Ropivacaine test dose in extradural anaesthesia

Morton and colleagues have described the use of 0.75% ropivacaine for extradural anaesthesia, and in the process have clearly demonstrated the unsuitability of this agent as a test dose. The aim of an extradural test dose is to identify inadvertent i.v. or intrathecal catheter placement. It should have high sensitivity, but produce symptoms that are safe. Morton and colleagues have administered large i.v. doses of ropivacaine to two patients, despite negative test doses of ropivacaine 22.5 mg. This is not surprising, as Scott and colleagues gave 10 mg min−1 i.v. for up to 15 min before producing symptoms. We do not, however, suggest the use of a larger dose of ropivacaine as a test dose. While this may increase sensitivity in identifying vascualar cannulation, dangerously high spinal anaesthesia may result in intrathecal injection. Wahedi, Nolte and Klein have produced safe spinal anaesthesia with 0.75% ropivacaine 22.5 mg in non-pregnant subjects, but there are no data to clarify a safe maximum intrathecal dose of ropivacaine in the obstetric population.

The most sensitive pure local anaesthetic test dose for identifying intravascular injection of which we are aware is lignocaine 1 mg kg−1, proposed by Michels, Lyons and Hopkins. However, Richardson and Wissler described abrupt and life-threatening high spinal block after intrathecal test doses of only 45 mg of lignocaine in two patients. Michels’ test dose is therefore clearly potentially dangerous.

In summary, we suggest that it is not possible to use a pure local anaesthetic test dose of sufficient dosage to identify the intravascular space without producing dangerously high spinal anaesthesia if injected intrathecally. We see no alternative but to use separate agents to identify inadvertent intravascular or intrathecal injection. Intrathecal catheter placement can be excluded easily using bupivacaine 10 mg. We remain unconvinced of the sensitivity and specificity of adding adrenaline to this test dose, and indeed have some concerns over its safety. Perhaps the addition of a potent short-acting opioid such as remifentanil warrants some investigation in this role? The final logical but somewhat cumbersome suggestion is to follow a negative intrathecal test of bupivacaine with a second test of lignocaine 1 mg kg−1.

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Sir,—We thank Drs Dressner, Adams and Klein for their interest in our article. We agree that ropivacaine 22.5 mg is an unsuitable test dose and we should have given greater emphasis to this in our article. But we do not think that this could have been predicted by the work of Scott and colleagues. Our test dose was given over 20 s, considerably faster than the 10 mg min−1 used by Scott and colleagues. We do not agree that intrathecal catheter placement can be easily (our italics) excluded with bupivacaine 10 mg. Prince, Shetty and Miles compared plain bupivacaine 8 mg by the subarachnoid and extradural routes, and could only discriminate reliably between the two routes by testing straight leg raising after 10 min. We do not agree with the suggestion of adding remifentanil to the test dose; this would seem to be exchanging the known risks and limitations of current test doses for the unknown.

Our own routine practice is to use lignocaine 80 mg as a “test dose”. It is our opinion that this dose, if given rapidly, elicits symptoms of early systemic toxicity. We acknowledge that the addition of adrenaline 1 in 200,000 may increase sensitivity and specificity. The potential for high subarachnoid block is real but at least there is early clear evidence of inadvertent subarachnoid placement of the catheter. The crucial point about test doses, and one we continually emphasize to our trainees, is that a complacent attitude to a negative test dose is dangerous; a negative test dose does not completely exclude misplacement of the catheter. When an aspiration test and a test dose are negative, large doses of local anaesthetic must still be given slowly. We believe that this, and the lower toxicity of ropivacaine compared with bupivacaine, is why the two patients in our study who received accidental i.v. injections did not come to serious harm.

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Use of inhaled nitric oxide in the critically ill

Sir,—We read with interest the article by Cuthbertson, Stott and Webster on the increasing use of inhaled nitric oxide (iNO) in the critically ill. We would disagree with their statement that there is no evidence that iNO improves patient outcome. One study in neonates with persistent pulmonary hypertension demonstrated a reduction in the need for extracorporeal membrane oxygenation (ECMO) from 71% of patients in the control group to 40% in those receiving iNO (P = 0.02). There was no difference in mortality between the two groups (7.1% in the control group vs 6.6% in the iNO group). A study of iNO in neonates with hypoxic respiratory failure demonstrated a decrease in the use of ECMO from 54.5% in controls to 38.6% in those receiving iNO (P = 0.014). Again, mortality was similar (16.5% in the control group vs 14% in the iNO group). ECMO is expensive, invasive and unavailable in many centres. It is associated with morbidity, but has been shown to reduce mortality in term neonates with respiratory failure. Using iNO to reduce the use of ECMO would seem appropriate. The children involved in studies of iNO are assessed neurodevelopmentally at 18–24 months, which may provide further support for the use of iNO. We accept that it would be wrong to extrapolate the studies directly to adults, although they indicate that there is a real role for iNO, and we await studies in older children and adults to resolve the place of iNO in these groups.

iNO is stored at 1000 ppm in nitrogen, an asphyxiating mixture. In most units, iNO is added distally to the ventilator without oxygen monitoring. This is potentially dangerous. Providing less than 80 ppm of NO is used, as recommended by Cuthbertson, Stott and Webster, depression of P0.2 is less than one-tenth of the administered P0.2. This requires that the inspired NO concentration should be measured at all times, as they recommend, to ensure that no more than NO 80 ppm is given. NO is also available at varying lower concentrations for use in calibration of monitoring equipment. If a cylinder containing 200 ppm is used to provide an inspired NO concentration of 80 ppm, depression of P0.2 is 40% of the set P0.2.

We consider that the risks of administering a hypoxic mixture
using NO are real but they have been supplantted by concerns about only NO and the higher oxides rather than its nitrogen carrier. It is important that those who use iNO appreciate all of these risks.

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Management of the airway and ventilation during resuscitation

Sir,—In their recent article on the management of the airway and ventilation during resuscitation, Gabbott and Baskett mentioned an intubation aid used to confirm tracheal intubation called SCOTT.1 I would like to point out that SCOTTI (Sonomatic confirmation of tracheal intubation) was first marketed by Penlon in April 1995. This was in response to a need for a device capable of providing “instant” confirmation of tracheal intubation and against a background of ever increasing risk of litigation against anaesthetists. Although the device generated a great deal of interest, sales proved disappointing and at the end of 1996 a commercial decision was taken to discontinue marketing the SCOTTI device. I believe a similar device continues to be available in the USA, although I am not aware of any devices working on a similar sonomomatic principle being marketed elsewhere.

I trust this information is of interest and clarifies the current position regarding the SCOTTI device.

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Weaning: is the work of breathing via a tracheostomy tube similar to that via a tracheal tube?

Sir,—Both the editorial by Shneerson1 and a letter by Bapat and Verghe2 highlight interesting aspects on difficult weaning from ventilation. While agreeing fully with the main message of the letter2 which emphasizes the advantages of cuff deflation to facilitate weaning, I have reservations about the comment made on the work of breathing in spontaneously breathing patients.

It is logical to assume that a decrease in peak intrapulmonary pressure to approximately 15 mm Hg from a normal value of 2.5 to 6 mm Hg while breathing normally through a tracheal tube of 9.0 mm internal diameter (id) reflects an increase in the work of breathing. But is it appropriate to extrapolate this observation and assume that a tracheostomy tube of size 8.0 or 9.0 mm id produces changes of similar magnitude? Work of breathing is dependent mainly on three factors: elastic resistance of the lung; non-elastic viscous resistance; and resistance to air flow. Increase in the work of breathing caused by airway instrumentation is generally believed to be a result of the increase in air flow resistance, even though one study could not confirm this.3 While it is true that resistance to flow via a tube is dependent much more on internal diameter than on length, halving the length of a tube can reduce resistance by up to 50%4 and doubling the length can double the resistance.5 This holds true when flow is laminar. (The relationship between the length of a tube and resistance encountered is complex and there is no analogue for Poiseuille’s law in the case of turbulent flow.) The length of a size 9.0 mm id tracheostomy tube is 10.5 cm (Portex, profile cuff type) compared with tracheal tubes cut at 21–24 cm. However, firm conclusions on the work of breathing in tracheostomized patients may only be made from clinical investigations, and not from simple application of mathematical formulae.

Another point worth mentioning is that if a patient starts speaking soon after deflation of a tracheostomy tube cuff without closing its external orifice, there may be a plug of mucus partially obstructing the tip of the tube. This is probably one of the causes of a dramatic improvement of any patient after removal of a tracheostomy tube or extubation!

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4. Mushin WW, Jones PL. Flow of fluids through tubes:

Sir,—We appreciate Dr Kodakat’s interest in our comments. The main objective of our letter was to emphasize that a tracheal tube or a tracheostomy tube increases the workload of breathing relative to the normal respiratory tract.

The peak flow rate of a normal subject at rest is 0.3–0.5 litre s⁻¹. Flow rates greater than 0.25 litre s⁻¹ via a tube of less than 10.0 mm internal diameter (id) are turbulent. Therefore, Poiseuille’s equation which deals with laminar flow can only be used partially to deduce flow characteristics during intubation. The diameter, length, curvature and material of a tube affect its flow resistance, and thus different tracheostomy tubes, even of similar size and diameter, may have different flow resistances.

To achieve normal ventilation, work is performed to overcome the elastic and frictional impedances of the lungs and chest wall. A greater swing in pleural pressure is required to achieve a given flow resistance. The elastic and frictional impedances of the lungs and chest wall are used partially to deduce flow characteristics during intubation. Poiseuille’s equation which deals with laminar flow can only be used partially to deduce flow characteristics during intubation.

Elastic impedances predispose to fatigue. The peak flow in such patients could easily increase to 1 litre s⁻¹, resulting in a markedly increased flow resistance with increasing flow rate in a non-linear manner, and could add substantially to the total work of breathing. In this group of patients, intrapleural pressure could even be greater than −15 mm Hg. In contrast, in patients with severe respiratory muscle weakness, changes in intrapleural pressure could be smaller. However, both groups of patients may benefit from a reduction in respiratory resistance caused by cuff deflation.

We agree that for a given patient the work of breathing through a tracheostomy tube would be less compared with an equal diameter tracheal tube. There are no comparative studies of the effect of different sized tracheal tubes or tracheostomy tubes on intrapleural pressure in patients with respiratory failure. Our comment that “changes in intrapleural pressure caused by a size 9.0 mm tracheal tube could be similar to that of a size 9.0 mm tracheal tube” was based on these factors. It was not meant that different sized tubes or tracheostomy tubes have the same flow resistance.

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Continuous intra-arterial blood-gas monitoring

Sir,—After reading the study on the usefulness of continuous intra-arterial blood-gas monitoring in patients undergoing thoracic procedures, I feel it necessary to comment on some of the issues raised by the investigators.

The authors stated that the Paratrend 7 (PT7) blood-gas monitoring system has been validated by only one set of investigators in collaboration with the manufacturer. I would like to draw their attention to a multicentre study from North America independently validating the PT7 system. Second, it is stated that a broad range of pH, Pco2 and Po2 values were not evaluated in the original studies. I would like to point out that a similar range of pH (7.1–7.57), Pco2 (2.65–6.85 kPa) and Po2 (8–68 kPa) values have been evaluated in an earlier study by Venkatesh, Clutton-Brock and Hendry.

The accuracy of the base excess and bicarbonate calculations are a function of the algorithm built into the blood-gas analysers and the monitor and accuracy of the Pco2 measurement. Dissociation in accuracy between the two methods for those two variables may be a reflection of differences in the algorithm or accuracy of the Pco2 measurement.

While discussing the accuracy of the PT7 and blood-gas analysers, it is stated that “the accuracy and variability of blood-gas analysers are not known exactly”. Studies by Hansen and colleagues, and Metger and colleagues have clearly demonstrated the bias and precision of blood-gas analysers when measuring blood-gas tensions in blood. The authors have alluded to several factors which influence the accuracy of blood-gas analysis. Even controlling for the factors listed, the accuracy and reproducibility of blood-gas analysers remains an issue. It must be remembered that most blood-gas analysers do not use primary quality control material such as tonometered blood. This significantly limits any conclusions which can be drawn on the accuracy of an intravascular device. While studies such as these are important, clinical outcome and cost effectiveness studies are the ones which will influence the wholesale acceptance of intravascular sensing devices.

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Sir,—I read with interest the article by Zollinger and colleagues independently confirming the good clinical performance of the Paratrend 7 (PT7) multiparameter intra-arterial blood-gas monitoring system first described by this institution. Several points raised by the article require further discussion. The authors gave the impression that the sensor may be re-calibrated when inserted into a vessel by using data from a bench top blood-gas analyser. This is incorrect. Comparison between values obtained with the PT7 and bench top analyser can be made and the original calibration curve adjusted in the light of this comparison; this is somewhat different to re-calibration. When inserted into a vessel it is not possible to recalibrate the sensor without loss of sterility. The authors also stated that the PT7 can measure blood-gas variables at 37°C or at patient (intravascular) temperature. In fact, all readings by the PT7 are made at patient (intravascular) temperature and then corrected, if required, to 37°C, as opposed to bench top analysers which measure at 37°C and correct to patient temperature. A subtle difference, but differences in the
temperature correction algorithms may lead to discrepancies in displayed values. With regard to the statistical evaluation of the data, there are three principal methods of describing relationships between continuous variables: (1) assessment of whether the value of one variable to be predicted from any known value of the other variable, for which the technique of linear regression is used; and (3) assessment of the level of agreement between the values of the two variables by calculating the mean difference between readings (bias) and the degree of variability (precision).

For the study in question it is perhaps useful to know that high (or low) values from one monitor are reflected by high (or low) values from the other. Calculation of correlation coefficients and P values indicates how likely it is that a high (or low) value from one monitor is reflected by a high (or low) value from the other. No information on the level of agreement between the actual values displayed can be derived from this type of analysis. The PT7 monitor is designed to measure intra-arterial blood-gas tensions, not predict the values displayed by another monitor, thus calculation of regression statistics does not provide useful information on the performance of the sensor. Thus, results from correlation statistics should be interpreted with caution in this type of study and the technique of linear regression analysis should not be used.

The conclusion that the level of agreement between HCO₃⁻ and base excess was relatively poor requires further comment. As part of an ongoing study in our department, 28 arterial blood samples were aspirated from patients in our intensive care unit and analysed simultaneously by a single operator using two bench top blood-gas analysers (ABL 300; Radiometer, Copenhagen, Denmark and NOVA Stat Profile Plus 9; NOVA Biomedical, Waltham, MA USA). For assessment of bicarbonate concentration and base excess, the bias between machines was −0.74 mmol litre⁻¹ and −0.57 mmol litre⁻¹, respectively. The precision of these readings (1.96 σ) was 4.50 and 4.47 mmol litre⁻¹, respectively. These values are of a similar magnitude to those obtained when comparing the PT7 system with the blood-gas analyser available for the study in question.

Finally, presentation of the levels of bias and precision in the units of the variables can be misleading. For example, for the above analysis the levels of bias and precision appear almost equal. However, the normal range for bicarbonate is numerically considerably larger than base excess and if bias and precision are calculated in percentage terms a different appraisal of the level of agreement is evident. For the data on bicarbonate, bias and precision were −1.96% and 16.92%, respectively. For the data on base excess, bias and precision were −43.25% and 206.72%, respectively. Therefore, when this type of data are presented, and values for bias and precision presented in the units of measurement, percentage bias and precision should also be quoted to give a more comprehensive assessment of the level of agreement.

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Sir,—Thank you for the opportunity to respond to the comments of Dr Graystone and Dr Venkatesh on our article. They highlighted some important technical aspects. During use of the intra-arterial sensor system, a correction of the Paratrend 7 (PT7) values on the basis of those obtained by in vitro blood-gas measurement is possible. This correction is dependent on measurement or recalibration, the difference being rather semantic. However, as described, no such correction was used in our study. This is important when rating the agreement of PT7 with laboratory in vitro blood-gas measurements, which is considered to be the gold standard, and also when comparing results of our study with previous clinical work, where such corrections were used.

Furthermore, we agree that the algorithms built into the PT7 monitor and the bench top blood-gas analysers are crucial for the accuracy of all calculated parameters. Both the correction for measured blood-gas values from (intravascular) patient temperature to 37°C (and vice versa) and calculation of bicarbonate and base excess rely on these algorithms. Therefore, a study on the accuracy of a new system, which provides these values, indirectly includes comparison of the algorithms incorporated in this new system with those of the in vitro blood-gas analyser. Interestingly, the accuracy of these calculated variables which are displayed on the PT7 monitor has not been clinically investigated previously. The conflict remains, that “... the clinical performance of an optode-based blood-gas monitor must be judged in comparison with the gold standard, the well validated blood-gas analyzer, for which clinical performance cannot be specifically quantified”. At the time of preparation of our manuscript the PT7 sensor had been validated by only one group of authors. Since then Abraham, Gallagher and Fink have reported on its clinical evaluation in different intensive care units. They followed a study design different from ours with special emphasis on continuous monitoring during a prolonged period of time. However, the results of their study may have been influenced by the fact that eigth different bench top analysers from two different manufacturers were used in three different institutions. Furthermore, we stated that the PT7 system had been validated for limited blood-gas ranges only. This is true, as the ranges reported by Venkatesh, Clutton-Brock and Hendry do not include clinically critical values of hypoxaemia and hypercapnia. However, we had the opportunity to monitor extreme values of arterial P0₂ (6.1 kPa) and P0₂ (9.5 kPa) during thoracoscopic surgery. Calculation of bias and precision is considered standard statistics for comparing this type of data. In addition, linear regression analyses were performed to facilitate comparison with previous work. It is important to note that both types of statistical analyses consistently revealed good agreement between PT7 derived and blood-gas analyser derived values for arterial P0₂, P0₂ and pH, and also consistently documented significant scattering between PT7 derived and blood-gas analyser derived values such as bicarbonate and base excess.

Finally, although clinical outcome and cost effectiveness studies are lacking in many other monitoring tools currently used in anaesthesia and intensive care, we agree fully that evidence-based, outcome-related data are mandatory for general acceptance of continuous intra-arterial blood-gas monitoring.

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The future of ethical treatment

Sir,—Dr Mohr and Kettler raised important issues in their article on the ethical aspects of resuscitation. Previously published guidelines have stressed the importance of obtaining patient views in decisions on whether or not to resuscitate. In one large study examining the views of the elderly on admission to hospital, 186 patients from a total of 400 were deemed mentally incompetent to complete a questionnaire satisfactorily. In such cases more emphasis may be placed on the views of relatives, and with an ageing population this is likely to occur more frequently.

Experience in the USA has shown that the judicial review of continuation of “futile” end of life treatment often fails to resolve differences between medical attendants and the next-of-kin. Unfortunately, in many cases the catalyst to a decision is the health insurance provider who may cap the amount spent on such care. We must be careful that we share the load of the decision making process without overburdening patients, relatives or ourselves with what will undoubtedly be increasingly difficult ethical dilemmas as the population becomes relatively more aged.

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Sir,—Dr Shirley raises the issue of overburdening patients, relatives and health care providers with the decision making process in ethical conflicts. In general, physicians are obligated to help patients and their surrogates to make competent decisions. However, it is important to distinguish between true advice and the more paternalistic approach of leaving the decision to the doctor. We agree that the expectations on the decision making capacity of patients and their relatives should not be over-estimated; it might be limited because of the emotional involvement and lack of medical knowledge and comprehension. In case of conflict, such as the surrogates’ wishes to continue futile treatment, we have to accept the obligation and responsibility family members feel towards their relatives. However, refusal of futile treatment should not be judged as unjustified paternalism, as in some situations physicians or other health care providers have to solve dilemmas and make decisions which they cannot transfer to relatives.

We feel that the concept of patient’s (and surrogate’s) autonomy should not be abandoned, but its value is not absolute. The question that must be answered is how to distinguish between justified and unjustified refusal of patients’ or relatives’ wishes. The apparent dilemma might be solved by an approach summarized as clinical pragmatism: a method of assessing the relevant facts; diagnosing the moral problems; considering the options; and negotiating goals and offering an (ethical) acceptable treatment plan that coheres with agreed goals. In several ethical dilemmas, a process of creative problem solving might be appropriate instead of asking the attorney to make the decisions for us.

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