Sir,—We read with interest the report of a critical incident by Gupta and Harry1 illustrating the risks of inserting a connector after cutting a paediatric tube. This problem, encountered with Portex 8.5-mm paediatric tracheal tube connectors, was described originally by May and Kirton in 1993.2 At about the same time, we had a similar experience with a 3.5-mm internal diameter (id) tracheal tube (TT) produced by Rusch, Germany. Immediately before anaesthesia was induced in an 8-month-old boy with an incarcerated inguinal hernia, the 3.5-mm id TT had been cut to 12 cm. While trying to insert the plastic 15-mm connector into the proximal end of the cut tube, I noted that the connector buckled inwards, occluding its lumen. The damaged connector was removed and a fresh connector, originally packed with a 3.5-mm id TT, was inserted into the cut tube without damage. Anaesthesia was uneventful.

Subsequent careful inspection of pre-packed paediatric tubes (Rusch, Waiblingen, Germany; Portex Ltd, Hythe, England; Mallinckrodt Medical UK Ltd) showed that: (1) the proximal end of all commercially available paediatric tubes is pre-widened by the manufacturer (e.g. 6–8 mm of a 3.5-mm id TT is pre-widened to approximately 4.5 mm id) (fig. 1); and (2) the tube section of the plastic 15-mm connector is thin and vulnerable (fig. 2).

Thus while it is very easy to insert the original plastic 15-mm connector into the original pre-widened paediatric TT, it requires a very smooth touch to insert the same connector without damage into a tube from which the pre-widened section has been cut away. In our case, the failed attempt to insert the 15-mm connector actually widened the proximal end of the cut tube sufficiently for another connector of equal size to be inserted without any damage.

With respect to our data, we strongly recommend moderately stretching the proximal end of cut tubes crosswise at 9 and 12 o’clock with, for example, an artery forceps or a similar instrument, to avoid damage to the plastic 15-mm connector while it is being inserted into the cut tube. To test the reliability of such self-widened cut tubes into which connectors had been inserted as described, we placed several under water, increased the pressure within them and found that the tubes were always airtight. The elasticity of the tube and the conical shape of the tube section of the connector both work to advantage here. Our method has worked without flaws in all subsequent operating room applications.

Another possibility of avoiding the critical incident described by Gupta and Harry3 is to use a commercially available special paediatric airway adapter (available from 2.5–5.0 mm id; Ohmeda, Madison WI, USA) if the tube is to be cut. The tube part of this adapter is thick and resistant (see fig. 2) and can be inserted into a cut tube without damage. Nevertheless, here too widening of the cut paediatric tube as described above facilitates insertion of this special paediatric airway adapter. Additionally, this special paediatric airway adapter features minimal dead space and an integral luerlock port for sampling end-tidal values.


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**Figure 1** End and side views of an original (A) and manually cut (B) paediatric tracheal tube. Note the different internal diameters.

**Figure 2** C = Plastic 15-mm connector with a vulnerable tube section provided with a commercially available paediatric 3.5-mm id tracheal tube. D = Special paediatric airway adapter, 3.5 mm id, with a resistant tube section and integral luerlock port.