Portable ultrasound for central venous cannulation

Editor,—We read with interest the article by Hatfield and Bodenham together with the accompanying editorial encouraging the use of ultrasound-guided central venous cannulation. At our hospital we have found this device of particular use as a teaching tool, especially when central venous lines are sited in awake patients with renal or haematological disease. EMLA cream has been shown to be effective for central venous cannulation in a recent study. We have found that the use of EMLA applied 40 min before ultrasound-guided insertion provides: (i) adequate topical anaesthesia; (ii) no distortion of the anatomy; and (iii) a good interface between probe and skin similar to ultrasound gels.

The trend towards using ultrasound-guided techniques is gaining momentum in the USA with the publication of similar work. We would advocate the use of EMLA in conjunction with ultrasound in non-anaesthetized patients and in the intensive care unit where line insertion is sometimes associated with unpleasant memories. This combination improves the chances of successful cannulation while minimizing patient discomfort.

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Induction of anaesthesia in morbidly obese patients

Editor,—I welcome the advice given by Aono and colleagues for induction of anaesthesia in morbidly obese patients. I would like to comment on several points.

While the lateral position they describe may certainly be of benefit for performing inhalation induction of anaesthesia, it is significantly more difficult to achieve an adequate seal with a face mask because there is no occipital support to apply counter pressure to the head. If for any reason the patient becomes apnoeic, artificial ventilation of the lungs becomes a challenge.

As they rightly point out, it is not uncommon for morbidly obese patients to suffer from some degree of gastro-oesophageal reflux that places them at risk of aspiration during anaesthesia. Aside from awake intubation, rapid sequence i.v. induction must still be the preferred method of securing and protecting the airway in these patients. In addition, to resort to an unfamiliar method of intubation in such a high-risk group surely has implications in itself.

Would it not be easier to perform tracheal intubation in the left lateral decubitus position? The tongue automatically falls to the side requiring only tongue ‘lift’ to see the cords. In the right decubitus position, it is certainly more difficult to move the tongue out of the field of view because there is a tendency for it to slip off the laryngoscope blade.

Finally, is the patient’s airway really protected from inhalation of vomit in the lateral position? I do not believe that this is the case and to perform inhalation induction of anaesthesia in these patients surely places them at a greater risk of aspiration. I hope that adopting the lateral position does not lead to a false sense of security.

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Editor,—EMLA cream does work as an ultrasonic coupling medium, but it is not sterile and it is not a disinfectant. If I were having a central line inserted, I would prefer to have the cream removed and my skin cleaned with disinfectant before skin puncture. I cannot endorse central line insertion through a layer of EMLA cream as I believe it is an infection risk.

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whether in the left or right decubitus position, is not a major issue. In both positions, it is not difficult to see the cords using a laryngoscope blade to move the tongue away from the field of view.

We cannot say that the airway is really protected from aspiration in the lateral position. However, in our experience of emergencies such as postoperative bleeding after tonsillectomy, rupture of oesophageal varices, etc, inhalation induction of anaesthesia in the lateral position has been performed safely without evidence of inhalation of vomit. In such cases, we apply a slight head-down position by removing the headrest or pillow in addition to using the lateral position.

Induction of anaesthesia in the lateral decubitus position is probably an unfamiliar method to most anaesthetists. We wonder if Dr Cupitt’s concerns arise from this unfamiliarity. We certainly agree that it is dangerous to use a technique which is unfamiliar in a high-risk patient. Thus we recommend that familiarity with this induction technique is obtained first in healthy patients. After experience with several cases, even a new trainee can easily perform this technique.

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Oxygen concentration distinguishes pulmonary from bowel gas leak

Editor,—A 52-yr-old male with ankylosing spondylitis underwent artificial ventilation in the intensive care unit (ICU) for right-sided lower lobe pneumonia. Bronchoscopy had been normal but a chest radiograph suggested fluid collection at the right base of the lung. Ultrasonic examination of the chest was performed on day 4 after ICU admission to exclude a right empyema: it revealed a large subdiaphragmatic collection. Insertion of a pig-tail catheter under ultrasound guidance into the collection failed to drain any fluid; the procedure was repeated under CT guidance with drainage of 500 ml of pus. A repeat CT scan 4 days later showed that a considerable amount of fluid remained in the abscess cavity. A larger drain was inserted and connected to an underwater seal. Shortly after this procedure, a moderate air leak developed from the new drain which was bubbling and swinging, although not in time with ventilation.

The source of the leak was unclear. A chest X-ray (Fig. 1) showed a pocket of air but it was not clear if this was intra-abdominal or intrathoracic. An air leak from the lung would require a definitive chest drain, in case the existing drain became occluded with pus and debris. An abdominal source would suggest a perforated viscus and would require immediate laparotomy. It was therefore imperative to determine the source of the air leak.

An oxygen analyser was attached to the air outlet of the chest drain bottle and $F_{O_2}$ was increased from 0.40 to 1.00. The oxygen percentage of the gas from the chest drain increased immediately to 93% and we concluded that this was a pulmonary air leak. A chest drain was inserted.

The patient died 10 days later from multiple organ failure. At post-mortem examination, a large subpulmonary cavity was found above the diaphragm. We believe that this novel method of distinguishing a pulmonary from a bowel gas leak has not been reported previously.

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Transient ischaemic attack during deep cervical plexus block

Editor,—I was interested to read the review article on regional anaesthesia for carotid endarterectomy and would like to solicit the authors’ views on a serious complication which occurred during deep cervical plexus block in our unit. A 49-yr-old, 51-kg woman was scheduled for elective carotid endarterectomy after two episodes of amaurosis fugax; Doppler ultrasound indicated an 80% left-sided
stenosis. She was premedicated with lorazepam 1 mg on
the morning of surgery and noted to have an arterial pressure
of 190/100 mm Hg in the anaesthetic room, having been
normotensive at the time of preoperative evaluation. Deep
cervical plexus block was performed with 0.375% bupivacaine 20 ml in divided doses at C3, C4 and C5,
using an immobile needle technique. Paraesthesiae were
obtained over the cutaneous distribution of C3 and C4
before injection, and aspiration tests were negative. After
the third injection was complete, the patient began to
complain of slurred speech and drowsiness. She became
irritable with a short period of restless muscle activity
which was epileptiform in nature and progressed over
2–3 min to a deep coma with a Glasgow coma score of
3/15. Spontaneous respiration was maintained at all times
and 100% oxygen was administered by Waters circuit and
face mask. Arterial pressure increased to 230/130 mm Hg
during this episode, responding to a bolus dose of esmolol
1 mg kg\(^{-1}\). After 25 min of airway support, the patient
took to awake and within a few minutes had made a
complete recovery, with no residual neurological deficit.
Surgery was cancelled.

The patient was subsequently started on antihypertensive
therapy and underwent surgery 4 weeks later under general
anaesthesia. In the intervening time, he reported three
episodes of transient dysarthria which resolved
spontaneously. A shunt was used electively during cross-
clamping and her postoperative recovery was unremarkable.

It was unclear at the time whether the episode of
unconsciousness was caused by vertebral artery or
subarachnoid injection undetected by aspiration, or whether
this was a transient ischaemic attack which was embolic in
nature. In the light of the patient’s subsequent intermittent
dysarthria, I would suggest the latter is the most likely
explanation, although I recognize that intravascular
absorption undetected by aspiration can occur. Although
care was taken not to apply pressure over the carotid artery
during placement of the block, it is possible that this volume
of injectate placed in close proximity to it may have
worsened the carotid occlusion. A volume of 20 ml in a
thin woman may have been excessive. I would welcome
the views of Drs Stoneham and Knighton.

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1 Stoneham MD, Knighton JD. Regional anaesthesia for carotid

Editor, —Thank you for the opportunity to reply to the
further correspondence about our review article on regional
anaesthesia for carotid endarterectomy. We agree with
Dr Johnson that this is a most interesting case, the cause
of which is not immediately obvious. We agree that the
differential diagnosis includes: intravascular injection
(particularly vertebral artery); transient ischaemic attack
(TIA); carotid artery compression by the local anaesthetic;
and local anaesthetic overdose. However, subarachnoid
injection seems a most unlikely cause as the duration of
unconsciousness was so short. Local anaesthetic over-
dose is also unlikely as the patient only received
0.375% bupivacaine 20 ml (bupivacaine 75 mg) which is
1.47 mg kg\(^{-1}\).

Dr Johnson may be right in his supposition that TIA was
the cause of this event, particularly because of the similarity
between the initial transient dysarthria during the event and
the subsequent TIA presenting in the same way. If this is
so, the TIA may even have been unrelated to the deep
cervical plexus block. However, intravascular injection of
local anaesthetic is difficult to rule out as a primary cause.
Deep cervical plexus block has an excellent safety record
although it has many potential side effects. However,
intravascular placement of a block needle is relatively
common, occurring in up to 30% of patients, which is
why careful aspiration must always be undertaken before
injection. One of us (M. D. S.) has experience of a patient
undergoing carotid endarterectomy who suffered a sudden
coma and an epileptic seizure lasting just 30 s after
accidental injection of as little as 5 mg of lidocaine into
the internal carotid artery by the surgeon during operation.
The time course and description of the two events are
similar although the duration of unconsciousness in this
case is longer than one might expect.

There is a third possibility. If 0.375% bupivacaine 20 ml
were injected in close proximity to the vertebral artery,
rapid systemic absorption could occur, transiently leading
to very high local anaesthetic concentrations in the
vertebral artery which could cause cerebral local anaesthetic
 toxicity. This could explain the cerebral irritability and
unconsciousness. Plasma concentrations of bupivacaine
would not be increased because of redistribution, so there
is no real way to prove or disprove this.

Although the patient described by Dr Johnson did not
apparently have a grand mal seizure, she displayed restless
muscle activity. If she had displayed any other stigmata of
seizure, such as associated loss of continence or tongue-
biting, then this would obviously make intravascular
injection or absorption of local anaesthesia a more likely
cause.

Finally, we are also interested to know whether the
decision to proceed with the operation under general
anaesthesia was made by the patient, the surgeon or the
anaesthetist, as whatever the cause of this event, it would
not appear to contraindicate regional anaesthesia on a later
occasion.

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The value of \(\Delta Down\) during haemorrhage

Editor,—\(\Delta Down\), which is the decrease in systolic arterial pressure (SAP) with each mechanical breath, is a sensitive indicator of preload and has been shown repeatedly to be a sensitive indicator of preload both experimentally and clinically. Recently, in septic patients undergoing artificial ventilation, \(\Delta Down\) predicted the response of cardiac output to volume load better than pulmonary capillary wedge pressure (PCWP) and the echocardiographic left ventricular end-diastolic volume (LVEDa). However, Dalibon and colleagues found that \(\Delta Down\) was not better than mean arterial pressure (MAP) in reflecting the amount of blood loss during graded haemorrhage and retransfusion in pigs. I would like to argue that the apparent discrepancy as to the clinical value of \(\Delta Down\) stems from some problems in the methodology of the work of Dalibon and colleagues.

The authors compared changes in MAP, PCWP, LVEDa and \(\Delta Down\) during incremental withdrawal and retransfusion of blood in anaesthetized pigs undergoing artificial ventilation. They used the amount of shed–retransfused blood as an independent variable and constructed curves for the bleeding and retransfusion procedures for each variable. The authors judged the efficacy of each of the variables by: (a) regression coefficient; (b) area between the withdrawal and retransfusion curves (hysteresis); (c) minimal amount of blood loss which caused significant change compared with control during blood withdrawal; and (d) maximal blood loss which induced no change compared with control in the retransfusion phase. All variables changed significantly during bleeding, but analysis of the data led the authors to conclude that: LVEDa was an accurate variable of blood volume; \(\Delta Down\) did not add important information compared with MAP; and PCWP was the most reliable variable to avoid volume overload.

The authors assume that the effective intravascular volume during haemorrhage is dependent only on the amount of shed–retransfused blood which they regard as a ‘gold standard’ for changes in preload. This assumption disregards the well known physiological effects of severe haemorrhage, which include marked vasoconstriction, spleen contraction, possible ingress of extravascular fluid, decreased contractility, etc. Thus during retransfusion, MAP, PCWP and LVEDa were greater, and \(\Delta Down\) smaller, for each corresponding point during blood withdrawal, suggesting a much more contracted intravascular bed which may directly and significantly affect the effective intravascular blood volume. This physiological response explains the observed hysteresis between the bleeding and retransfusion curves. The authors’ definition of the ideal variable as one that changes linearly and with the smallest hysteresis with shed blood does not fit our understanding of the physiology of severe haemorrhage. It would have been preferable to examine the relationships of \(\Delta Down\) and PCWP to LVEDa.

I would also like to address the value of \(\Delta Down\). \(\Delta Down\) increased from about 1 to 10–11 mm Hg during haemorrhage and had the highest linear regression slope during the withdrawal part of the experiment. However, the author found it to be less accurate than MAP in assessing blood volume loss. Such comparison is highly dependent on the standard deviation of each variable at each point in time. As the authors adjusted tidal volume to obtain the ‘required \(PE_{CO_2}\)’ and as tidal volume is one of the determinants of \(\Delta Down\), this variability in tidal volume may have affected the results. In addition, mechanical ventilation was stopped for 30 s to estimate SAP during apnoea, in order to measure \(\Delta Down\). Such a long period of apnoea is unnecessary and may introduce an error in the measurement of \(\Delta Down\). We have recommended a period of apnoea of 5–10 s which is more than enough to obtain a stable SAP.

Previous studies have repeatedly recommended that \(\Delta Down\) should be expressed as a percentage of systolic pressure during apnoea (%\(\Delta Down\)), especially in the presence of significant changes in arterial pressure. Expressing \(\Delta Down\) in percent may have made this variable more significantly different than control values during both the withdrawal and retransfusion stages. Examining Figures 2 and 3 in Dalibon and colleagues’ article, we estimate that %\(\Delta Down\) increased from 1–2% of SAP during baseline to about 25% at maximal bleeding. Thus if the authors had used %\(\Delta Down\) they would have found that \(\Delta Down\) changed not 10-fold (between 1 and 10 mm Hg), but rather 25-fold (between 1% and 25%), increasing the sensitivity and correlation of this variable to blood loss.

The design of this study and the way in which the data were analysed led the authors to repeatedly claim that \(\Delta Down\) did not add important information in addition to MAP. Although it is obvious that when arterial pressure decreased to a mean of 31 mm Hg, the absolute value of \(\Delta Down\) had ‘no additional important value’, expressing \(\Delta Down\) in per cent may have completely changed the results of this study. In addition, the strength of \(\Delta Down\) lies in its ability to detect occult hypovolaemia (i.e. a decrease in blood pressure during apnoea of 5–10 s) which was measured but, surprisingly, not reported and not included in the analysis.

volume that is not accompanied by severe hypotension). Moreover, this variable is a dynamic variable of volume responsiveness, reflecting in a minimally invasive way the slope of the LV function curve.9

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1 Perel A, Pizov R, Cotev S. Systolic blood pressure variation is a sensitive indicator of hypovolemia in ventilated dogs subjected to graded hemorrhage. Anesthesiology 1987; 67: 498–502


9 Perel A. Assessing fluid responsiveness by the systolic pressure variation in mechanically ventilated patients. Anesthesiology 1998; 89: 1309–10

Editor,—First, we agree that we did not measure the effective intravascular volume in our study but this limitation was clearly cited in our article. Measurement of intravascular blood volume requires steady-state conditions which did not exist in our experimental conditions that mimicked a haemorrhage–resuscitation scenario. The lack of significant changes in packed cell volume and left ventricular end-diastolic area (LVEDa) during the whole procedure strongly suggests that significant fluid shifts and/or spleen release of blood cells did not occur, and thus that blood volume loss could be considered as a good approximate to intravascular blood loss. In any event, this point cannot explain the important hysteresis observed with ∆Down compared with the ‘gold standard’ LVEDa or even mean arterial pressure.

Second, we do not think that the tidal volume chosen in these animals could have influenced our results. Despite the fact that tidal volume was adjusted to obtain an end-tidal carbon dioxide value of 4.2–4.8 kPa, tidal volume did not differ greatly between animals (mean 15 (SD 2) ml kg⁻¹).

Third, ventilation was stopped for 30 s to allow measurement of cardiac output, but measurement of ∆Down was performed within 10 s, as recommended previously by Perel and his colleagues.

We agree with Perel that expression of ∆Down as a percentage of systolic arterial pressure (SAP) is probably preferable. If we had used this expression, we agree that the correlation between blood loss and ∆Down would have been improved during the haemorrhagic part of our experiment. However, it should be emphasized that expression of ∆Down as a percentage of SAP did not significantly modify the poor correlation with blood loss and the hysteresis observed during the resuscitation phase (Fig. 1). We must conclude that in our experimental conditions, ∆Down was unable to detect ‘occult hypovolaemia’, at least during the resuscitation phase.

We would like to emphasize that our study had many limitations that were discussed in our article. The results may apply only to pigs and not to humans. But it is important to consider that the pathophysiology of haemorrhage and haemorrhagic shock is complex and that ∆Down does not depend on preload alone. The main result of our study was that important differences may occur between the haemorrhagic and resuscitation phases. Thus further clinical research efforts are mandatory before ∆Down can be applied as a routine variable to monitor blood volume in our patients.

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Subconjunctival block for cataract extraction and keratoplasty

Editor,—A survey of British ophthalmologists has shown that only 7% of consultants usually perform penetrating keratoplasty under local anaesthesia.¹ The two most commonly used local anaesthetic techniques for ophthalmic surgery in the UK are retrobulbar and peribulbar injections but both may produce potentially serious complications.² These include perforation of the globe, intravascular injection, retrobulbar haemorrhage, direct damage to the optic nerve and puncture of the optic nerve sheath resulting in injection into the subarachnoid space. An alternative local anaesthetic technique which avoids the potential complications associated with retrobulbar and peribulbar injection is subconjunctival block. This involves administration of a topical anaesthetic followed by a small volume injection of local anaesthetic with epinephrine beneath the superior conjunctiva using a 27-gauge needle.

There is increasing use of local anaesthetic techniques for cataract surgery and it has been demonstrated that anterior segment surgery can be performed using subconjunctival block with a single injection at the 12 o’clock position.³–⁵ This is the technique of choice with anaesthetists for cataract surgery in our department with more than 80% of cataract extractions being performed satisfactorily using this method. We have recently extended the use of subconjunctival block to enable corneal graft surgery to be performed using this technique.

Between September 1997 and April 1999 there were 25 keratoplasties, both lamellar and penetrating, performed in our department. Eleven of these were performed under general anaesthesia and 14 during combined topical and subconjunctival block. General anaesthetics were administered if the patient declined or was deemed unsuitable for a local technique. Venous access was obtained so that sedation could be given if required. Of the 14 cases performed during subconjunctival block, seven required i.v. sedation with a small dose of midazolam. Topical anaesthesia was achieved with 5–6 drops of 0.4% oxybuprocaine. Four subconjunctival injections were given, each approximately 0.1–0.2 ml of 2% lidocaine with epinephrine 1 in 200 000 at the 2, 4, 8 and 10 o’clock positions, approximately 5 mm from the limbus. Surgery was commenced 5 min after administration of the block.

This technique does not provide an immobile eye and requires some patient cooperation following careful preoperative assessment. Of the 14 cases, there were no problems with excessive eye movement which required discontinuation of the procedure, and there were no cases of increased intraocular pressure. After operation, one of the 14 patients required paracetamol for minor discomfort. All patients remained well and were discharged later the same day. They all returned to the ophthalmic outpatient department the following morning where the grafted eye was examined and any problems with the graft or postoperative discomfort was assessed and treated as required.

This technique is technically simple and quick to perform, and avoids the potentially serious complications of peribulbar and retrobulbar injections. It provides good operating conditions and there have been no significant problems with excessive eye movement or increased intraocular pressure. Although we have studied only a small number of cases, it appears that this technique provides good anaesthesia and operating conditions with minimal risk of complications and may be a significant step forward in the anaesthetic management of patients for corneal graft surgery.

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⁵ Redmond RM, Dallas NL. Extracapsular cataract extraction under local anaesthesia without retrobulbar injection. Br J Ophthalmol 1990; 74: 203–4

Low-dose clonidine infusion during labour

Editor,—We read with interest the article by Tremlett and colleagues on the use of epidural bupivacaine and clonidine for analgesia in labour.¹ However, we are concerned that their conclusions regarding the condition of the baby are not supported by the data presented. They stated that there were no adverse effects on the baby based on umbilical arterial and venous pH values and a single neurologic and adaptive capacity score (NACS) performed 4 h after delivery.

Originally described by Amiel-Tison and colleagues,² NACS is a neonatal neurobehavioural assessment based on 20 criteria, each scored from 0 to 2. Assessments are made 15 min after birth and repeated at 2 and 24 h. A score of 35–40 indicates a neurologically vigorous infant while those of 34 or less represent possible problems. The test is designed to distinguish neonatal depression caused by maternal drug administration from that resulting from birth trauma or perinatal asphyxia. Amiel-Tison and colleagues have suggested that drug effects produce low scores immediately after birth that usually recover by 24 h. Conversely, low scores resulting from trauma or asphyxia take several days to return to normal.³ More recently, investigators have chosen to omit the 15-min assessment, relying on values obtained at 2 and 24 h.
In the study by Tremlett and colleagues, mothers were allocated randomly to receive an epidural infusion of 0.03% plain bupivacaine, 0.03% bupivacaine with clonidine 19 μg h⁻¹, or 0.03% bupivacaine with clonidine 37 μg h⁻¹, with mean values for NACS 4 h after delivery of 26.0, 27.7 and 25.4, respectively. By presenting only one value it is impossible to differentiate drug effects from those resulting from trauma. Furthermore, although we are not told how many babies scored less than 35, with such low mean scores it is likely that many were disturbingly low, making the authors' claims that no adverse outcome occurred invalid.

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Editor,—Thank you for the opportunity to reply to Drs Russell and Rutter. They ask if we are correct in concluding that there were no adverse effects on the baby from using an epidural infusion of bupivacaine and clonidine. We assessed the condition of the babies using the neonatal neurologic and adaptive capacity score (NACS), first described by Amiel-Tison and colleagues.1 We were interested to observe whether the infants of mothers who had received clonidine were more sedated than our control (bupivacaine alone) group. Although these scores are usually undertaken at two separate times to differentiate drug effects from trauma, we undertook a single early assessment, and did not undertake a late (24 h) score as we felt it unlikely that the addition of clonidine would lead to increased birth trauma. A trend to low early scores would suggest (but not prove conclusively) drug sedative effects.

Beforecommencing the study, we practised undertaking this examination on several newborn infants and also undertook a pilot study. We found two areas of difficulty. First, it was difficult to undertake the examinations at 2 h after delivery; most mothers had been through long and difficult labours, often involving medical intervention and had undergone repeated assessments for this study. Two hours after delivery was also a time when parents wanted to be alone with their newborn child, and thus we delayed the NACS score until 4 h after delivery in the interests of our subjects. Second, despite being used to performingneonatal developmental assessments, we found the NACS score difficult to undertake. In particular, we found that the five tests of adaptive capacity did not produce consistent, reproducible results. This is perhaps hinted at in the original article by Amiel-Tison and colleagues when they state that with these elements of the test ‘the maximal response may not be elicited easily.’ Therefore, our final scores exclude the adaptive capacity scores (total of 30 not 40). We should have stated this in our article. Despite these difficulties, NACS scores in the clonidine groups at this relatively early (4 h) time did not differ from the control group. An easier and more relevant measure of drug sedation is needed, and a simple clinical measure, such as time to first effective feed, would be interesting.

We continue to believe that no adverse effects caused by addition of clonidine were demonstrated, but in view of the small numbers involved and the fetal trace assessment, further work is warranted.

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Efficacy and safety of the superficial cervical plexus block for carotid endarterectomy

Editor,—We recognize Stoneham and Knighton’s important contribution to the promotion of regional anaesthesia for carotid endarterectomy but their assertion that ‘superficial block is rarely used alone for carotid surgery’1 is not supported by data from the USA. Local audit data confirm that 40% of all regional blocks performed at the University of Michigan Medical Center for this procedure involve superficial block alone, which also appears to be the procedure of choice at a number of other centers.2 We are conducting a questionnaire study to document the current practice of this procedure in North America.

There have been only two randomized controlled studies comparing superficial cervical plexus block with deep or combined block.3,4 Both studies showed that the two blocks were equally effective. The earlier suggestion that the deep block provides better postoperative analgesia3 was not substantiated4 and in fact it appears that analgesia is better after superficial block alone.4

It is generally accepted that the superficial block is easier to perform, easier to teach and associated with fewer complications than the deep block.1,5 In contrast with deep block,5 there does not appear to be any report in the
literature of a serious complication resulting from superficial block. This should make us all more cautious about practising the alternative, more risky technique which has no added benefit for the patient.

We note the comment of Stoneham and Knighton that a prospective, randomized, controlled comparison of regional compared with general anaesthesia is about to start in the UK.\(^1\) In the light of published reports, consideration should be given to this study being a comparison of general anaesthesia with superficial block, rather than with deep block. One of the outcome measures of a study is likely to be the complications of the anaesthetic technique. If this study were to compare the deep (or combined) block with general anaesthesia, a result less favourable to regional anaesthesia might well result. This potentially inherent bias might be avoided by studying the superficial block.

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1 Stoneham MD, Knighton JD. Regional anaesthesia for carotid endarterectomy. Br J Anaesth 1999; 82: 910–19
2 Lee KS, Davis CH, McWhorter JM. Low morbidity and mortality of carotid endarterectomy performed with regional anaesthesia. J Neurosurg 1998; 89: 483–7
3 Stoneham MD, Doyle AR, Knighton JD, Dorgie P, Stanley JC. Deep or superficial cervical plexus block for carotid endarterectomy surgery. Anesthesiology 1998; 89: 907–12
4 Pandit JJ, McLaren ID, Crider B. Superficial or combined deep and superficial cervical plexus block for carotid endarterectomy. Anesth Analg 1999; 88: 596

Third, when we were working at the University of Michigan Medical Center, the majority of vascular anaesthetists were using the combined block for carotid surgery. If, from their “local audit data”, 40% of regional blocks at the University of Michigan Medical Center were superficial, then presumably 60% (i.e. the majority) were deep blocks. We are delighted that as our randomized comparison of deep and superficial block showed no difference between the two in terms of additional local anaesthetic supplementation, superficial block alone has become more popular at that institution. However, anecdotal evidence would suggest that this has not occurred elsewhere in the USA.

We also take issue with the statement that superficial block is superior to deep or combined cervical plexus blocks. In contrast, our randomized comparison of deep and superficial blocks\(^3\) showed that, if paraesthesia was elicited during placement of the deep block, this made the deep block more effective. This has been confirmed by others using a nerve stimulator\(^2\) which is now the authors’ preferred technique for placement of the deep block.

The study of Pandit, McLaren and Crider comparing superficial block with combined deep and superficial block\(^3\) apparently showed better analgesia in the superficial group. However, in the superficial group more local anaesthetic was used (superficial group mean 116 (SEM 3) mg, combined group 93 (10) mg), pain scores were higher (4.3 (2.4) vs 3.4 (0.6)) and more fentanyl was administered during operation (54 (14) µg vs 34 (14) µg, although these differences did not achieve significance in their small study (n=10 in each group). It seems plausible that using more local anaesthetic for the block and more fentanyl could reduce analgesic requirements after operation.

We believe that while there are theoretically more complications associated with deep block, in clinical practice this has not been shown to be the case. The case report cited by Pandit in support of the dangers associated with deep block was in fact a situation where a superficial cervical plexus block was not appropriate.\(^4\) The patient had undergone previous cervical irradiation, resulting in deep tethering of the skin of the neck and loss of tissue planes. Subcutaneous injection of local anaesthetic was thus not possible, nor could the external jugular vein be identified, and hence superficial block was contraindicated. A deep cervical plexus block was performed successfully using anatomical landmarks. The block, although very successful, unfortunately resulted in transient respiratory impairment as a result of pre-existing contralateral hemidiaphragmatic paralysis. However, this was managed conservatively and the operation was completed successfully with no local anaesthetic supplementation required.

From the small number of patients studied by Pandit and ourselves, it is not possible to make statements about the safety of deep and superficial cervical plexus blocks as neither study set out to investigate side effects. Perhaps a large prospective study such as the GALA trial of general vs regional anaesthesia underway in the UK may yet show

Editor,—Thank you for the opportunity to reply to Drs Pandit, McLaren and Crider. Several factors contributed towards our making the statement that superficial block is not often used alone for carotid surgery. First, an extensive review of the literature revealed very few reports of superficial cervical plexus block being used alone for carotid endarterectomy. In contrast, in most a combination of deep and superficial plexus block or the deep block alone was used. Second, in a recent survey of members of the Vascular Anaesthetic Society of Great Britain and Ireland (to be published shortly), of 77 anaesthetists currently providing regional anaesthesia for carotid endarterectomy, most (71%) used a combination of deep and superficial blocks with a minority using superficial block alone or local infiltration.
complications resulting from deep block. However, until the evidence is available, we believe that the anaesthetist should use either or both blocks depending on the clinical situation, operator experience and individual preference.

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**Videoendoscopic parathyroidectomy**

Editor,—Because of developments in videoendoscopic surgery, the concept of ‘surgery in closed spaces’ has become popular. After surgery in the retroperitoneal space, videoendoscopic techniques have become available for parathyroid surgery. Removal of the pathological gland or glands can be achieved either by a gasless video-assisted endoscopic technique or by creating an artificial space with carbon dioxide insufflation.1 Massive carbon dioxide diffusion and absorption have been reported during the latter procedure.2

We have recently managed two patients who were treated successfully with videoendoscopic surgery for primary hyperparathyroidism. The diagnosis was made by laboratory tests in a 63-yr-old female and a 19-yr-old female. For preoperative localization and confirmation of the diagnosis, ultrasonography, magnetic resonance imaging and sestamibi scintigraphy were used. General anaesthesia was induced in both patients with fentanyl, propofol and atracurium and maintained with sevoflurane and 50% oxygen in nitrogen. ECG, $E_CO_2$, $S_PO_2$ and invasive arterial pressure were monitored continuously during surgery. The surgical approach was through a 1.5-cm incision between the jugular incisure of the sternum and the thyroid isthmus. Cutaneous and subcutaneous tissues were dissected. The sternothyroid and sternohyoid muscles were retracted. A Foley catheter was inflated beneath the muscles using a 5-ml saline solution, thus creating a potential space. By using two Verres needles that were inserted from the medial and caudal aspects of the incision, two 2-mm trochars were placed. A carbon dioxide pressure of 5 mm Hg was obtained through a 10-mm trochar (Fig. 2). In both patients, under direct vision, the right lower parathyroid adenomata were found and excised totally within their capsules. Intraoperative measurement of parathyroid hormone (PTH) showed a decrease of 80% from initial plasma concentrations in both patients. Frozen section examination revealed hypercellular parathyroid tissue. Both patients were discharged from hospital on the first day after operation. Minimal scarring, decreased need for postoperative analgesia, early mobilization, decreased duration of hospitalization and early return to work are the major advantages of this technique. In appropriate patients, videoendoscopic parathyroidectomy can be an alternative to conventional parathyroidectomy. The surgeon and anaesthetist should evaluate each patient individually before making a decision on the preferred surgical technique.

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**Magnesium: physiology and pharmacology**

Editor,—We would like to commend Drs Fawcett, Haxby and Male1 for their excellent and comprehensive review of the role of magnesium. They highlighted the problems with

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1 Stoneham MD, Doyle AR, Knighton JD, Dorje P, Stanley JC. Prospective, randomised comparison of deep or superficial cervical plexus block for carotid endarterectomy surgery. *Anesthesiology* 1998; 89: 907–12
3 Pandit JJ, McLaren ID, Crider B. Superficial or combined deep and superficial cervical plexus block for carotid endarterectomy. *Anesth Analg* 1999; 88: S96–3
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Fig 2 One 10-mm and two 2-mm trochars are inserted for operative manipulation.
measurement of serum magnesium and the factors affecting its interpretation. They emphasized that a better measurement is intracellular ionized magnesium, which is limited by both expense and availability. Atomic absorption spectrophotometry is a reliable method with which to measure lymphocyte magnesium concentrations and the latter has been shown to correlate with myocardial concentrations in patients undergoing cardiac surgery. In their review, Fawcett, Haxby and Male suggest that there is 'no evidence that isolated hypomagnesaemia is proarrhythmic or that myocardial magnesium depletion precipitates arrhythmias'. A recent study by Zuccala and colleagues examined changes in serum and lymphocyte cellular magnesium in 33 elderly patients undergoing surgery for hip fracture repair under general anaesthesia and its association with the development of ventricular arrhythmias. They found that magnesium depletion was the major determinant of the postoperative increase in the severity of arrhythmias, even after adjustment for potassium and calcium serum concentrations. The most significant predictor was a decrease in cellular magnesium greater than 6 nmol mg⁻¹. This value corresponded to a change in serum magnesium concentration of 0.125 nmol litre⁻¹, calculated from the equation given below. It should be emphasized that absolute serum and cellular magnesium concentrations did not correlate and neither value was a predictor of arrhythmia.

\[ \Delta_{\text{cell}} \text{Mg} = 1.59 + (\Delta_{\text{serum}} \text{Mg} \times 35.26) \]

where \( \Delta_{\text{cell}} \text{Mg} \) = perioperative difference in lymphocyte magnesium (nmol mg⁻¹) and \( \Delta_{\text{serum}} \text{Mg} \) = difference in serum magnesium (nmol litre⁻¹).

This small observational study suggests that hypomagnesaemia is proarrhythmic, even in the presence of a normal serum potassium concentration. Use of this simple calculation may help identify patients at risk of developing arrhythmias in the postoperative period when only the serum magnesium value is available. The significance of therapeutic intervention on morbidity and mortality in the elderly surgical population, such as those with hip fractures, is as yet unanswered. We await the outcome of future clinical studies with interest.

D. MacLeod
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Editor.—The area of hypomagnesaemia, its measurement and aetiology in the genesis of cardiac arrhythmias is a complex and largely unresolved issue. In their letters, Drs MacLeod used myocardial magnesium concentrations from one group of patients (those undergoing cardiac surgery) and extrapolate these findings to another group of patients (elderly, emergency orthopaedic patients) in whom myocardial magnesium concentrations were not measured. In this latter group, absolute magnesium concentrations did not serve as a predictor for arrhythmias but only for a decrease in lymphocyte cellular and serum magnesium. None of this evidence contradicts or adds to the review article. I agree, however, that further large studies are required if this complex area is to be addressed with any degree of certainty.

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Itching after use of starch solutions

Editor.—We read with interest the article by French and colleagues describing the prevention of hypotension during spinal anaesthesia for elective Caesarean section by preloading with 10% pentastarch. To avoid polypharmacy and to the probable benefit of the feto-placental unit, smaller doses of vasoconstrictors were administered to those receiving starch preloading. No perioperative side effects were reported in 160 patients.

Long-term i.v. infusions of starches can induce severe pruritis in approximately one-third of patients. There is evidence that starches are taken up into vacuoles in the mononuclear phagocyte system with additional deposition around small cutaneous nerves. These findings correlate with the severity of pruritis, diminishing as symptoms improve. Pruritis has been documented to last more than 3 yr, and it has been postulated that some patients may have impaired ability to metabolize such starches.

It would be interesting to know what proportion of French and colleagues’ patients experienced short- or long-term pruritis with the 15 ml kg⁻¹ volume administered, especially as intrathecal opioids were not used. It would be unfortunate if we were unable to use the documented haemodynamic benefits of preloading with starches if a significant proportion of mothers were to suffer long-term itching.
Editor,—The problem of itching related to starches has been known since the early 1980s.¹ Most of the evidence comes from isolated case reports and retrospective studies. The incidence of 32% reported by Gall and colleagues² was in patients with otological problems who received long-term infusions. There was little statistical analysis quoted in the English version of their earlier paper.³ Another prospective study did not find such a high incidence with low-dose starches,⁴ and some studies have reported pruritis in 5% of patients who have never received starches.⁵

A recent well conducted, prospective, multicentre investigation of 544 patients who received hydroxyethyl starch (HES) for various surgical and medical indications puts some perspective on the problem.⁴ A 1% incidence of pruritis was reported overall, but all cases of pruritis were in patients undergoing otological procedures who had also received nafidrofuryl. The authors concluded that the incidence was low in otological patients if the total dose of colloid did not exceed 300 g. In addition, for the common result of the Tuohy needle passing through the anterior midline? Indeed, Harrison states that the midline gap is unknowingly present, does this imply that the Tuohy needle must breach the ligamenta flava?

I doubt if my practice will change having read this article

Anatomy of the lumbar epidural region
Editor,—I read with interest the article by Harrison¹ detailing the anatomy of the lumbar epidural region, as studied using computerized tomography. This study and others he quoted have confirmed the presence of a midline gap between the ligamenta flava. This leaves me with some degree of confusion with regard to insertion of lumbar epidural catheters. With the standard midline approach of an epidural technique, the classical teaching is to feel for the “pop” as the Tuohy needle pierces the ligamenta flava and enters the epidural space. However, we are now realizing that the ligament exists in two halves with a midline gap.

Two points spring to mind. The first is regarding the actual cause of the sudden loss of resistance that we have all felt during our own clinical practice. Is it a result of the Tuohy needle passing through the anterior edge of the interspinous ligament? Or is it simply the fact that with patients in whom the “pop” is very apparent the ligamenta flava are likely to meet in the midline? Indeed, Harrison states that the midline gap is not always a constant feature.

If a gap is unknowingly present, does this imply that the “pop” is less evident and thus the risk of dural puncture greater? This brings me to my other point. Assuming the ligament to be complete in the lateral regions, would the lateral approach to the lumbar epidural space produce a lower incidence of dural puncture in view of the fact that the Tuohy needle must breach the ligamenta flava?

was based on evidence from an entirely different population of patients. Perhaps our findings will give us the answer.

4. Cox NH, Popple AW. Persistent erythema and pruritus, with a confluent histiocytic skin infiltrate, following the use of a hydroxyethyl starch plasma expander. Br J Dermatology 1996; 134: 353–7

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1 Cox NH, Popple AW. Persistent erythema and pruritus, with a confluent histiocytic skin infiltrate, following the use of a hydroxyethyl starch plasma expander. Br J Dermatology 1996; 134: 353–7
but I have been reassured and now understand why it is that some patients “pop” more than others.

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Editor,—Dr Cupitt’s letter raises a point which has also intrigued me. I have had various thoughts about the sensation which is felt on the needle entering the epidural space. It is possible that, were the approach to be truly in the midline, the needle may pass between the ligamenta flava, but that because of their proximity there would be sufficient grip for the feeling of give to be noted when the needle entered the space. However, I feel that it is more likely to be true that the midline is only attained infrequently, and that in general the ligament is traversed by the needle. I do not know if the lateral approach is associated with a different dural puncture rate, but it is the route which I routinely use for performing epidurals.

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This issue of the International Anesthesiology Clinics is a product almost exclusively of Boston, Massachusetts, with a single chapter contributed from workers from Cincinnati. Four of the authors are ‘RRT’ rather than ‘MD’ and this gives the book a technical and mechanical slant. It is a slim volume of 150 pages, nicely produced and presented, containing eight short chapters on aspects of respiratory management in critically ill patients. Only one of the chapters is more than 20 pages, and fortunately this is one of the best.

In this book, the editors aimed to provide a short survey of acute respiratory failure, and have included contributions on basic principles in addition to articles on recent developments. Perhaps because of the limited space available for each contribution, the articles on recent developments emerge from this constraint considerably better. In particular, there is a good and sceptical chapter on new modes of ventilatory support, an excellent account of ancillary therapies for patients with ventilatory failure, and a useful overview of weaning patients from mechanical ventilation. This topic has received a fair degree of attention in recent years and thus is well worth a review of this sort. Unfortunately the ‘difficult to wean’ patient is only offered the usual homilies such as ‘early recognition and correction of the causes of weaning failure’, but this sadly reflects the present state of the art.

The chapters that do less well are those that attempt to cover large swathes of physiology, such as ‘pulmonary mechanics and graphics’, and cardiopulmonary interactions. For those who are not certain, ‘graphics’ is the plotting of variables, such as flow–volume loops, which is now accessible and popular thanks to technical advances in ventilators. The consequence of squeezing a quart into a pint pot is that a lot of the basic material becomes incomprehensible at best, and misleading or incorrect at worst. The style becomes declamatory rather than explanatory, and many aspects of ‘shorthand physics’ can actually mislead.

The best chapter is by Robert Kacmarek on ventilator associated lung injury. This is a careful and thoughtful analysis of current information, with useful comparison and synthesis of the data. The author is not afraid to add his own interpretation, which is exactly what is required in a book of this size.

The standard of editing is high, and the only obvious overlap is the presence of the same figure in two different chapters. The weakest chapter is that on the pathophysiology of the acute respiratory distress syndrome, which contributes little to previous work, and indeed ignores much of very recent research into cellular mechanisms.

For the practitioner already aware of the background of these topics, this book may offer a useful update. It will be no help to those studying the more basic facts for examination purposes. I will read it in future for its two best chapters, and forget the others.

G. Drummond


This book is the result of the deliberations of the task force on epidemiology of the International Association for the Study of Pain. The foreword by John Loeser states that the report serves as a collation of available information and sets the standards for future studies. There is no doubt that the authors have achieved a very full collation of present knowledge on pain and predisposing risk factors, which must be essential reading for any physician involved in the treatment of chronic painful conditions.

The book starts by discussing the potential of epidemiology. This is highlighted by describing the impact that epidemiology had had in the field of medicine, and how epidemiology can be applied to health service planning, increasing the efficient working of the health care service, identifying pain syndromes and their natural history, and prevention. Epidemiological methods are described clearly, together with the shortcomings and difficulties that can be encountered. The importance of both preventive and clinical studies is stressed, being the very core of clinical epidemiology.

The book then moves forward to review the state of knowledge on a variety of factors relating to pain and various painful conditions. The chapter on psychological factors gives an excellent review of the various psychosocial factors that impact on the development of chronic pain, both causative and resultant. Gender considerations show definite age by sex differences in the prevalence of many pain conditions, but there is little evidence to explain these differences. Cross-cultural issues are reviewed. The need for family counselling to support the suffering pain patient as a prerequisite to expensive medical intervention or financial support is delineated, in addition to the importance
of such considerations in the understanding of the phenomenon of pain. Differences in the handling of suffering relate to spiritual dimensions, and pain phenomena have universal and ethnospecific aspects. Illness beliefs are as deadly or healing as drugs. The impact of age on pain is discussed for children and older people.

Chronic post-surgical pain comprises 20% of attendances at chronic pain clinics, and well designed prevalence studies show that one-third or more of patients undergoing surgery complain of ongoing problems. Other unpleasant symptoms include phantom sensation, numbness and allodynia. There is a large gap between patients’ expectations of their operation’s ability to relieve pre-existing symptoms and their experiences after operation in terms of unresolved morbidity and new problems. Phantom limb pain can be prevented by early counselling intervention and aggressive enhancement of function, reducing the incidence to 2% in a series of 2000 amputees. No pre-emptive analgesic studies compete with such numbers.

Other conditions reviewed include central post-stroke pain, migraine and headache, facial pain, temporo-mandibular disorder pain, neck pain, shoulder pain, low back pain and knee pain. Work-related risk factors are considered, especially for neck and low back pain, together with non-work-related risk factors. There is a common plea from all authors for prospective studies with periodic follow-up and large sample sizes to enable knowledge to develop.

This book is an excellent text that reviews the problem of pain in society and for the individual, highlighting known pre-disposing risk factors, and the paucity of knowledge as to the real impact or lack of impact of medical and surgical treatment. All physicians, indeed health care workers, who deal with chronic painful conditions should read at least the general chapters, if not the entire book. It is well presented and thought provoking.

T. Nash


The aim of this book is to provide the basic principles required for the satisfactory management of acute (postoperative) and chronic pain. The bias of the book will appeal mostly to those with an interest in chronic pain. Acute pain is covered better in more specialist texts.

The first two chapters cover neurophysiology, biochemistry, pain measurement and pain management services. These chapters are well researched with up to date references. The reader will obtain a clear understanding of the different pain types: nociceptive, neuropathic, visceral, psychogenic and cancer.

Chapters 3 and 4 on primary analgesics (opioids and NSAID) are restricted to the mechanisms of action, classification, routes of administration and side effects of these drugs. Opioid tolerance, opioid dependence and opioids in non-malignant pain are well referenced. COX-2 NSAID receive good coverage but there is no mention of NO-NSAID.

Chapters 5 and 6 on secondary analgesics provide excellent overviews of the wide range of drugs used, concentrating on the mechanisms of action and guidelines for use. The 222 references provide more than enough additional reading.

Peripheral and central neural block are covered in chapters 8 and 9. Again, the emphasis is on basic principles. The chapters cover most of the blocks in common use and despite limited space provide some very precise detail, an indication of the writer’s practical experience.

Professor Shipton has had a problem of what to include in his handbook. The more clinical chapters on drug delivery (spinal implants, PCA, syringe drivers), intervention therapies (cryoanalgesia, radiofrequency, neurolytic agents), stimulation analgesia (acupuncture, TNS, spinal cord stimulation), surgical intervention (rhizotomies, cordotomy, microvascular decompression), regional pain syndrome and the sympathetic nervous system, psychotherapy and rehabilitation, have been chosen because they exemplify the principles of general management. The book is not a reference for individual pain conditions. The last chapter, ‘The future’, comprising 39 pages and 233 references, is an excellent review of the possible future directions in pain relief.

In summary, this is a well researched handbook covering the basic principles of pain management. It gives an up to date, rapid understanding of the subject.

D. Eastwood


It took me a few moments to work out why it was called TEAL but once I had mastered that, the rest was easy. The software was supplied as a single CD-ROM and took less than 5 min to install on my Toshiba notebook PC running Windows 98. I also tried it on a desktop machine running Windows 95, and again it installed rapidly and flawlessly. The instructions say that it will also run under Windows 3.1x, Windows NT or on an Apple Macintosh (O.S. version 7.1 or later). The installation program placed an icon labelled ‘knowledge finder’ on the desktop—a double click to run it.

The instruction booklet is clear and easy to read, but not needed often as the software is very straightforward and intuitive to use. I now had access to the full text of every issue of the four journals, British Journal of Anaesthesia, Canadian Journal of Anaesthesia, Anesthesia and Analgesia and Anaesthesiology for the 5-yr period 1994–1998. Searching is a simple matter and an article can be addressed in several ways. First, a small window offers the four

This is an excellent text which draws together several world leading authors to provide a wide ranging review of the current state of knowledge of the basic science of opioids and their role in clinical analgesia. The 18 chapters move from the gene structure and function of opioid receptors, to the clinical use of opioids in cancer, visceral pain and obstetrics. As a clinician, the chapters on the basic science of opioids provide an excellent and up to date review of many topics, including opioid receptors, endogenous opioids, supraspinal and spinal mechanisms, and peripheral opioid analgesia. In particular are discussions around the clinical relevance of peripheral opioid receptors and pre-emptive analgesia, the specific clinical circumstances under which such concepts may be used and the reasons why they are not always effective. Inevitably, the basic science chapters are not easy reading and require a degree of concentration that is not necessary for the more familiar content of the clinical chapters. The chapters are well referenced and reflect the rapid changes that have taken place in recent years, especially in basic sciences. Of 101 references in the opioid receptor chapter, an impressive 82 were less than 5 yr old and only a single reference was not published in this decade.

As the introduction states, the text is aimed at providing a 'comprehensive overview of the often controversial and confounding use of opioids in pain control. Serving both scientists and clinicians . . .' This mixed readership affects the perception of the different chapters depending on one’s own area of knowledge and expertise. Inevitably, some of the clinical chapters were disappointing as they reflected a somewhat simplistic and personal approach to current practice and provided a sometimes embarrassingly short reference list of standard tests. Does this reflect the current state of clinical research overall? Not entirely, as the chapter on patient-controlled analgesia provided insight into some of the clinical problems and challenges that affect us.

We are all aware that factors such as inter- and intra-patient differences within clinical studies cause significant problems in trying to design high-quality studies and interpret the results. However, the confusing and variable presentation of the data within the section on epidural analgesia was disappointing. Not least was the inclusion of at least one reference as evidence for lack of effectiveness when the study had originally concluded that addition of bupivacaine was beneficial, and the reference to a table labelled as PCA data in the epidural discussion.

The book has been well constructed with cross-references between the different chapters and surprisingly little repetition in the content, which probably reflects some hard work by the editor. However, one annoying aspect was that some of the figures and tables had inadequate legends so that they could not easily be interpreted on their own
and frequent searches through the text were required to understand their significance.

Overall, this is an excellent book which is of a manageable size to read in its entirety; it also provides an excellent source of material for individual topics. I would recommend it for departments and especially for clinicians who may have lagged behind in their reading of basic science material and who relish the challenge of interpreting such work and identifying new clinical applications for it.

J. Peacock