Correspondence

Machine and monitoring failure from electrical overloading

We wish to report a case of electrical failure of a target-controlled infusion (TCI) pump and a Datex-Ohmeda AS/3 monitor as a result of accidental overloading of one of the auxiliary mains socket outlets on the rear panel of an Aestiva 3000 anaesthetic machine.

An ASA 1 patient was undergoing a rhinoplasty under propofol total intravenous anaesthesia (TIVA) using a Diprifusor Master TCI infusion pump. The anaesthetic machine and the AS/3 monitor had been checked in accordance with the Association of Anaesthetists guidelines [1]. Although the Diprifusor pump did not have a battery back up, it functioned satisfactorily when plugged into the mains outlet. At the time of the operation, the pump was plugged into a four-socket extension board placed on the floor. Towards the end of an uneventful operation, the surgeon asked for a nasal splint to be prepared. The splint (Thackray Instruments, Powys, UK) is made of a thermoplastic material, which is moulded to fit the nasal contour by immersion in very hot water (approximately 90 °C). Hence, one of the theatre staff, without the knowledge of the anaesthetist, plugged an electric kettle (rated at 2200 Watts) into the four-point extension board on the floor (to which the Diprifusor pump was connected). It was not realised that the extension lead was actually connected to the auxiliary power outlet on the rear panel of the anaesthetic machine (Fig. 1). As soon as the kettle was switched on, all the equipment connected to the auxiliary power outlets of the anaesthetic machine failed instantly. This included the AS/3 monitor and the Diprifusor infusion pump. As a result, TIVA and instrumental monitoring were interrupted. We immediately switched to inhalation anaesthesia (isoflurane and nitrous oxide) and continued clinical monitoring until instrumental monitoring was brought in from the anaesthetic room. It took several minutes before it was realised what had happened and the problem was resolved. Fortunately, the patient was unharmed.

The current British and European Standard for the anaesthetic workstation states that anaesthetic machines can be supplied with up to four auxiliary mains sockets [2]. These may accept a standard mains plug as with the Datex-Ohmeda Aestiva 3000. The outlets are intended to provide electric supply to anaesthetic equipment such as monitors, electrically operated vaporisers (Tec 6) and infusion pumps. The current load that each of these sockets can handle limits the type of electrical equipment that can be connected to these power outlets. The Aestiva has three sockets rated at 1 A and a fourth outlet rated at 2 A (Fig. 2). Each outlet is provided with a circuit breaker, which turns off the power to the socket in the event of an electrical overload [3]. In addition, there is a 'master' circuit breaker for all four outlets, which limits the total outlet current to 3 A. In theory, if the power load drawn from an individual socket were to exceed its current rating, the outlet circuit breaker would cut off the power to *that* socket only, without affecting the function of the remaining three sockets. However, this incident demonstrates that if the current rating of the appliance is very high, it is possible for the 'master' circuit breaker to open *in addition to* the individual circuit breaker, thereby cutting off power to all four sockets. The current drawn by the kettle was 10 A, much in excess of the capacity of the auxiliary sockets.

This incident highlights several safety issues.

Safety checks. The anaesthetic equipment checklist must include inspection of the auxiliary power outlets in the rear panel of the machine. The anaesthetist must ensure that only appropriate anaesthetic equipment is connected to the sockets and that the current rating of the equipment does not exceed the limits of the individual circuit breakers. Connection of appliances such as forced air warmers and extension sockets to these auxiliary power outlets must *never* be permitted. Extension boards with multiple sockets are particularly dangerous, as a wide range of electrical appliances used in an operating theatre environment can potentially be connected to them and thereby to the anaesthetic machine. All members of the theatre staff should also be made aware of the risk of such practice, and no one except the anaesthetist should be allowed to plug any device into the

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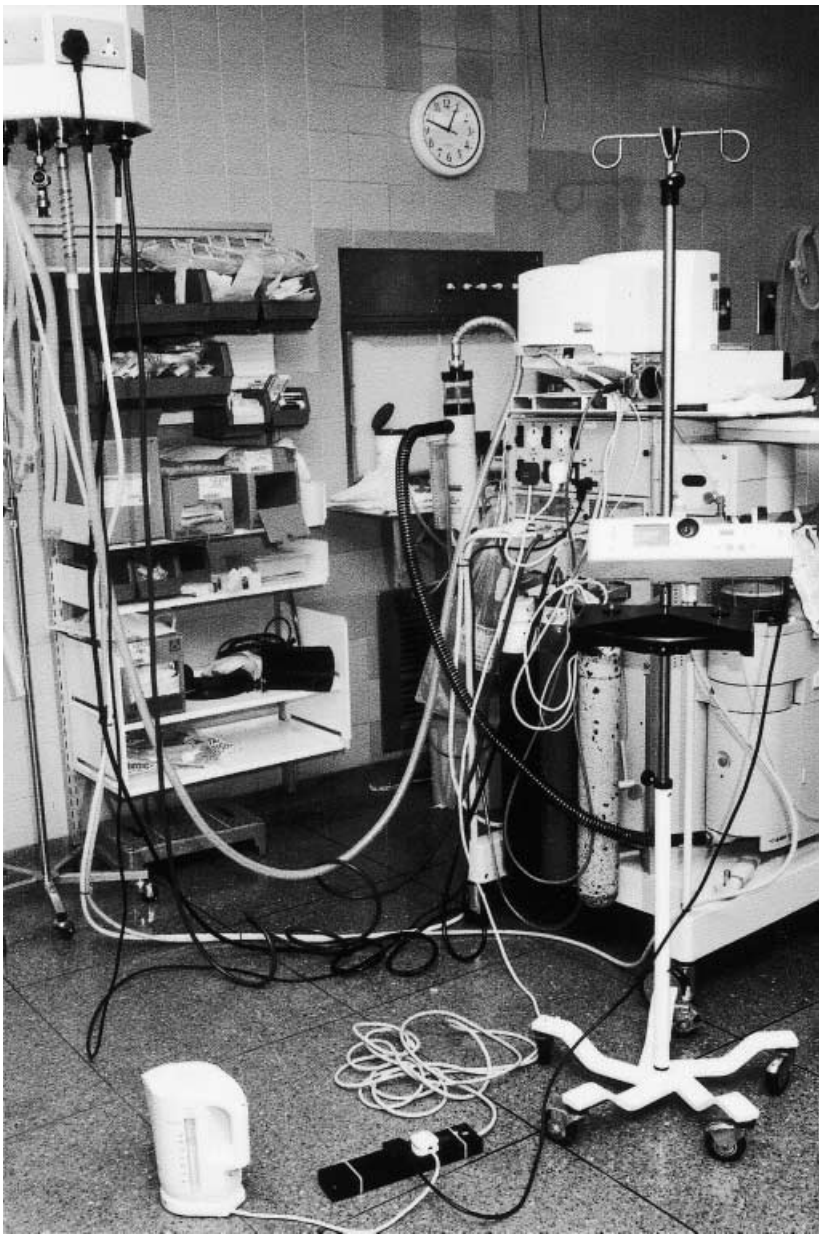

Figure 1 Arrangement of electrical components involved in the incident.

anaesthetic machine at any time. In the same vein, the anaesthetic machine should always be plugged directly into a wall or pendant-mounted mains socket – never into an extension lead.

Design safety. The present international standard governing the design of anaesthetic workstations allows auxiliary mains socket outlets to be mounted on the anaesthetic machine, which accept a standard three-pin mains plug. This incident would have been averted if the outlet sockets were designed *not* to accept such a plug.

Inappropriate use of domestic equipment. Appliances such as electric kettles do not meet safety standards for theatre equipment with regard to power consumption and earth leakage currents and are unsafe in such an environment.

Finally, it must be accepted that the use of a Diprifusor infusion pump without a battery back up was unsafe practice. This has since been addressed.

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Emergency physicians: additional providers of emergency anaesthesia?

I was interested to read the editorial (Lockey & Black. *Anaesthesia* 2002; **57**: 629–31) having recently returned from two years working in a 'Level One' trauma centre in the USA. The editorial suggests, on the basis of the American National Emergency Airway Registry [1] which collected data from 35 centres in the US, that the American solution to the problem of providing emergency anaesthesia and intubation in the Accident and Emergency department is a satisfactory one. In the centre in which I worked, Physician delivered 'anaesthesia' occurred both in the emergency room and the intensive care unit. My experience has led me to the following conclusions, which do not support emergency physician delivered anaesthesia.

1 When an emergency physician is delivering anaesthesia, he/she is removed from their traditional role in the emergency room. One needs therefore additional emergency department medical staff to manage the patient to the same standard.

2 Patients in the emergency department and intensive care are frequently

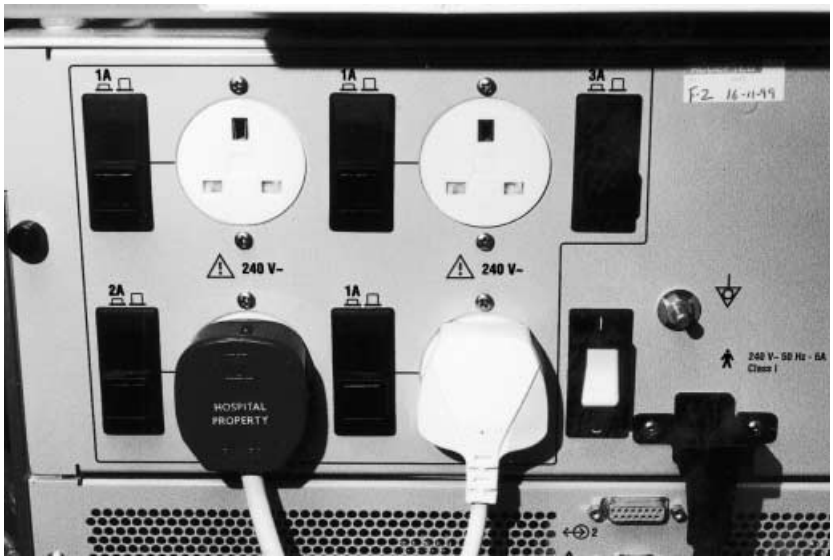

Figure 2 Auxiliary power outlets on the rear panel of the Aestiva 3000.

those with the most challenging airways to manage.

3 Trauma patients with head injuries and thoracic injuries (including aortic injuries) provide special challenges and will not be well managed with a 'recipe'-type Rapid Sequence Induction.

4 The American system relies on the 24-h presence of an attending emergency physician and in 'Level One' trauma centres, an attending anaesthesiologist (we attended all trauma calls, our role frequently being the supervision of trainee emergency physicians in airway management).

5 In my experience, failed intubations and unsatisfactory administration of anaesthetic drugs and sedation were more common in patients who had anaesthesia delivered by the emergency physician.

In order that we maintain a high standard of anaesthetic care to our patients, we should ensure that adequately trained and experienced anaesthesia staff are available to attend the emergency department 24 h a day.

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Reference

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Measurement of systemic oxygen uptake during low-flow anaesthesia with a standard technique vs. a novel method

The recent article (Leonard *et al. Anaesthesia* 2002; **57**: 654–8) is important from the point of view that one needs to calculate oxygen uptake (V_{O_2}) to ensure that adequate oxygen flow is maintained during low-flow anaesthesia in a circle system. There is an easier method of calculating V_{O_2} from the oxygen concentrations in the inspired and expired gases, which are measured and displayed by most of the modern anaesthesia monitors.

In my practice of low-flow anaesthesia, I ensure that the addition of oxygen into the circle system is slightly over the V_{O_2} of the patient. The two principles that I follow to attain the lowest possible flow during low flow anaesthesia are:

1 There should be an adequate fresh gas flow into the circle system to compensate for the V_{O_2} every minute (assuming nitrous oxide is not used) and to overcome trivial leaks. I would accept the lowest flow that would ensure that the reservoir bag or the ventilator bellows remain full in spontaneous and controlled ventilation, respectively.

2 An appropriate amount of oxygen should be added to the circle, which depends on the amount of oxygen consumed by the patient. This is worked out by the following formula:

$$F_iO_2\ (\%) \times V_{tI} - F_eO_2\ (\%) \times V_{tE} = O_2 \text{ consumed per breath.}$$

where F_iO_2 is the fractional inspired concentration of oxygen, F_eO_2 is the fractional expired oxygen concentration, V_{tI} is the inspired tidal volume and V_{tE} is the expired tidal volume.

$$V_{O_2} = O_2 \text{ consumed per breath} \times \text{Respiratory rate.}$$

For a quick calculation, I would multiply the $F_iO_2 - F_eO_2$ difference by the minute ventilation to calculate the V_{O_2}.

An important question is whether the oxygen consumption calculated by the above-mentioned formula is limited by the amount of oxygen flowing into the circle system?, i.e. the patient cannot take up more oxygen than that given to him.

Two things I would watch out for:

The $F_iO_2 - F_eO_2$ difference. The difference is usually about 5% in an awake individual. A widening in the difference may mean that the lungs are extracting as much oxygen as possible from the inspired gases (is the oxygen inflow into the circle limiting oxygen uptake?), or a high end-tidal carbon dioxide concentration is reducing the F_eO_2.

Response of oxygen consumption to increasing oxygen inflow into the circle. I perform this to ensure that the inflow of oxygen into the circle system is not the limiting factor for the patient's oxygen consumption. I increase the fresh gas flow of oxygen and recalculate oxygen consumption to ensure that the supply of oxygen exceeds the use by the patient.

One of the drawbacks of using low fresh gas flow in a circle system is the possibility of delivering a hypoxic mixture. Application of the concept of oxygen consumption and calculating it may help to reduce the risk of the patient becoming hypoxic.

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Cardiac output determination using compliance

Last February, I described a method for deriving cardiac output by dividing the product of compliance and mean arterial pressure by the time constant of the arterial system (Campbell. *Anaesthesia* 2002; **57**: 185). A recent letter has been published in which the theory is accepted but concerns are raised regarding two practical issues (Linton. *Anaesthesia* 2002; **57**: 715). One concern is that errors will occur from the variable increase in systolic pressure in peripheral arteries so that measurements of the radial artery systolic pressure will not equal the aortic systolic pressure. My response is that these increases have been recognised for many years and are associated with the pulse pressure wave becoming more peaked and narrow as it passes along the artery. When, however, the mean arterial pressure in the peripheral vessel is compared with the mean aortic pressure, they are almost identical [1]. As it is the mean arterial pressure that is used for the cardiac output measurement, there is little error from this source.

The other concern expressed by Linton is that reflected waves within the arterial system produce distortions that prevent an accurate measurement of the time constant. A trace was shown to support this view. To this I would add that distortions are also commonly produced from the transducer/catheter combination from a variety of artefacts [2]. As these distortions occur around the dicrotic notch, accurate readings can still be obtained from the end part of the diastolic trace just before the upswing of the next pulse pressure wave.

On the trace submitted by Linton, the time constant is approximately 1.25 s and the mean arterial pressure is in the region of 70 mmHg. Taking a reasonable adult arterial compliance of 1.6 $ml.mmHg^{-1}$ and using these figures as an example, the cardiac output would be 5.4 $l.min^{-1}$ with a systemic vascular resistance of 1040 $dynes.s^{-1}.cm^{-5}$, and a stroke volume of 60 ml at a heart rate of 90 $beats.min^{-1}$.

Multiple readings of the selected part of each exponential curve will improve accuracy and averaging these over a period of about 20 s will eliminate errors from variations in stroke volume. Perhaps greater accuracy of the time constant would be available through an optical or perhaps an ultrasonic system in preference to the older technology of liquid-filled pressure transducers.

For many years, clinicians have accepted complicated expensive methods for measuring cardiac output so that a new simple method will be viewed with scepticism. If, however, accuracy equivalent to the standard methods can be achieved, the exciting potential exists for cardiac output monitoring to be available for every major surgical procedure.

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Connector mix-up on an anaesthetic machine

We would like to report a recent ventilator malfunction that occurred as a result of a design feature making it possible to attach the ventilator driving gas pipeline to the scavenging outlet.

The pre-operative anaesthetic machine check was carried out and nothing unusual was detected. General anaesthesia was induced for a Caesarean section. The ventilator was switched on and the bellows went up and down more slowly than determined by the ventilator settings. More importantly, the patient was not adequately ventilated as detected by capnography and auscultation. A rapid check of the anaesthetic machine did not reveal any cause for the malfunction. The patient was hand ventilated for the remainder of the procedure.

Postoperatively, a more detailed examination of the anaesthetic machine, a Modulus II Plus manufactured by Ohmeda (Madison, WI, USA), revealed that the oxygen pipe that supplies gas to drive the bellows was mistakenly connected to the scavenging outlet underneath the bellows housing. Our daily anaesthetic machine check did not detect this problem because when the drive hose is connected in this fashion the bellows will still move, although with a limited range of travel. A reduced tidal volume is produced, accompanied by the bellows leaking gas and deflating at a slow rate. The manifold connections for both the driving and scavenging pipes are very similar with internal diameters of 17 mm and 19 mm, respectively (Fig. 3). The oxygen pipe may be attached with ease to the scavenging connector. There are no distinguishing features on either connector to aid reconnection.

Figure 3 Ventilator manifold of Modulus II Plus Anaesthetic Machine.

The distributors of the machine in Ireland (Oxygen Care Teo) – have been informed of the problem and have fitted an alternative manifold with a 30-mm scavenging connector that will not accept the driving gas pipeline. They state that newer Datex-Ohmeda machines have this 30-mm connector on the manifold. There are approximately 14 Modulus II Plus machines in the South of Ireland. The same manifold may also be found on other Ohmeda anaesthetic machines including the Excel 210 and 410. Oxygen Care Teo have told us that they are actively seeking out this manifold type in order to modify it to prevent similar misconnections. Anaesthetists should be aware of the possibility of this mix-up that may escape detection during the routine anaesthetic machine check.

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A reply

Thank you for the opportunity to reply to Drs Croinin and Keogh's letter. They report that is possible to switch hoses on 17-mm and 19-mm manifold connections. An attempt to switch hoses on a Mod II Plus was performed. The 17-mm hose would go onto the 19-mm fitting with additional force applied. The 19-mm hose (0225-0807-700) that we supply with our Scavinging assembly would not stay on the 17-mm fitting.

In the O & M manual, pre-operative checkout procedures figs 4–9 and 4–10 show the correct connections for the 17-mm and 19-mm hoses. If the pre-operative check procedures are performed with the 17-mm and 19-mm switched on, the manifold failures will be encountered. A failure will be noted on page 4–16 step 12 where the reading will be lower than the 3.3–4.3 l specified, and no up-and-down movement of the bellows will be visible. On page 4–19 and 4–20 steps 15 and 16 will also fail. If the Ohmeda pre-operative check procedures are completed this condition would be detected.

The Checklist for Anaesthetic Apparatus published by the Association of Anaesthetists of Great Britain and Ireland states: 'It is not intended to supplant any pre-anaesthetic checking procedures issued by manufacturers, but should be used in conjunction with them'.

The connection installed into the manifold (0236-0478-700) was changed from 19 mm to 30 mm on ECO 96-3500. Standards were being drafted calling for a 30-mm conical connector for Anaesthetic Gas Scavenging Systems.

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ENT surgeon pipped at the post by the laryngeal mask airway

We write to describe a novel (mis)use of the laryngeal mask airway.

A fit and well 3-year-old girl required a general anaesthetic to remove a cherry seed from her nose. Following induction with 100 mg propofol, a size 2 classic laryngeal mask airway was inserted upside down and attached to an Ayre's T-piece to administer oxygen in air. The laryngeal mask airway cuff was inflated to create a seal. Two gentle puffs of positive pressure expelled the seed from her nose. There is a small potential risk of eustachian tube barotraumas using this technique. We used a gentle (sneeze-like) puff of positive pressure therefore minimising the risk of eustachian tube insufflation. The laryngeal mask was then re-inserted (this time in the correct position) to allow the child to breathe spontaneously until she emerged from anaesthesia. No surgeon required.

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Airway management is simpler with the existing tracheal tube

A recent article (Fisher *et al. Anaesthesia* 2002; **57**: 253–5) describes a new method of airway management using a microlaryngeal tube during percutaneous tracheostomy. The procedure involved the existing tracheal tube being replaced with a longer 5.0-mm internal diameter microlaryngeal tube. Prior to tracheostomy, the cuff was deflated and the resulting gas leak prevented by a pharyngeal throat pack. I consider there are inherent problems with this particular technique of airway management that need to be addressed.

(a) The airway pressure may increase following ventilation through a smaller microlaryngeal tube in patients with poor lung compliance, and adequate ventilation may be impossible to achieve. Mechanical ventilation via a microlaryngeal tube can lead to hypercarbia. The rise in arterial carbon dioxide may be detrimental in patients with raised intracranial pressure.

(b) There have been numerous studies evaluating various airway devices including the laryngeal mask [1], the intubating laryngeal mask [2], the airway management device [3], the Combitube [4] and a microlaryngeal tube [5] being used during retrograde translaryngeal tracheostomy. None of these has achieved widespread acceptance. Major complications including loss of airway and hypoxic episodes have been reported in about 14% of patients during the process of changing the existing tracheal tube to a micro laryngeal tube during translaryngeal tracheostomy [6].

(c) Accurate needle and guidewire placement in the trachea is an important step during percutaneous tracheostomy prior to insertion of single or multiple dilators. Life-threatening complications can occur during the needle placement, dilatation and subsequent placement of a tracheostomy tube. Fibreoptic bronchoscopy is increasingly used and has been found useful to guide these steps during tracheostomy [7]. In addition, continuous endoscopic guidance has been shown to increase the safety of percutaneous tracheostomy [8]. This series reports a case of oesophageal dilatation and placement of a tracheostomy tube necessitating thoracotomy for the oesophageal repair. The use of a microlaryngeal tube during tracheostomy does not allow passage of a bronchoscope to visualise the key steps of the

procedure. The oesophageal tear could have been avoided by bronchoscopy.

(d) In patients with thick secretions or blood clot in the airway, it would be difficult to perform an effective suction via a microlaryngeal tube during tracheostomy.

Despite the growth of multiple percutaneous techniques in recent years, there has been a decreasing trend of immediate or early complications of tracheostomy since the introduction of the Ciaglia serial dilatational technique. This may be as a result of more training and increasing use of bronchoscopy during the procedure. We believe using a bronchoscope through the existing tracheal tube should lead to a safer percutaneous tracheostomy. The use of a microlaryngeal tube prevents this option.

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A reply

Thank you for allowing us to take the opportunity of replying to Dr Mallick's letter. Dealing with the points raised sequentially.

Control of ventilation. We agree that the control of ventilation during tracheostomy is important. Provided that the ventilator is adjusted to the 'constant volume' mode and that the inspired tidal volume is adjusted so that the exhaled tidal volume and minute ventilation remain close to or greater than that recorded prior to commencing the tracheostomy, then the arterial carbon dioxide tension can be maintained at an optimal value. Under these conditions, the peak airway pressure will rise due to the resistance of the smaller tracheal tube; however, there is no or little change in the plateau pressure. Correct packing of the pharynx will reduce any gas leak to a minimum.

If an ordinary tracheal tube is used with a bronchoscope, then depending on the relative sizes of the relative size of the tracheal tube and the bronchoscope, the effective diameter of the tracheal airway may well be reduced to a value not dissimilar to that of a microlaryngeal tube.

Following insertion of the tracheostomy tube, ventilation can still be maintained via the micro laryngeal tube. This allows for the correct placement of the tracheostomy tube to be confirmed before the original airway is lost. This is not always possible when the original tracheal is used and pulled back as the tracheostomy tube is inserted into the trachea.

Changing the existing tracheal tube for a microlaryngeal tube. In the series reported, the tracheal tube was always changed over a gum elastic bougie. The risk of losing the airway during this part of the procedure was recognised as a potential problem. The fact that no difficulties were encountered using this simple precaution contrasts dramatically with the figure of 14% incidence of complications suggested by Dr Mallick.

Use of the bronchoscope. It was pointed out in the original paper that a disadvantage of this method was that it did not allow the utilisation of a bronchoscope. We agree that the use of a bronchoscope for confirming the placement of the guide wire and tracheostomy tube is desirable. Since the publication of the paper, we have been extending our technique so as to permit the use of a bronchoscope while maintaining the airway with a microlaryngeal tube. The oesophageal tear reported in the series represents a major complication. This problem may even occur despite the use of a bronchoscope. The relevant point here is that the misplacement of the tracheostomy tube was readily identified without the loss of airway control. The situation could be rectified without loss of the patient's oxygenation or ventilation. In the case reported, the patient was not put at risk, the tear was explored and repaired in the neck and did not require a thoracotomy.

Suctioning via a microlaryngeal tube. During this series, no procedure needed to be abandoned due to difficulty with maintaining an airway. On a number of occasions following re-intubation, the microlaryngeal tube needed to be cleared of secretions using a suction catheter of the appropriate size. It is feasible that torrential haemorrhage could occur during tracheostomy with the formation of large clots. It would be anticipated that the percutaneous procedure would need to be abandoned in such an instance.

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Stylet for reinforced laryngeal mask airway

With more widespread use of the reinforced laryngeal mask airway (RLMA), many devices have been created to facilitate its insertion, since it has a longer and more flexible tube (Fig. 4A) than the classic laryngeal mask airway. These are classified into two categories: outer and inner devices. The outer devices (standard Magill forceps [1], Bosworth introducer [2]) have potential disadvantages of causing trauma and occupying the oral space. The inner devices (metal stylet, small tracheal tube [3], combined metal stylet and tracheal tube [4]) also have some problems. It is difficult to remove them after the insertion of the RLMA when the stylet is tightly fitted to the RLMA. If it is too loose, however, it can rotate along its axis. We would like to describe an improved combination metal stylet and tracheal tube (Fig. 5B).

Our idea was to use the cuff of the tracheal tube to fix the stylet against the RLMA. It allows tight fixation by inflating the cuff and smooth removal by deflating it after insertion of the RLMA. To produce this stylet for a size 4 RLMA, we used a cuffed tracheal tube (ID 4.5 mm, Lo-Contour™, Mallinckrodt, Ireland), Nelaton's catheter (No.6, Mizuho, Japan) and a PVC-covered stylet for children (Portex, UK) (Fig. 5A). First, the children's stylet is passed into the Nelaton's catheter. Then, the Nelaton's catheter is passed into the tracheal tube (Fig. 5B). Adequate lubrication of the stylet and

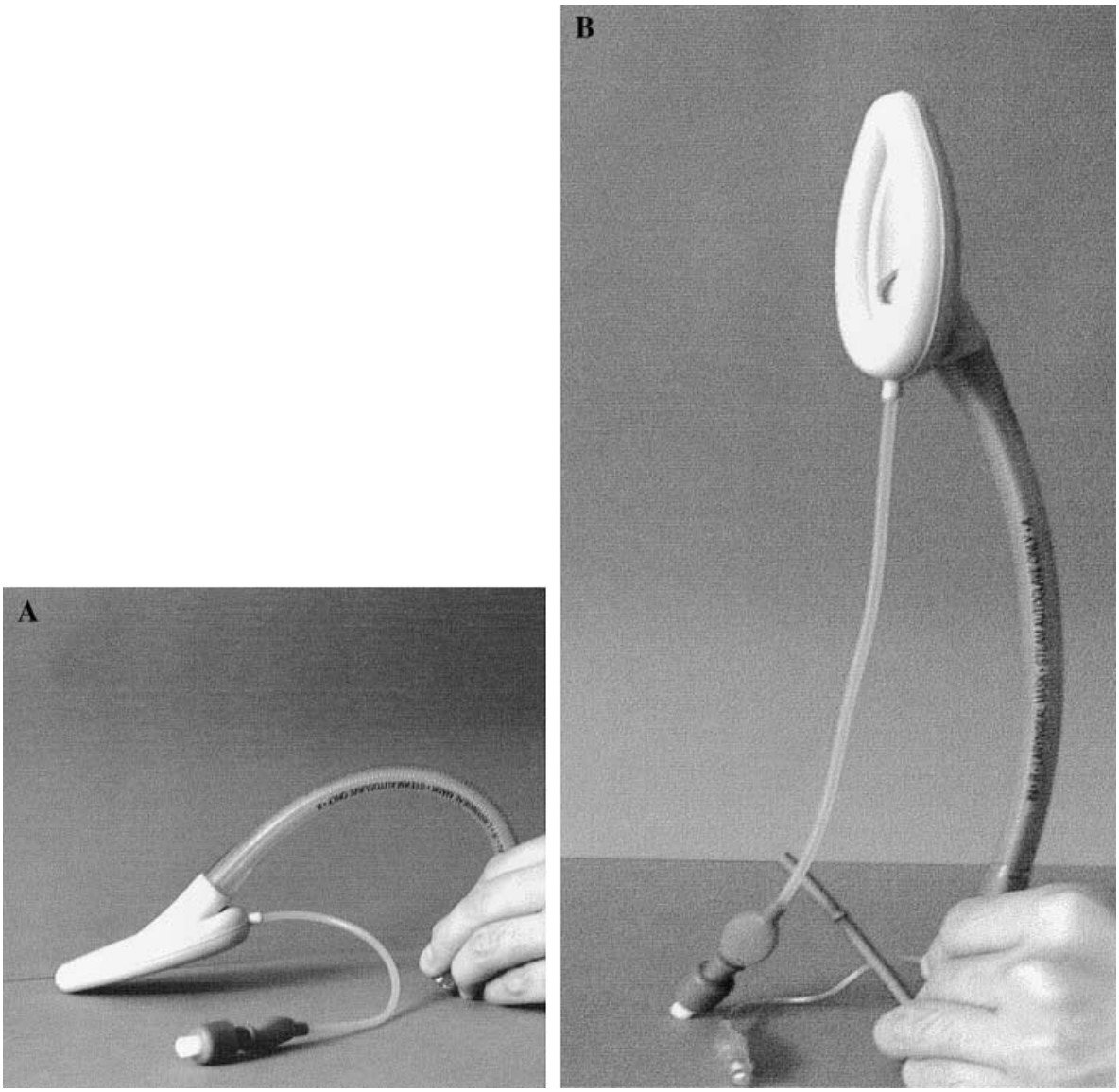

Figure 4

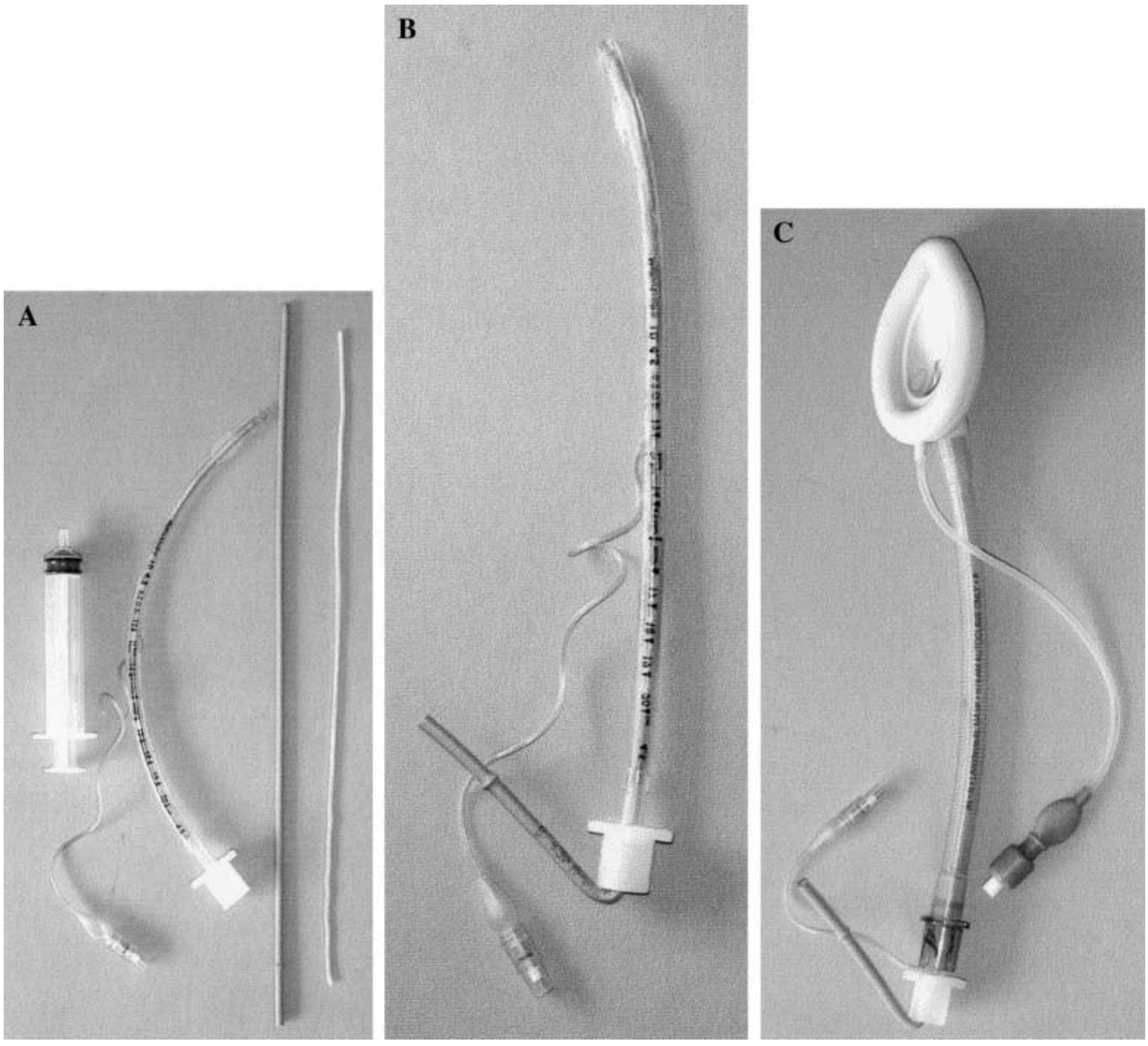

Figure 5

tracheal tube allows smooth insertion of the stylet into the RLMA. The length of the tracheal tube just fits the size 4 RLMA, so the distal end does not protrude through the grill of the mask (Fig. 5C). To fix the stylet against the RLMA, the cuff of the tracheal tube is inflated with approximately 0.5 ml of air. This arrangement allows the tube of the RLMA to be rigid enough (Fig. 4B) to insert it easily without trauma. After insertion of the RLMA, the cuff of the tracheal tube is deflated, allowing smooth removal of the stylet without displacing the RLMA. Since we have no adequate stylet for adults to give rigidity to the tracheal tube, the Nelaton's catheter enlarges the stylet. We have found this stylet with a cuffed tracheal tube is convenient for insertion of the RMLA.

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Joint disagreement

We read with great interest the case report entitled 'Cannabis abuse and laryngospasm' (White. *Anaesthesia* 2002; **57**: 622–3). However, we disagree with the author's conclusion that there exists 'an association between cannabis smoking and peri-operative laryngospasm'.

The report documents a case of laryngospasm during general anaesthesia in a patient with a 'five a day cannabis-containing cigarette habit'. The author mentions that prior to extubation, direct laryngoscopy under deep anaesthesia proved the laryngopharynx to be clear of debris. We assume, though are not told, that extubation then occurred under deep anaesthesia. We do not accept that cannabis was the most likely cause of laryngospasm. Laryngospasm could have been caused by an inadequate depth of anaesthesia (be that either insufficiently deep or insufficiently light), or by the tobacco smoke contained within the 'joint' [1]. Common things occur commonly, or in this case common things lead to common complications. To infer from one case report that the laryngospasm was due to the cannabis content of a tobacco-containing cigarette is a large assumption and not statistically sound. A cause–effect relationship cannot be established in a situation where an equally likely trigger exists as a confounding variable, namely cigarette smoke.

To demonstrate an association between cannabis use and postextubation laryngospasm, one should compare the frequency of laryngospasm in groups of patients controlled for tobacco smoking, i.e. compare tobacco smokers with those who smoke tobacco and cannabis or compare non-smokers with those who smoke cannabis joints containing no tobacco.

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Cannabis smoking and anaesthesia

I read with interest the account of severe laryngospasm following extubation of a patient who was a known cannabis smoker (White. *Anaesthesia* 2002; **57**: 622–3). Over 2 years ago, I encountered unexpected problems during the induction and maintenance of anaesthesia in a patient who had knowingly withheld the fact that he was a cannabis smoker. The 34-year-old man was 5 foot 6 inches tall, weighed 94 kg and was scheduled for surgical removal of wisdom teeth as a day case. The Assessment Clinic blood pressure was 115/80 mmHg.

He had no complaints other than heartburn with occasional reflux. He rarely drank alcohol but admitted to smoking 10–20 cigarettes a day. He denied any problems with his chest and had had an anaesthetic one year previously, which he said had been uneventful. He had beta-Thalassaemia Trait with a haemoglobin of 14 $g.dl^{-1}$. The patient was asked not to smoke on the day of surgery and given ranitidine 150 mg to take on the evening and morning before the operation.

Pre-operatively, he was assessed as being 'fit and well' and had drunk only water 3 h before. In the anaesthetic room, he seemed to be very nervous with a pulse rate of 95 min^{-1} and blood pressure of 150/80 mmHg. Midazolam 2 mg, metoclopramide 10 mg and dexamethasone 8 mg were given intravenously. Otrivine nose drops were administered. After pre-oxygenation, anaesthesia was induced with fentanyl 100 μg, propofol 200 mg and the lungs ventilated with oxygen and sevoflurane via a facemask and circle system. Succinyl choline 100 mg was given to facilitate intubation with a cuffed nasotracheal tube size 6.5 mm. A pharyngeal throat pack was inserted. Spontaneous respiration returned quickly but he was very difficult to 'settle', even with a very high inspired concentration of volatile anaesthetic, initially sevoflurane in oxygen. The saturation fell significantly when nitrous oxide was added and so maintenance was continued with 100% oxygen and sevoflurane was changed to a high percentage of isoflurane. Ventilation was assisted manually and with the application of continuous positive airway pressure via the circle system. The position of the nasotracheal tube was checked visually and with auscultation of the chest. There were no signs of bronchospasm.

To achieve a satisfactory level of anaesthesia, three boluses of propofol 50 mg were required, and two additional increments of midazolam 1 mg. Ketorolac 10 mg was also given and local anaesthetic infiltrated by the surgeon. The surgery was performed as quickly as possible. Despite the high concentration of volatile and intravenous anaesthetic, the blood pressure and pulse rate remained high at the pre-induction levels. The end- tidal carbon dioxide varied between 5 and 7 kPa. Also, during induction he seemed to have a short fit or convulsion. I thought that this might have been caused by the propofol, but this reaction was also seen during emergence from anaesthesia. There were no respiratory problems or laryngeal spasm following extubation. He recovered rapidly and the oxygen saturation was 100% breathing 5 $l.min^{-1}$ of oxygen via a Hudson face mask. No further analgesia was required. This was a memorable 'stormy' anaesthetic.

When I saw the patient postoperatively he said: 'How was it?' (as if he was expecting there to have been a problem).

I said; 'Actually you were very difficult to anaesthetise.'

He replied: 'Well if I tell you something it might incriminate me.'

I said: 'You may tell me anything in confidence.'

He replied: 'I smoked cannabis last night.' He denied taking any other drugs. I advised him to refrain from smoking cannabis for as long as possible before an anaesthetic and also emphasised the importance of giving doctors a full drug history. He was discharged from the Day Unit 3 h after the end of surgery.

I had no previous experience of knowingly anaesthetising patients who were habitual cannabis smokers. On discussing this with my colleagues, it was felt that perhaps the patient had also been taking 'class A' drugs. However, it could be said that all the responses seen were due to the effects of cannabis.

As a result of slow elimination, cannabinoids may be present in the tissues of users for weeks and may interact with a number of anaesthetic agents. Cannabis smoking is associated with an impairment of lung function, similar to that associated with tobacco smoking [1]. Cannabinoids have complex actions on seizure activity and exert both anticonvulsant and proconvulsant effects. They may also cause a variety of psychological effects such as anxiety, and physical signs including tachycardia [1]. It was fortuitous that I had given dexamethasone (to reduce any swelling caused by the surgery). Dexamethasone is also recommended as prophylaxis for cannabis smokers undergoing general anaesthesia [2].

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Blood transfusion in the critically ill

In their recent editorial, Goldhill and colleagues (Goldhill *et al*. *Anaesthesia* 2002; **57**: 527–9) conclude that a haemoglobin concentration down to 8 $g.dl^{-1}$ or lower is safe in the critically ill. Where is the evidence for this? Certainly, prospective randomised controlled studies in this area are limited. The authors quote three papers by Hebert and colleagues, which all reported results and subgroup analysis from the same Transfusion Requirements in Critical Care (TRICC) trial. In the TRICC trial [1], a restrictive transfusion strategy (haemoglobin target of 7.0–9.0 $g.dl^{-1}$ and transfusion trigger of 7.0 $g.dl^{-1}$) was found to be at least as effective, and possibly superior to, a liberal transfusion strategy (haemoglobin target of 10.0–12.0 $g.dl^{-1}$ and transfusion trigger of 10.0 $g.dl^{-1}$). However, the average haemoglobin in the restrictive group was 8.5 $g.dl^{-1}$. On the basis of this evidence, it is not possible to comment on the safety of a haemoglobin of 8 $g.dl^{-1}$ or less.

The authors also dismiss case reports in which anaemic patients with chronic obstructive pulmonary disease (COPD) requiring ventilation were successfully weaned after their haemoglobin was increased to at least 12 $g.dl^{-1}$ [2]. In support of their argument, the authors quote one of the subgroup analysis of the TRICC trial [3]. This subgroup analysis looked at all patients requiring mechanical ventilation and consisted of a heterogeneous group of critically ill patients and not a subgroup of patients with COPD.

We are not advocating liberal transfusion policies but feel that more evidence is required before conclusions can be made about the optimal haemoglobin level in critically ill patients.

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A reply

We are pleased that Drs Jefferson and Ball read our editorial. However, a re-reading will confirm that our conclusion was actually that the real and potential harm of a blood transfusion should be balanced against the evidence indicating that a haemoglobin (Hb) concentration down to 8 $g.dl^{-1}$ or lower is safe in the critically ill.

If they care to delve into some of the 55 references, they will find plenty of evidence on the limits of acute anaemia but little or none that a Hb level of 8 $g.dl^{-1}$ or even lower causes serious harm. Our editorial acknowledges the limitations in Hebert's paper, but it is the best critical care evidence currently available.

We did state that the subgroup analysis of the TRICC trial did not confirm the five case reports of successful weaning of COPD patients after transfusion. This does not imply a dismissal of the case reports. However, objective evidence in the form of a larger controlled study of the benefits of transfusion in facilitating weaning of COPD patients has not been forthcoming even though 4 years have elapsed since Schonofer *et al*. published their article. We do not know, or suggest, an optimal haemoglobin concentration for the critically ill. Our article focused on the balance between the advantages of a higher Hb and the risks of an allogenic blood transfusion.

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Not so free flowing

Devices to prevent backflow of blood or solutions in intravenous lines have been readily available for some time. In our hospital we have available such a one-way valve device, namely, the R-Lock® (Article-No. MF1543), manufactured by Impromediform Gmbh (Fig. 6). We decided to ascertain to what extent flow rates were affected by using this device when administering intravenous fluids.

We attached a standard blood giving set (Baxter Ref RMC2071B), to a 500-ml bag of 0.9% saline. This was suspended from a height of exactly 1 m.

Figure 6

To the end of the giving set we initially attached a 16G Vygon Biovalve® cannula. We then measured the volume of 0.9% saline that passed through this system over a 2-min period. Subsequently, we placed a R-Lock® device between the cannula and the giving set and repeated the same measurement.

With no R-Lock® device attached, a flow rate of 163.5 $ml.min^{-1}$ was recorded. However, when the R-Lock® device was attached, a flow rate of only 56 $ml.min^{-1}$ was achieved. This equates to a reduction in flow rate of 66%. Neither we, nor many of our colleagues realised how significantly this particular device impairs flow through an otherwise satisfactory fluid giving set. As our study has shown, the R-Lock effectively converts a grey (16G) cannula to a pink (20G) one with regard to its flow characteristics.

We feel that these devices should therefore be used with caution, especially in cases where there is potential need for significant fluid replacement.

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Referring to earlier work would have refined conclusions

The authors (Conti *et al. Anaesthesia* 2002; **57**: 540–3) omitted to cite our study on the effect of small doses of alfentanil on breathing [1] because (personal communication) our patients were anaesthetised but theirs were awake. This is true, but our findings might have helped them to define the effect better. They measured the breathing at 5-min intervals after injection of alfentanil 10 $\mu g.kg^{-1}$. In our anaesthetised patients, the mean time to peak effect (i.e. to the longest expiratory time) after 2–5 $\mu g.kg^{-1}$ was 82 s, and the mean half-life of effect was 146 s. By 5 min, the effect would have diminished.

This could explain why Conti *et al.* saw a reduced minute ventilation, but could not find significant changes in either tidal volume or respiratory rate; any change in minute ventilation can occur only if one or other or both of these variables change. By referring back to our work, and noting that the ventilatory rate in their patients was less than baseline at 5 min, even if not statistically significant, they could have presumed that alfentanil affects ventilatory rate.

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Two-stripe Tuohy, when too deep?

The appearance in our unit of unusually marked Tuohy needles has prompted a review of the history of the markings on epidural needle shafts and catheters and of the origins of the technique of estimating the length of catheter inserted into the epidural space.

We have encountered four 16G Portex Tuohy needles with 2-cm gradations dividing the 8-cm shaft in four, instead of the usual 3-cm blank tip and five 1-cm divisions (Fig. 7). The needle tip, shaft length and catheter were normal. Each of the needles was used successfully for epidural catheter insertion. Identification of the epidural space, estimation of its depth, and subsequent estimation of length of catheter insertion were not difficult in any of the four cases. There were no instances of dural puncture.

The manufacturer has been unable to explain the manner by which the chemical etching process went awry. To our knowledge, this style of marking is not standard for any other country. Different needle lengths and styles of marking exist currently (e.g. the B/Braun range of epidural needles is marked to the tip in 1-cm gradations) but none employs markings other than 1 cm.

Cheng in 1958 was the first to report an epidural needle with depth markings, which he manufactured from a conventional 16G spinal needle [1]. The shaft was 8 cm long and graduated in centimetres and millimetres, allowing measurement of the depth from skin to epidural space. He claimed to be able to estimate the length of catheter insertion within the epidural space, although it is unclear from his description whether the catheter he employed had markings. Lee, in 1960, described a Tuohy needle

Figure 7

with depth markings (4 cm blank tip and four 1-cm markings), and asserted a reduced incidence of dural puncture as its principal advantage [2]. In 1962, Lee described a catheter with a blind end, single side hole and graduations that made possible accurate measurement the depth of catheter insertion [3], although it was not until 1974 that Doughty formally described a method of precise estimation of depth of catheter insertion within the epidural space [4].

When encountering an unmarked or unusually marked epidural needle, one may have difficulty estimating the depth of needle and catheter insertion, and may have an increased risk of dural puncture. Several methods have been described to overcome these problems [5–7].

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