Dexmedetomidine for resection of a large phaeochromocytoma with invasion into the inferior vena cava

Editor—Dexmedetomidine is increasingly used in patients on mechanical ventilation in intensive care units.1 Perioperative use of dexmedetomidine provides a steady haemodynamic course and blunts fluctuations at stressful moments like intubation and extubation.2 In phaeochromocytoma surgery, dexmedetomidine could be a useful anaesthetic adjunct in minimizing episodes of abrupt arterial hypertension expected during manipulation of the tumour. We report use of dexmedetomidine in a patient with a large phaeochromocytoma with invasion into the inferior vena cava (IVC), in whom adrenalectomy with excision of the invaded part of IVC was planned. The patient received a 2-week course of phenoxymenzamine and propanolol before surgery. On arrival in the operating theatre, arterial pressure through an intra-arterial catheter in the left radial artery measured 120/65 mm Hg. A loading dose of dexmedetomidine 2 mg kg\(^{-1}\) was infused over 10 min followed by an infusion at 0.7 mg kg\(^{-1}\) h\(^{-1}\). Anaesthesia was induced with fentanyl 100 mg, thiopentone 250 mg, rocuronium 50 mg and esmolol 30 mg. The highest arterial pressure (AP) during intubation was 145/80 mm Hg. Intraoperative monitoring included ECG, AP, CVP, saturation, end-tidal carbon dioxide and volatile agent, airway pressure and temperature. Anaesthesia was maintained with isoflurane 0.6% in oxygen and nitrous oxide, remifentanil at 0.1 mg/C0\(^{-1}\) min\(^{-1}\) and cisatracurium. Labetalol 20 mg had been administered before direct tumour manipulation. During direct tumour manipulation, the remifentanil was increased to 0.2–0.3 mg kg\(^{-1}\) min\(^{-1}\). Sodium nitroprusside was administered at a low dose between 0.2 and 0.7 mg kg\(^{-1}\) min\(^{-1}\). Two doses of esmolol 20 mg were given to control spurious increase in AP. The AP during dissection around the tumour ranged from 80/40 to 145/90 mm Hg. Upon clamping of IVC, the remifentanil, nitroprusside and dexmedetomidine infusions were stopped. During the IVC clamping period of 25 min, the AP was stabilized with phenylephrine and epinephrine (total dose of 1 mg and 340 mg, respectively), and ranged from 70/35 mm Hg to 120/65 mm Hg. Upon release of the IVC clamp, the AP dropped to 80/40 mm Hg, which quickly returned to above 110/60 mm Hg with fluid and dopamine infusion. The dopamine infusion was stopped before the end of surgery (total dose 5.81 mg). The surgery lasted 4 h 21 min and the patient was extubated awake uneventfully 15 min later. A morphine patient-controlled anaesthesia was prescribed for postoperative pain relief. The patient made an uneventful recovery.

In resection of a large phaeochromocytoma with IVC invasion, haemodynamic instability especially with severe episodic hypertension from surgical stimuli and tumour manipulation are expected. Preoperative α-blockade, intraoperative vasodilators and increasing anaesthetic depth are common measures to smoothen out the haemodynamic course and prevent hypertensive crises.3 Remifentanil is effective in blunting the sympathetic response to noxious stimuli and has been used in phaeochromocytoma excision to control intraoperative haemodynamic instability,2 but significant hypotension and bradycardia, and a large increase in plasma catecholamine levels and marked hypertension during manipulation have been reported.5 Dexmedetomidine, a highly selective α2-adrenoceptor agonist, has sedative and analgesic properties.6 It attenuates sympathoadrenal responses to tracheal intubation and surgical stimuli and has a significant anaesthetic-sparing effect when used intraoperatively.7 8 In order to blunt the intubation stress, we administered a high loading dose of dexmedetomidine of 2 mg kg\(^{-1}\) before induction and the patient remained haemodynamically stable during intubation. After the loading dose, the infusion was maintained at 0.7 mg kg\(^{-1}\) h\(^{-1}\) until clamping of the IVC. In the remaining surgery, haemodynamic stability was maintained with inotropic support, which was stopped at the end of surgery.

In summary, we describe the management of a patient for excision of a large phaeochromocytoma with invasion into a major vessel, in whom dexmedetomidine was found a useful anaesthetic adjunct to maintain steady haemodynamics and to prevent abrupt hypertensive crises.

A. Y. C. Wong
C. W. Cheung
Hong Kong, China

Unrecognized malfunction in computerized patient simulators

Editor—Life-like computerized patient simulators are now widely used in clinical training. In addition to their established role in crisis resource management education,1 2 simulators are also being used to teach pre-clinical physiology3 and to evaluate clinical performance.4 The widespread adoption of simulation-based crisis resource management training is a direct analogy to similar training in aviation and has been driven by the need to provide education that should reduce both medical error and risks to patients.5 This growth in medical simulation has been

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facilitated by the commercial availability of simulators, but it has taken place without independent verification of simulator performance characteristics and without defined standards of simulator adequacy. This contrasts with the aviation experience where standards for simulation are defined in advisory circulars and regulations. Such standards are intended to ensure that the level of simulation fidelity meets the goals of training and evaluation; thereby ensuring uniformity, while minimizing the risk of negative transfer (whereby inappropriate learning in a poor quality simulation could transfer to real life).

In an attempt to independently verify the performance characteristics of one brand of commercially available simulators, we undertook a multi-site comparison of the performance of six fully operational METI (Medical Education Technologies Inc., Sarasota, FL, USA) simulators. The METI Human Patient Simulator (HPS) is a plastic manikin with respiratory and cardiovascular functionality. This includes mechanical lungs, palpable pulses, heart and lung sounds; with life-like cardiorespiratory data that are displayed using standard clinical monitors. The HPS is operated by a computer program with sophisticated physiological and pharmacological models that rely on feedback from a range of peripheral devices. Clinical problems or scenarios are created by manipulating these integrated physiological and pharmacological models using METI-HPS scenario files.

To generate physiological data for comparison, we distributed an identical scenario file to the participating centres. Five of the simulators were METI-C simulators running software version 5.5, and simulator 1 was a METI-B running software version 5.2. The simulators were all breathing room air and half were intubated with a tracheal tube. The scenario began with a 2 min physiologically normal baseline, followed by automatic transitions that included progressive haemorrhage and increases in both shunt fraction and oxygen consumption. The final transition to total neuromuscular blockade resulted in death from hypoxaemic cardiac arrest. Three iterations of the scenario were run on each simulator. At 1 min intervals the simulated physiological data were automatically recorded to log files. Data were analysed using repeated measures of analysis of variance using SYSTAT version 10.

The data from simulator 6 were strikingly different from the others, caused by an unrecognized defective gas analyser that was impairing the physiological model. These data were excluded from the statistical analysis. The physiological trends recorded from the five remaining simulators were similar, but there were statistically significant differences (P<0.0001) between simulators for heart rate, systolic and diastolic blood pressure, cardiac output, tidal volume, ventilatory frequency, alveolar partial pressure of oxygen and carbon dioxide, and arterial partial pressure of carbon dioxide. The differences were greatest for tidal volume and are shown in Figure 1. There were no statistically significant differences within any one simulator during the repeated iterations of the scenario.

We were surprised by the variability that we found. One simulator, although in use and apparently functioning normally, had a significant hitherto unrecognized malfunction. The five remaining simulators showed variability that although life-like, was greater than we had expected from computerized simulators. We assume that some of this variability may have been caused by the presence of a tracheal tube, but this remains unproven.

The important finding was an unrecognized malfunction in a fully operational simulator. This is important when considering the educational and evaluative roles that simulators are likely to play in the future. We must be confident that simulators are both reliable and valid, not only because of the need to avoid negative transfer and potentially harmful training, but also in the interests of fairness to examination candidates when simulators are used in certification and re-certification. Our initial question regarding the variability in simulated physiological performance in properly functioning simulators remains inadequately answered.

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A. L. Garden1
B. J. Robinson2
C. U. Arancibia3
T. J. Carron3
S. Monk4
J. Vollmer4
W. Heinrichs4
C. Grube5
B. M. Graf5
E. B. Johnson3

1Boston, MA, USA
2Wellington, New Zealand
3Richmond, VA, USA
4Mainz, Germany
5Heidelberg, Germany

4 Rosenblatt MA, Abrams KJ. The use of a human patient simulator in the evaluation of and development of a remedial prescription for an
Pacemakers and defibrillators: anaesthetic implications

Editor—I read with interest the review article concerning the anaesthetic implications of pacemakers and defibrillators, by Salukhe, Dob and Sutton.1 I enjoyed reading the article and felt that it clarified a very complex topic that often causes much angst to anaesthetists, when presented with patients with implantable devices in situ.

In the current climate, mobile telephones are becoming more accepted in the hospital and operating theatre environment. Many hospitals are now changing their policies involving the use of mobile phones in theatres, allowing their use at distances of 1 metre or more from anaesthetic machinery.

However, it must also be remembered the potential for mobile phones to interfere with pacemakers has been recognized for the past 10 yr.2,3 Interference of pacemaker function caused by electromagnetic interference (EMI) from mobile phones occurs mainly when the phone rings, or is switched on or off. The effect on pacemaker function may be inappropriate inhibition of pacing, triggering of pacing stimuli, reversion to asynchronous mode or change in the rate of pacing. Interference occurs most frequently when unipolar leads are used and when the device is programmed to a high sensitivity.4 Hayes and colleagues found that placing a mobile phone directly over a pacemaker caused interference in 20% of cases, and in 7.2% of cases caused symptoms.5

In view of the fact that mobile phone manufacturers advise keeping mobile phones >15 cm away from pacemakers, operating department personnel should be reminded of this fact and the potential for EMI-induced problems when looking after a patient with an implantable pacemaker in theatres.

W. Marchant
London, UK