

CORRESPONDENCE

Anaesthesia and myotonia

Sir,—We read with interest the review article on anaesthesia and myotonia [1]. Myotonia dystrophica is an uncommon disorder with as yet no consensus of opinion regarding the ideal anaesthetic for these patients. Propofol has been used in this disorder with variable responses, including prolonged recovery, altered dose-response curves and precipitation of the myotonia. We report three additional cases where propofol was used successfully for induction and maintenance of anaesthesia.

The first case was a 53-yr-old man with moderate myotonia dystrophica, ischaemic heart disease (including myocardial infarction 1 yr previously) and marked peripheral vascular disease. He presented with an acute on chronic ischaemic leg, requiring urgent exploration of his femoral artery.

Temazepam 10 mg was administered orally 1 h before operation. After preoxygenation, anaesthesia was induced with fentanyl 250 µg, propofol 50 mg (the effect noted), followed 1 min later by another 25 mg. A propofol infusion was then given at a rate of 6 mg kg⁻¹ h⁻¹ for 15 min, then 4 mg kg⁻¹ h⁻¹ for another 10 min and finally 2 mg kg⁻¹ h⁻¹ [2] for the remainder of the operation. Atracurium 20 mg was also given. No other sedative or analgesic drugs were used. The patient underwent femoral embolectomy without any major problems.

The propofol infusion was discontinued 55 min after induction. Within 3 min his respiratory efforts were adequate and the trachea was extubated. However, although respiration was satisfactory and the airway was well maintained, it was a further 65 min before verbal contact was made with the patient.

The immediate postoperative period was uneventful but on day 7 he began to develop increasing weakness, including bulbar weakness, and he died on day 14.

The second case was a 27-yr-old female with moderately severe myotonia dystrophica causing cataracts, marked muscle weakness, slurred speech and swallowing difficulties. However, she was mobile and had no respiratory or cardiovascular system involvement. She was admitted for laparoscopic cholecystectomy.

The patient was premedicated with temazepam 10 mg, and heparin 5000 u. was administered s.c. After preoxygenation, fentanyl 100 µg was given and the effect observed. Three 25-mg increments of propofol were given at 1-min intervals to assess the patient's sensitivity to propofol. When it was evident that the patient was not sensitive, propofol 125 mg was given followed by atracurium 15 mg. Anaesthesia was maintained with 0.5–1.0 % isoflurane and nitrous oxide in oxygen. The operation and anaesthetic proceeded uneventfully with a further 100 mg of fentanyl and 10 mg of atracurium being required. Morphine 10 mg was administered at the end of operation. The patient made a rapid recovery and was fit to leave theatre recovery 35 min after the anaesthetic.

The third case was female with moderate myotonia undergoing total abdominal hysterectomy. She had mild muscle weakness with no other problems (she had only been diagnosed because of family screening.) After premedication with temazepam 10 mg, anaesthesia was induced with alfentanil 2 mg followed by propofol 140 mg given slowly. Intubation was performed without formal paralysis and anaesthesia was maintained with a propofol infusion of 10 mg kg⁻¹ h⁻¹ followed by 8 mg kg⁻¹ h⁻¹ followed by 6 mg kg⁻¹ h⁻¹ [2], and an infusion of alfentanil 1.5 mg h⁻¹. The operation lasted 40 min and the patient rapidly regained consciousness. Her trachea was extubated within 5 min of termination of the infusions. She made an uneventful recovery and was fit to go to the ward within 45 min.

The effect of propofol on myotonic patients is clearly unpredictable. There have been several reports of its use and adverse effects, including prolonged recovery [3, 4], marked sensitivity to its depressant effects on induction [3] and precipitation of myotonia on induction [5]. A computerized delivery system was used by Tzabar and Marshall [6] with no immediate problems but was found to be associated with markedly prolonged recovery.

Hopefully, the cases we have described will add to the cumulative knowledge of the effect of propofol on myotonic patients. In general they emphasize the unpredictability of this condition, with clinical severity not necessarily being a useful

marker. Propofol should only be administered therefore if indicated and in a very slow controlled way. We agree with the recommendation of Russell and Hirsch [1] that ICU facilities should be available whenever a myotonic patient is anaesthetized.

G. W. JOHNSON
S. CHADWICK
P. EADSFORTH
I. HARTOPP

Department of Anaesthesia
North Manchester General Hospital
Manchester

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Negative extradural pressure may not be caused by tenting of the dura

Sir,—I read the most interesting article by Shah [1] and the correspondences by Serpell [2] and Shah [3] on extradural and subarachnoid pressures. In the discussion section of Shah's article he stated, "During extradural pressure, the pressure of the advancing needle indents the dura [4, 5] and creates a sub-atmospheric pressure in the extradural space" [1]. The recent introduction of combined spinal and extradural techniques casts doubt on this theory.

The evidence of extradural pressure being negative, including the hissing sound of air as it is being sucked through the needle, is well established. One combined spinal and extradural technique is to pass a spinal needle, for example a 27-gauge Whitacre needle, through the extradural needle after the latter has reached the extradural space. In doing so, one feels that the spinal needle has to be advanced for a few millimetres beyond the tip of the extradural needle before touching the dura. This indicates that the tip of the extradural needle is separate from the dura, not tenting it. Moreover, one has to advance the spinal needle for another 2–3 mm before feeling the characteristic "snap" of the dura. This indicates laxity of the dura before the spinal needle pushed it forward. It is also against the dura being already "dimpled" by the extradural needle. Accordingly, tenting of the dura by the extradural needle may not be the cause of the negative extradural pressure.

E. ABOULEISH
Department of Anesthesiology
University of Texas at Houston
USA

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Sir,—Thank you for the opportunity to reply to Dr Abouleish's comments on my article on extradural pressure [1]. Sub-

atmospheric pressure in the extradural space was first reported by Janzen [2] in 1926. From his observations and experiments, he concluded that the negative extradural pressure was an artefact arising in the extradural space when the advancing needle produced an indentation in the dura. Two years later Heldt and Moloney [3] claimed that there was a true negative pressure of between -1 and -10 mm Hg in the extradural space. Since then, there has been continued argument over the cause of negative extradural pressure.

Almost universally, the reports of extradural entry pressure state it to be subatmospheric. Advancing an extradural needle without a stylette may produce a hiss when air enters the extradural space. Sagarnaga [4] exploited this sign to identify entry into the extradural space. The likely cause of this hiss is air rushing in to occupy the space created by dural indentation. Disconnecting the syringe after the loss of resistance technique may also produce a hiss. However, this hiss is caused by air rushing out of the extradural space.

Dawkin's [5] indicator consists of a vertical nylon tube, 10 cm long, with an internal diameter of 3 mm, filled with saline. The effect of gravity provides a positive pressure to the column of saline. Using a 1-ml syringe as a modified Dawkin's indicator, Chester [6] noticed that the saline meniscus descended by 2–14 mm when the needle entered the extradural space. He claimed that the extradural entry pressure was -2 to -14 mm of saline. However, a syringe measures volume and not pressure. The maximum depression in the level of saline in his 1-ml syringe indicator was 14 mm. The volume of saline occupying 14 mm of length of a 1-ml syringe is 0.25 ml of saline. Therefore, the maximum amount of saline that entered the extradural space in his patients was 0.25 ml. Advancing the extradural needle caused a cone-like indentation in the dura in dogs; the base of the indentation had a radius of 1.5 cm [7]. If an extradural needle penetrated 3 mm into the space and produced an indentation of 1 cm radius, the volume of the cone-like space created would be 0.3 ml. In Chester's patients the maximum amount of saline aspirated into the extradural space was nearly the same as the volume of space created by dural dimpling. This suggests that the extradural space is potential only. If there was naturally occurring subatmospheric pressure in the extradural space, one would expect aspiration of a larger volume of saline.

In my study, Macintosh balloons exerted a positive pressure. Yet they failed to deflate in the extradural space, again suggesting that the extradural space is a potential space with the dura resisting inward movement.

A pressure of approximately 15 cm H₂O of cerebrospinal fluid supports it from its inner aspect. An advancing needle may easily displace the cerebrospinal fluid and indent the dura mater which is a tough membrane [8]. Therefore, it is likely that during the combined spinal and extradural technique, the spinal needle further indents the already indented dura. The amount of dural indentation depends on the sharpness of the needle and also the speed of insertion [2].

J. L. SHAH
Department of Anaesthesia
City Hospital
Birmingham

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Ambulatory extradural analgesia

Sir,—Buggy, Hughes and Gardiner [1] concluded that mobility during extradural analgesia for labour pain is unsafe. Although our techniques are referenced [2], the regimen adopted for extradural analgesia in their study is different from the one used at Queen Charlotte's Hospital.

The degree of motor and sensory block incurred during extradural analgesia is related to the quantity of bupivacaine administered per hour. In the study of Buggy, Hughes and Gardiner, bupivacaine 30 mg was administered into the extradural space to establish the block (15 mg in the test dose and then 15 mg with fentanyl 30 µg, 3 min later). Our technique involves an initial subarachnoid dose of bupivacaine 2.5 mg with fentanyl 25 µg, followed by top-up doses of 0.1 % bupivacaine 10 ml (10 mg) with 0.0002 % fentanyl (20 µg). Bupivacaine 30 mg used in this way, in divided doses, provides analgesia for an average of 4 h [2]. It seems pointless therefore to compare the two groups.

We feel that women can accurately assess their own ability to walk during extradural analgesia. The ability to walk depends on more complex neurological processes than can be assessed by abstract clinical tests for posterior column function. Indeed, on formal testing, many of the women in our delivery suite who walk quite safely while receiving extradural analgesia, at various stages of their labour, do have a degree of impairment in the long tracts. However, they are accompanied by another adult and are observed closely. In this way, we have had no serious problems as a result of walking in over 6000 "ambulatory" extradurals performed since September 1992. Many patients with a permanent posterior column deficit secondary to disease are ambulatory.

The combined spinal–extradural technique that we use is not associated with a "relatively high incidence of post-dural puncture headache". If the Whitacre needle is passed no more than twice, the rate of headache not attributable to dural puncture by the Tuohy needle is 0.13 %.

The analgesia produced by Buggy, Hughes and Gardiner was "consistently suboptimal". This compares with complete pain relief in 91 % of women at 8 min, and all women at 20 min, using our regimen [4].

M. COX
G. LAWTON
S. GOWRIE-MOHAN
T. PRIEST
A. ARNOLD
B. MORGAN

Queen Charlotte's and Chelsea Hospital
London

- Buggy D, Hughes N, Gardiner J. Posterior column sensory impairment during ambulatory extradural analgesia in labour. *British Journal of Anaesthesia* 1994; **73**: 540–542.
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Sir,—Thank you for the opportunity to reply to the comments of the Queen Charlotte's group. It was not our intention to compare directly their regimen of combined spinal–extradural analgesia with ours using the extradural component alone. As stated in our report, we sought to define the neurological effects of the extradural component, that is 0.1 % bupivacaine (15 ml) combined with fentanyl 2 µg ml⁻¹. We found a significant incidence of posterior column sensory impairment 30 min later [1]. The Queen Charlotte's workers did not report the time interval between their intrathecal and extradural analgesia and commencement of walking, but it seems reasonable that their patients would have exhibited a similar degree of posterior column signs, if they had been specifically sought. The effect of bupivacaine 2.5 mg in combination with fentanyl 25 µg administered *intrathecally* on posterior column sensory modalities awaits further investigation. We speculate that it may be equivalent to the additional 15 mg we gave *extradurally*.

In their original report [2], these workers stated that they used

15 ml, not 10 ml, of 0.1 % bupivacaine (15 mg) with fentanyl 2 µg/ml (30 µg) as their first extradural top-up, and also that a 2.3 % overall incidence of post-dural puncture headache occurred.

We are not so sure that women can adequately assess their own ability to walk during extradural analgesia. Much of the work on the role of proprioception in the physiology of walking was of necessity conducted in animal models, but it highlights the importance of posterior column spinal cord function in walking [3]. Moreover, we found a negative correlation between patients' subjective assessment of their ability to walk (taken while they were standing) and the presence of posterior column sensory impairment, suggesting that women may have a false sense of security induced by preservation of motor function [1].

We acknowledge that some patients with posterior column deficits secondary to pathological processes (for example diabetics) are ambulatory, but this must be distinguished from the transient, iatrogenic neuropathy of extradural analgesia. We believe that we, as anaesthetists, would be responsible in the event of mishap as a result of walking during the effective period of the extradural block. Nevertheless, the Queen Charlotte experience of over 6000 uneventful ambulatory extradurals is impressive, and suggests that the potential hazard to safe mobilization may be theoretical. However, we note that they now acknowledge the presence of posterior column sensory deficits in their own patients, and that accompaniment by another adult is mandatory. We feel this point has been underemphasized, and should be stressed to obstetric anaesthesia units and mothers who may be about to undertake a programme of ambulatory extradural anaesthesia. We stress that we are not opposed to the concept of ambulatory extradural analgesia, but with patient safety of paramount importance, a cautious embrace of this modification of conventional extradural analgesia is indicated.

D. BUGGY
N. HUGHES
J. GARDINER
*Department of Anaesthesia
Rotunda Hospital
Dublin*

1. Buggy D, Hughes N, Gardiner J. Posterior column sensory impairment during ambulatory extradural analgesia in labour. *British Journal of Anaesthesia* 1994; **73**: 540–542.
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Postoperative delirium in the elderly

Sir,—I read the recent review on postoperative delirium in the elderly [1] with interest. This article highlights almost all of the points in this poorly understood topic.

One of the causes of postoperative delirium and confusion which was not discussed in this article is hypocapnia. Hypocapnia can occur during anaesthesia, when controlled ventilation and a high minute volume are used, especially with a circle absorber. It is not uncommon to see end-tidal carbon dioxide readings of 3–4 kPa in routine anaesthetic practice [2].

The major effect of hypocapnia in the central nervous system is a reduction in cerebral blood flow and shrinkage of the brain. Cerebral blood flow may be reduced by 30 % or more as a result of the direct effects of hypocapnia on cerebral vessels [3]. The symptoms of hypocapnia are not influenced by the rate of reduction of end-tidal carbon dioxide [4]. This could cause clouding of consciousness and analgesia because of depression of the reticular formation. In patients with a P_{aCO_2} of less than 3.5 kPa resulting from hyperventilation, there is evidence of post-operative prolongation of reaction time lasting 3–5 days [5].

In order to avoid this complication it is important to keep a close watch on end-tidal carbon dioxide in elderly patients, especially during controlled ventilation. End-tidal carbon dioxide in these patients should not be allowed to decrease below 4.5 kPa unless specifically indicated.

D. BOSE
*Department of Anaesthetics
Torbay Hospital
Torquay*

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Sir,—We did in fact mention hypocapnia as a potential risk factor for delirium (page 680, col 2, para 2), although there are no reports to our knowledge of delirium (as opposed to abnormal psychometric test results) developing in hypocapnic patients. Having said that, we agree with the points raised by Dr Bose.

S. O'KEEFE
*Department of Geriatric Medicine
Royal Liverpool University Hospital
Liverpool*

Condensation on tracheal tubes is commonly seen with oesophageal intubation

Sir,—I wish to comment on the review of accidental oesophageal intubation by Clyburn and Rosen [1]. They end their brief discussion on the use of condensation of water vapour as a clinical test by stating that "this sign has, not surprisingly, been found to be extremely unreliable, and is of limited value even as a supplementary sign." Two references are quoted [2, 3]. The extent of the unreliability of condensation or misting is not discussed, and is, I feel, not appreciated by many anaesthetists.

In one well documented case, bilateral chest movement, condensation in the tube and the presence of "rhonchi" were interpreted initially as tracheal intubation with bronchospasm [4]. Gillespie and colleagues [2] studied 15 patients. When a single breath was given, condensation was seen on all eight tracheal tubes (100 %) and on two of seven oesophageal tubes (29 %). Andersen and Hald [3] studied 40 patients. Condensation was seen in all 40 tracheal tubes (100 %) and in 34 of 40 oesophageal tubes (85 %).

During a study of oesophageal intubation [unpublished], I tested the negative pressure oesophageal detector device before and after ventilation through a tracheal tube placed in the oesophagus. Thusfar, in the first 60 adult patients, all 60 tracheal tubes have had condensation (100 %), while 42 of 60 oesophageal tubes (70 %) had very obvious condensation. In 18 of the 60 oesophageal tubes (30 %), there was either no condensation ($n = 14$) or slight condensation seen only on close inspection ($n = 4$). These values yield a sensitivity of 100 %, a specificity of 30 %, a positive predictive value of 59 % and a negative predictive value of 100 % for condensation on tubes in detecting oesophageal intubation.

The assessment of condensation was subjective, and I was not blinded (as the oesophageal detector device had just been tested on the oesophageal tube). My results are, however, similar to the results of the blinded study conducted by Andersen and Hald [3].

When my findings are combined with those of previous studies [2, 3], we have a total of 115 patients, with 215 observations for condensation. The data suggest that: the absence of condensation indicates oesophageal intubation reliably (negative predictive value = 100 %); and the presence of obvious condensation is not a very specific indicator of tube position (specificity = 27 %, positive predictive value = 58 %).

R. P. HARIDAS
*Department of Anaesthetics
University of Natal
South Africa*

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Peripheral nerve damage and regional anaesthesia

Sir,—I wish to comment on the recent critical editorial on nerve injury during regional anaesthesia [1]. I agree with the authors that it is probable that only a proportion of neural symptoms after regional anaesthesia occur as a direct result of the anaesthetic technique. However, there is a paucity of satisfactory epidemiological data with regard to the incidence of such symptoms and their aetiology. In their editorial, Moore, Mulroy and Thompson appear to confuse two different issues: the ability of various needle designs to provoke neural injury [2, 3] and the extent to which the provocation of paraesthesiae results in neurological symptoms after regional anaesthesia [4].

I wish to address the points raised in the editorial which concern needle design studies. I agree that there is a need for clinical data in this area, indeed the most recent article discussed this in detail [3]. However, in the absence of adequate clinical investigations, it is not acceptable for Moore, Mulroy and Thompson to dismiss the evidence from carefully conducted animal studies [2, 3] as not being of relevance, merely because it does not fit their own hypotheses. Furthermore, these two studies differ considerably in their fundamental aims and therefore it is fallacious of Moore, Mulroy and Thompson to dismiss them as a single entity. The first study examined the ability of intraneurally, but extrafascicularly, positioned needles to enter nerve fascicles [2]. In this respect, there is an advantage in the use of short-bevelled needles. In the second study [3], the needles were deliberately placed intrafascicularly in order to investigate the axonal consequences of accidental fascicular impalement; here there was an advantage in the use of long-bevelled needles and perhaps “pencil-point” needles [3, 5]. Moore, Mulroy and Thompson are clearly confused with respect to this important anatomical difference as they appear to believe that both of these studies investigated the consequences of intrafascicular penetration. Furthermore, the use of the emotive word “harpooning” to describe the techniques of lesioning sciatic nerve used in these two studies is not appropriate to a scientific review. Clinical studies, however challenging, need to be performed to confirm the optimal bevel design of regional anaesthesia needles and clinical recommendations cannot be made until such data are available. Nevertheless, such clinical studies need to be guided by the results of basic science studies [2, 3].

Furthermore, contrary to the beliefs of Moore, Mulroy and Thompson, it is very simple to differentiate between extra- and intrafascicular needle placement by using a sophisticated nerve stimulator. Much smaller currents are needed to stimulate axons from an intrafascicular, as opposed to extrafascicular, electrode. However, it should be noted that the majority of nerve stimulators sold as being suitable for regional anaesthesia are not capable of reliably operating within the required range.

In addition, the incidence of neuropathy symptoms after intrafascicular injury of axons is known from studies of microneurography (when percutaneous electrodes are deliberately inserted into human nerve fascicles to obtain single axon recordings) [5, 6]. During the experiments the majority of subjects reporting paraesthesiae as nerve fascicles are impaled and 10% of subjects experience persistent paraesthesiae for a few days after the experiment, but serious complications are rare [6]. The electrodes used in microneurography are slightly smaller than regional anaesthesia needles. However, it is known from animal studies that the needles used in regional anaesthesia produce a proportionately greater degree of axonal injury when fascicles are accidentally impaled [3, 5].

Prospective, randomized, clinical studies investigating both needle design and the role of nerve stimulators in preventing

regional anaesthesia-related neuropathies are required to complement existing animal data. However, the implication of the last sentence of Moore, Mulroy and Thompson “that we should refrain from publishing evidence from carefully conducted basic science studies until the clinical data are available, because medicolegal cases may be compromised” is clearly ludicrous.

A. S. C. RICE
Nuffield Department of Anaesthetics
John Radcliffe Hospital
Oxford

1. Moore D, Mulroy M, Thompson G. Peripheral nerve damage and regional anaesthesia. *British Journal of Anaesthesia* 1994; **73**: 435–436.
2. Selander D, Dhuner K-G, Lundborg G. Peripheral nerve injury due to injection needles used for regional anaesthesia. *Acta Anaesthesiologica Scandinavica* 1977; **21**: 182–188.
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Sir,—The editorial on peripheral nerve damage and regional anaesthesia by Moore, Mulroy and Thompson [1] is remarkable for several reasons. One expects a well designed and important message in an article with such a prominent position. Unfortunately, this editorial does not fulfil these expectations; instead it illustrates the authors’ rather old-fashioned views and limited respect for basic science.

In short, the editorial contains the following messages:

- (1) Animal data are of little value and should not be allowed to guide our clinical practice.
- (2) Clinical adverse events whose frequency does not reach statistical significance do not happen and therefore need not be considered in clinical practice.
- (3) Adverse events where patients are injured should not be reported in medical publications, as this could prejudice the outcome of court proceedings.
- (4) Eliciting paraesthesiae while performing neural block it not combined with any risk of neural injury.
- (5) The “axiom” of Moore, Mulroy and Thompson: “No paraesthesiae, no anaesthesia” is not outmoded.

These remarkable statements stand well for themselves, but some comments are needed.

(1) Animal experiments are often used in investigating mechanisms behind adverse effects of medical treatment. In many instances there are species-specific effects, for example because of enzymatic or immunological differences, which make direct application to humans uncertain. However, traumatic injuries and their consequences are less species-dependent, and nerve lesions caused by trauma by injection needles can be expected to be very similar between species. To disregard experimental results [2] from anaesthetic practice may prove deleterious for both patient and doctor.

(2) The lack of a statistically significant relationship between active paraesthesia and nerve lesions in the cited study [3] does not mean that this relationship does not exist. It is more likely to be a consequence of insufficient statistical power. The report by Plevak, Linstromberg and Danielson [4] arrived at the same values as Selander, Edshage and Wolff [3], but also did not reach statistical significance. In spite of this, the authors stated: “the higher incidence of neurological sequelae demonstrated in the PT (paraesthesia technique) group in both studies, allows us to conclude that paraesthesia should be avoided during axillary block.” This conclusion is supported by a meta-analysis, based on the results of both studies, in which the difference reached statistical significance ($P < 0.05$, Fisher’s exact test).

(3) It is indeed unfortunate that the medicolegal climate in the United States results in such suggestions. It would be unethical

not to inform our colleagues about possible risks and complications involved in medical treatment which may cause injury and suffering to our patients. Moore, Mulroy and Thompson, however, ignore their own recommendations when they publicize anecdotal information on six cases of neuropathy following the use of nerve stimulators in regional anaesthesia! [1].

(4) Paresthesia can be elicited in various ways. A tap on the "funny bone" that results in an ulnar paresthesia does not normally result in a neuropathy. Bonica wrote in 1954: "While it is true that repeated and rough probing of nerves may cause neurological sequelae, gently touching the nerve with the needle point does not cause any clinically apparent damage" [5]. I see no difficulty in accepting this statement, which agrees with our conclusion: "When performing a nerve block, paresthesiae should be elicited with the greatest care, or if possible avoided, in order to reduce the risk of nerve lesions" [3]. A means of minimizing needle trauma in peripheral nerve blocking is to use a short-bevelled needle which, with its very low risk of penetrating nerve fascicles, offers "safer" paresthesiae [2]. Neuropathies following the use of a nerve stimulator may be the result of misuse of the stimulator (too high energy level), an inability to differ between mechanically and electrically induced paresthesiae, or both.

(5) The "axiom" of Moore, Mulroy and Thompson has never been convincingly proven. Instead, there are several articles which do not show better success rates of axillary blocks performed with a paresthesia technique than without [3, 4, 6, 7].

D. SELANDER
Astra Pain Control AB
Södertälje, Sweden

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Sir,—Thank you for the opportunity to reply to the preceding correspondence which is *relative* to peripheral nerve damage and regional anaesthesia. Unfortunately, in attempting to defend their bench research, Rice and Selander have ignored the intent of the editorial. Insinuating that we were attempting to belittle their research and that of others is likewise "ludicrous" (Rice)—nothing could be further from the truth! Our own numerous investigations substantiate this statement.

Evidently, Rice agrees with the purpose of our editorial. He states, "Clinical studies, however challenging, need to be performed to confirm the optimal bevel design of regional anaesthesia needles and clinical recommendations cannot be made until such data are available," and "it is very simple to differentiate between extra- and intrafascicular needle placement, by using a sophisticated nerve stimulator."

To conclude, Rice indicates he has the "tools" (micro-neurography) so that a major, clinically statistically significant investigation could be conducted. Therefore, Rice and Selander should collaborate to prove or disapprove their theories as to whether long, short, or pencil-point needles, and/or the nerve stimulator will avoid or reduce to a minimum neuropathy when performing peripheral nerve block. Until then, the point made in the conclusion of the editorial is valid, that is: "until a prospective, blinded, major clinical study provides us with statistically significant *clinical* information, we believe that authors should not

draw conclusions relating to clinical practice and which may have significant medicolegal connotations."

D. C. MOORE
M. F. MULROY
G. E. THOMPSON
Department of Anesthesiology
Virginia Mason Medical Center
Seattle, WA, USA

A standard set of terms for critical incident recording?

Sir,—We were interested to read about the concept of critical incident reporting and the intention of constructing a critical incident register [1]. The authors take a critical look at describing the nature of such events and the construction of a suitable recording system. As we are working daily with a database for collection of drug side effects (adverse events), we wish to suggest some points which might be of assistance in this project.

The set-up of the monitoring system is influenced primarily by the intended use of the recorded information, for example is an overall safety evaluation the objective or is the information to be used for statistical analysis; is it sufficient to record only serious adverse events (critical incidents); is an assessment on a case-by-case basis necessary?

For evaluation of the safety profile of a drug, we use a computerized monitoring system where reported adverse events are attached to codes. The events are grouped in a hierarchical order and allocated to categories. One of the major advantages of working with a hierarchical computerized code system is the standardization of procedures and the consistency when dealing with a high number of case reports. Consistency and transparency of the recorded information are mandatory for the purpose of drug safety or any database of this type.

Reviewing a specific medical issue, the hierarchical structure offers the possibility of searching in a broad way, providing an overview and allowing a better assessment without losing cases reported with more detailed terms. For example, a higher term might be "block of the anaesthetic circuit" and lower terms would include "kinked tubing" and "sputum plug in the tracheal tube". This is especially important, as a reported incident is often described differently by different reporters. Also, our experience shows that reported terms are sometimes of little value compared with the actual description of what happened.

A crucial point is the method of reporting and the transformation of free text information of a case report into standardized terms and variables for a computerized system, to ensure the retrieval of the original medical history. For this we split the case report into parts—registration, source of information, summary log (what actually happened), patient, risk factors, other conditions of the patient, medication and dosage and event—and we established strict coding rules to be consistent in the way the case is entered onto the database.

In general we can say that the standardized hierarchical term glossary we use for reported adverse events is very comprehensive and seems to reflect physicians' needs. In addition, this list can be geared to the specialty of the group which uses it. In summary, a database is only as good as the information entered onto it.

M. STREB
S. REVELL
S. HILTON
F. Hoffmann-La Roche Ltd
Drug Safety
Basel, Switzerland

1. Banks IC, Tackley RM. A standard set of terms for critical incident recording? *British Journal of Anaesthesia* 1994; **73**: 703–708.

Sir,—Thank you for your comments following our article, which were well made. It is true that the intended use of the recorded information might affect the choice of terms in a critical incident

monitoring system. However, we were primarily trying to develop a set of terms that would both be compatible with the Read codes and also be of use for event recording. The Read codes are intended to be of sufficient detail, eventually, to be able to replace the written clinical record.

In order to statistically evaluate the incidence of any events or clinical details, there has to be some agreement or definition of the terms and how they are used. The Clinical Terms Project, which is responsible for co-ordinating the 1000 or so clinicians who are developing the terms, has nearly completed the collection of terms. The next stage will be to agree the context in which they are used (severity, certainty, timing, personnel involved, etc) and also to agree a few crucial definitions of how they are to be used. In particular, the hierarchies need to be as pure as possible. Thus although the terms are currently able to describe individual events, it will be possible in the future to use them for grouping and analysing multiple records.

In a minority of cases, in spite of the comprehensiveness of Read codes, it may still be necessary to record sections of free text to explain unusual or complex events. No practical terming system would aim to replace the English or any other language!

R. TACKLEY

I. BANKS

Anaesthetic Department
Torbay Hospital
Torquay

Hypothermia during liver transplantation

Sir,—Russell and Freeman [1] have clearly shown that during orthotopic liver transplantation, pulmonary artery temperature changes may be reduced by using a warm overblanket during surgery. In their introduction to the study, their hypothesis stated that they expected that active warming devices would reduce the risk of cardiac arrhythmia and the requirement for transfusion of blood and blood products. There was no difference noted between their groups in the amount of blood transfused, especially between the groups with minimal and maximal pulmonary artery temperature change. No information was given about blood products or cardiac arrhythmias.

There were temperature differences between the warming devices used. They stated in their methods that the electrical mattress was set at 39 °C. The group of patients anaesthetized on this mattress achieved the lowest pulmonary artery temperatures. The warm air mattress was set at 40 °C and achieved an intermediate result. The overblanket started from 42 to 48 °C for 45 min and could then reach 41.5 °C. This achieved the best result. It had the highest temperature difference from skin temperature. The results of these different temperatures on heat transfer to the patient were not discussed. A more fair comparison would have been to set them all at the same temperature.

Only one temperature was recorded in this study on which to base a comparison of active warming devices. The investigators acknowledged themselves that they could have underestimated hypothalamic temperature because the pulmonary artery was receiving cold blood. Other core temperatures would have been

useful to confirm their findings [2]. The investigators also mentioned that peripheral temperature is a component of temperature change, but did not measure it. These additional measurements could have calculated the heat loss, which may not have been as significant as the temperature changes measured in the pulmonary artery.

In summary, the investigators showed a lack of adverse effects of hypothermia, using active warming devices which were not comparable.

A. HOLDCROFT

Department of Anaesthesia
Hammersmith Hospital
London

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2. Imrie MM, Hall GM. Body temperature and anaesthesia. *British Journal of Anaesthesia* 1990; 64: 346–354.

Sir,—The aim of the study was to compare the ability of the three devices to prevent hypothermia during liver transplantation. There was no attempt made to compare the cardiac or haematological effects of the hypothermia as these have already been studied (references [2–6] in our article). We specifically excluded patients who were expected to lose large amounts of blood in order to standardize the groups, as explained in the text.

We acknowledged in the text that there were differences between the devices, both with respect to the temperature achieved and the power input. As explained in the article, the devices were used at the manufacturer's recommended settings. There may be good reasons, for example the risk of burns in relatively ischaemic tissues, why higher temperatures are not recommended for the electrical mattress and the warm air under mattress. There seems little logic in using the devices at less than maximum effectiveness at these are the settings at which one would presumably use them in normal practice. The aim was to compare the ability of the devices to maintain normothermia, and if the overblanket can do so by safely achieving a higher local ambient temperature, then this is a real advantage of the device.

Only one temperature was measured, that of the pulmonary artery, for reasons explained in the text. The most clinically troublesome effect is that of cardiac hypothermia at reperfusion; the hypothalamic temperature, while useful in explaining the physiology of temperature regulation, is not of particular clinical importance in this setting.

In summary, our article made no attempt to show the adverse effects of hypothermia, but did demonstrate a significant difference in the ability of these devices to prevent hypothermia during orthotopic liver transplantation, when used at the manufacturer's recommended settings.

S. H. RUSSELL

J. W. FREEMAN

Featherstone Department of Anaesthesia and Intensive Care
Queen Elizabeth Hospital
Birmingham