A role for presynaptic NMDA receptors in central sensitization in the spinal cord dorsal horn?

Editor,—Buerkle and colleagues report that intrathecal administration of remifentanil in rats that had received intraplantar formalin could wholly abolish nociceptive behaviour during phase 1 of the formalin test but was associated with only partial inhibition of glutamate release as measured by microdialysis. Intraplantar injection of formalin results in a biphasic behavioural response, the second phase of which is considered to represent NMDA receptor-mediated central sensitization. Phase 1 is characterized by paw flinching and licking corresponding to an acute barrage of small primary afferent fibre discharge and is followed, after a brief quiescent period, by a more protracted phase 2 period of similar behaviour associated with a lower level of tonic afferent activity. Buerkle and colleagues contend that “supramaximal doses of intrathecal remifentanil sufficient to inhibit phase 1 behaviour still permit sufficient glutamate release to permit spinal facilitation” via presumed activation of a post-synaptic NMDA receptor during phase 1. Their data merit further discussion in the light of recent anatomical and functional demonstrations of presynaptic NMDA receptors in the spinal cord dorsal horn.5 6

The spinal action of remifentanil is mediated by μ-opioid receptors at both pre and postsynaptic loci within the dorsal horn, and as shown in this and previous studies, opioid administration can abolish both phase 1 and phase 2 of the formalin test. However, where opioid effects are limited purely to phase 1 by intrathecal naloxone8 9 or by the use of remifentanil,10 only a partial suppression or delay of phase 2 behaviour has been reported despite abolition of the phase 1 behavioural response. Therefore, sensitization still occurs during phase 1 of the formalin test, and crucially, such sensitization may occur despite electrophysiological evidence indicating that opioid suppression of dorsal horn neurone firing during phase 1 may be virtually complete.11 This “remifentanil-resistant” component of central sensitization of the current study was considered by Buerkle and colleagues to be a consequence of incomplete opioid-mediated inhibition of glutamate release from primary afferent or interneurone nerve terminals during phase 1, similar to that reported previously with systemic morphine.12

If the initiation of central sensitization underlying phase 2 of the formalin test involves postsynaptic NMDA receptor activation, it would be expected to require sustained depolarization of nociceptive neurones in the dorsal horn. How can this be reconciled with the behavioural evidence of suppression of postsynaptic activity in this and other studies, and electrophysiological evidence of suppression of dorsal horn neuronal activation during phase 1 of the formalin test?10 12

Functional presynaptic NMDA receptors which potentiate the release of C-fibre primary afferent transmitters have been recently identified within the dorsal horn.13 14 It may be hypothesized that the enhanced release of glutamate during phase 1 demonstrated by Buerkle and colleagues may activate such presynaptic receptors which are depolarized by virtue of phase 1 afferent activity. This activation of presynaptic NMDA receptors would initiate a long-lasting Ca2+-mediated enhancement of primary afferent transmitter release and increase the gain of C-fibre synaptic transmission in the dorsal horn during phase 2. Fundamentally, presynaptic sensitization could occur despite opioid suppression of postsynaptic neuronal activity and would only become manifest behaviourally after opioid suppression of postsynaptic neuronal activity within the dorsal horn was removed.

Support for this hypothesis remains circumstantial in the absence of a selective antagonist of presynaptic NMDA receptors. However:

1. Yashpal and colleagues report that intrathecal lidoaine given after phase 1 of the formalin test suppressed Fos expression in the dorsal horn but did not suppress nociceptive behaviour in phase 2.15 Their data indicate that behavioural evidence of central sensitization persists despite suppression of an index of postsynaptic neuronal activation—again implicating a possible presynaptic component of sensitization.

2. There is evidence that presynaptic NMDA receptors may differ structurally from postsynaptic NMDA receptors and be insensitive to polyamines.16 17 Polyamines such as spermine which act as allosteric modulators of NMDA receptor function18 are not analgesic in the formalin test when administered intrathecally19 which may reflect lack of inhibition of presynaptic NMDA receptor function.

Thus central sensitization may occur via NMDA receptors on C-fibre primary afferent terminals despite effective block of postsynaptic responses by opioids. Such a possibility of a previously unrecognized component of spinal sensitization reinforces our previously expressed view20 that spinal presynaptic functional anaesthetics or analgesics (including μ-opioids) do not adequately inhibit the longer term consequences of painful stimulation.

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Editor,—We appreciate the comments about our study on the intrathecal injection of remifentanil, which inhibited the nociceptive behavioural responses evoked by formalin administration into the hind paw of the rat, but did not abolish the concomitant glutamate release at the spinal cord level, as assessed by the in vivo spinal microdialysis of cerebrospinal fluid in this model.1 The initial response of the formalin test (phase 1) is caused by activation of peripheral nociceptors and is followed by a second phase attributed to ongoing activity in primary afferents and increased sensitivity of the dorsal horn neurones.2,3 The latter effect is thought to result from glutamate-mediated N-methyl-D-aspartate receptor activation. Glutamate has been shown to be the major excitatory amino acid neurotransmitter in the central nervous system, playing a key role in central sensitization and spinal pain transduction mechanisms.4 We agree that the initiation of central sensitization may also involve presynaptic NMDA-receptor activation, as was suggested by Liu and colleagues.5,6 Thus the observed effects may be caused by a presynaptic sensitization, which becomes behaviourally apparent because of the short-lasting effects of the esterase-metabolized, ultra-short acting μ-opioid remifentanil or, which might be caused by the distinct differences in the temporal activation of pre- and postsynaptic NMDA-receptor activation. This hypothesis may be further supported by studies from Taylor and colleagues and Abbadic and colleagues,7 who reported that central sensitization mechanisms during phase 1 do not influence the magnitude of phase 2 after systemic delivery of remifentanil and that there exist regional differences of c-fos activation within the spinal cord laminae during complete suppression of phase 1 behaviour by remifentanil. In conclusion, we contend that in lieu of circumstantial evidence for the role of presynaptic NMDA receptors in central sensitization processes, further studies elucidating the role of pre- versus postsynaptic NMDA activation for central sensitization are warranted.

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Morbidity after day-case tonsillectomy in children

Editor,—Splinter and Rhine1 quote an incidence of vomiting after day-case tonsillectomy in children of 40%, despite antiemetic treatment. Their comment that this is unacceptably high is indeed true. I believe this has more to do with the anaesthetic technique than the surgical procedure itself. The incidence of nausea and vomiting after day-case tonsillectomy in 280 children in Salisbury, is under 5%. On a recent telephone questionnaire, carried out the day after surgery, the score, on a scale of 1–10 as recorded by parents, was a mean of 0.9 for nausea. The anaesthetic procedure adopted for this surgery involves propofol or sevoflurane induction, the insertion of a laryngeal mask, spontaneous ventilation with enfuran or the ondansetron 0.1 mg given i.v. after induction. Analgesia is provided with morphine and non-steroidal analgesics. Patients are encouraged to drink up to three hours before surgery and as soon as possible afterwards, usually within an hour. I.V. fluids are not administered.

I wonder if the very high incidence of vomiting often quoted is partly because of laryngeal intubation. Williams and Bailey studied the incidence of tracheal soiling by blood after intubation compared with a laryngeal mask airway for tonsillectomy.2 Over 50% of children whose tracheas had been intubated had blood in their lower airway compared with none in the laryngeal mask airway group. I would suggest that blood can just as easily pass into the stomach as into the trachea during tonsillectomy, when the airway is maintained with a tracheal tube. In contrast, a laryngeal mask will provide substantial protection by obstructing the oesophagus. I suspect that it is the swallowed blood that gives rise to the nausea and vomiting. I also believe that the use of halothane and intermittent positive pressure ventilation does little to reduce the incidence of nausea and vomiting in this group of patients.

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Editor,—Thank you for giving us the opportunity to respond to Dr Church’s letter.

The specific aetiology of vomiting after tonsillectomy is unknown and is presumably multifactorial. Factors known to contribute are the anaesthetic agents used, opioid administration, surgical technique and perioperative fluid management. Dr Church reports an incidence of nausea and vomiting after day case tonsillectomy of less than 5%. To the best of our knowledge, a reported incidence of less than 40% is unusual if a prophylactic antiemetic has not been used. It is difficult to explain this large difference as the anaesthetic technique described by Dr Church is not significantly different from that used by ourselves and others who have reported on this problem. The differences reported by Dr Church are the routine use of a laryngeal mask airway, the non-administration of perioperative intravenous fluids,
and the use of enflurane. Swallowed blood has always been known to be a significant contributing factor to postoperative vomiting following tonsillectomy. This becomes a significant factor immediately following surgery, when children usually swallow any blood present in the pharynx. The use of a laryngeal mask is obviously not a factor during the postoperative period. The hydration of patients has been shown to reduce the incidence of nausea and vomiting significantly during strabismus surgery, another procedure with significant nausea and vomiting postoperatively. It is difficult to perceive how its omission during tonsillectomy could contribute to reduction in the incidence of nausea and vomiting.

The incidence of nausea and vomiting following the use of halothane when compared to enflurane is, to the best of our knowledge, similar (i.e. approximately 15%-20%).

Given that nausea and vomiting following day case tonsillectomy are potentially serious problems, we would suggest that Dr Church’s technique should be investigated under controlled conditions and the results reported.

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Differential effects of nitrous oxide and propofol on myogenic motor-evoked responses

Editor,—Ubags and colleagues1 have made a further advance in the search for suitable anaesthesia techniques to permit motor-evoked potential monitoring during spinal surgery, by confirming the benefit of double over single-pulse stimulation. Despite that, responses were abolished by propofol in 2 of 12 patients who then required anaesthesia consisting only of sufentanil and 50% nitrous oxide with a neuromuscular blocking drug. The apparent absence of recall in their series will not entirely reassure many anaesthetists who would worry about awareness using this technique.

We would like to draw their attention to our study of transcranial single-pulse magnetic motor-evoked potentials in which anaesthesia was ensured by a methohexital infusion at up to 100 μg kg⁻¹ min⁻¹. Since that study was completed, the method has been simplified by die omission of ketamine and the inspired nitrous oxide concentration is usually maintained at around 0.25%. Eight patients have been monitored successfully with methohexital and alfentanil infusions, and a further four with methohexital and remifentanil at 1 μg kg⁻¹ min⁻¹, which allows for awakening within 20 min of stopping the infusions.

The transcranial magnetic response (usually tibialis anterior) after spinal column injury varies, depending upon the degree of neurological compromise, but typically lies within the range 10–300 μV under methohexital/remifentanil anaesthesia. Even better responses would be obtained using this technique with a double-pulse magnetic or electrical stimulus.

The authors omitted mention of premedication in their patients so it is worth emphasizing that a hypnotic agent such as a benzodiazepine is best avoided for motor-evoked potentials.

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Editor,—We thank Dr Watt and colleagues for their interest in our paper. The authors state that benzodiazepines should be avoided when motor-evoked potential monitoring is indicated. We feel the use of a benzodiazepine premedication is necessary to reduce the likelihood of awareness, especially when a nitrous oxide/opioid anaesthetic technique is used. All our patients received diazepam 10 mg orally, 1 h before surgery. We accidentally omitted mention of premedication in our paper.

The authors’ success with motor-evoked potential monitoring during anaesthesia with methohexital and very low concentrations of nitrous oxide is certainly promising. It is another indicator that tc-MEP monitoring during transcranial stimuli is possible, provided that special anaesthetic regimens are used. However, the application of multi-pulse stimulation paradigms obviates the need to avoid standard anaesthetic drugs. Multi-pulse stimulation has been shown to enable tc-MEP monitoring during propofol/opioid anaesthesia2 and isoflurane anaesthesia.3 Recently, specifically designed multi-pulse stimulators, capable of delivering up to four (magnetic) or 10 (electrical) successive transcranial stimuli have become commercially available. We believe that the introduction of these stimulators into clinical practice will result in a more widespread acceptance of motor-evoked potential monitoring.

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Anaesthesia for laparoscopic cholecystectomy in a patient with Eisenmenger’s syndrome

Editor,—We read with interest the article of Sammut and Pas1 describing the management of a patient with Eisenmenger’s syndrome for laparoscopic cholecystectomy and would like to make the following comments.

The physiological changes that occur with laparoscopy need to be considered carefully in these patients. The balance between pulmonary and systemic vascular resistance is critical in determining the degree of right to left shunt flow.2 One of the consistent haemodynamic changes observed with laparoscopy is an increase in systemic vascular resistance,3 which is helpful in this group of patients. While maintenance of systemic vascular resistance is important, one must also avoid rises in pulmonary vascular resistance, which will increase right to left shunt flow and worsen systemic hypoxia. Hypoventilation from sedative premedication or postoperative analgesia, rising arterial carbon dioxide from absorbed gas at laparoscopy, and increased pulmonary airway pressure as intra-abdominal pressure increases, all contribute to increases in pulmonary vascular resistance.

We recently had a 39-yr-old man with complex congenital heart disease and Eisenmenger’s syndrome present for laparoscopic cholecystectomy. This patient had uncorrected tetralogy of Fallot with a right-sided aortic arch, a large patent ductus arteriosus and right to left shunting via the ductus. He had had recurrent ventricular tachycardia which was treated with amiodarone; polycythaemia requiring regular venesection; previous bacterial endocarditis; and ischaemic heart disease, evidenced by angina following venesection on several occasions. Other medical problems included hypothyroidism and chronic schizophrenia with mild mental retardation. He became dyspnoeic on mild exertion and had seven episodes of cholecystitis in the four weeks before admission for cholecystectomy. On examination he was centrally cyanosed with marked clubbing and splinter haemorrhages. His pulse rate was 60 min⁻¹ and regular, arterial pressure 120/60. His jugular venous pressure was not elevated, he had pronounced right ventricular heave and a holosystolic murmur with a loud second heart sound. Most recent echocardiography revealed a mildly dilated left ventricle with moderate decrease in function; an overriding aorta (50% left ventricle and 50% right ventricle)
with a 2 cm ventricular septal defect and bidirectional shunting: a large right-sided patent ductus arteriosus with predominately right to left shunting; right ventricular hypertrophy; and a moderately dilated right heart with estimated right ventricular systolic pres-
ers of 50 mm Hg.

During general anaesthesia for his laparoscopic cholecystectomy the intra-abdominal pressure was restricted to less than 15 mm Hg; die patient’s systemic arterial pressure and oxygenation improved during die period of pneumoperitoneum without pharmacological intervention. During emergence from anaesthesia, die patient developed worsening hypoxia without changes in arterial pressure, heart rate or ventilation parameters. A visible increase in right ventricular heave developed, presumably reflecting increased pulmonary vascular resistance. Systemic oxygenation improved spontaneously over time as the depth of anaesthesia lightened and the trachea was extubated.

The same patient had a subsequent and unrelated admission for retinal detachment repair and during emergence from that anaesthetic, the same systemic hypoxia and pronounced right ventricular heave developed despite deep extubation and adequate spontaneous ventilation via a laryngeal mask airway. Again, oxygen saturation slowly improved as the patient awoke.

Changes in pulmonary vascular resistance need at least equal consideration as those of systemic vascular resistance in patients with Eisenmenger’s syndrome. A selective pulmonary vasodilator such as nitric oxide or inhaled prostacyclin may be a useful adjunct to the management of such cases especially in circumstances where systemic vascular resistance is already very high.

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3. Cunningham AJ, Turner J, Rosenbaum S, Rafferty T. Transoesophageal echocardiographic assessment of haemody-

Editor,—I read the article by Sammut and Paes1 with interest and agree that the theoretical risks of anaesthesia in patients with Eisenmenger’s syndrome (ES) are many and serious. I have anaes-
thetised a patient with ES for intracranial shunt.2 The absence of obvious cyanosis and clubbing in the case reported is surprising. It is clear that maintenance of systemic vascular resistance is a neces-
sary goal. It is worth mentioning that “fixed” pulmonary vascular resistance (PVR) precludes rapid adaptation to intraoperative haemodynamic changes and is usually unresponsive to pharma-
cological measures. The factors influencing PVR are important to know.

I remain unconvinced about “prophylactic” use of a norepi-
nephrine infusion, which is continued into die postoperative period. The authors stated that there is a correlation between systemic pressure and oxygen saturation. But the higher systemic venous oxygen saturation and decreased oxygen consumption with induction of anaesthesia and muscle paralysis contributes to maintenance of arterial oxygen saturation during the intraoperative period in patients with cyanotic congenital heart disease (CCHD).3 The authors did not mention any arterial blood-gas analysis. Peri-operative arterial blood-gas analysis is essential in a patient with ES undergoing laparoscopic cholecys-
tectomy. The increase in end-tidal carbon dioxide after pneumo-
peritoneum under-estimates Pao2, in patients with CCHD. Cardiorespiratory changes during laparoscopic cholecystectomy also increase the difference between end-tidal carbon dioxide and Pco2.4 Monitoring for gas embolism in the presence of right to left shunt during laparoscopic surgery is necessary.

I believe conventional open cholecystectomy is advisable in the presence of ES. Mechanical effects of pneumoperitoneum (decreased cardiac index, impaired venous return, decreased FRC, increased V/Q mismatch and pleural pressure), hypercarbia and acidosis, the possibility of gaseous embolism and thrombotic complications are major side effects of laparoscopic cholecystec-
tomy. These risks outweigh the benefits in a patient with ES. Slow persistent blood loss from die raw gall bladder bed as a cause of a nearly 50% decrease of haemoglobin concentration is difficult to understand. Were there any coagulation abnormalities?

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1. Sammut AS, Paes ML. Anaesthesia for laparoscopic chole-
4. Wahba RWM, Beique F, Kleinman SJ. Cardiopulmonary func-
In their letter, Drs Kruger and Moran suggest that pharmacological manipulation of the pulmonary circulation would be of value in these patients. Unfortunately, i.v. pulmonary vasodilators are contraindicated as more often than not they dilate the systemic vasculature as well and would clearly worsen the situation. Although both inhaled nitric oxide and prostacyclin have been shown to selectively reduce pulmonary vascular resistance in responsive patients, their role, in the conduct of anaesthesia for non-cardiac surgery in patients with Eisenmenger’s syndrome, remains uncertain and cannot be recommended as agents of first choice.

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5. Goldman AP, Delius RE, Deanfield JE, Macrae DJ. Nitric oxide

The strain of Staph. aureus cultured from the abscess was also found on the tip of the extradural catheter, in secretions from the wound and cannot be recommended as agents of first choice.

This case illustrates the potential risk of serious nosocomial infection when an extradural catheter is inserted in a patient with known multiple colonizations of pathogenic bacteria, as is often the case in an ICU patient. It is at least in part related to the reduced immunocompetence that follows major trauma. This observation raises a question about the optimal management of extradural catheters under these circumstances. Is there, for example, any benefit from tunnelling such catheters, as seems to be the case with central venous catheters? A retrospective study with a rather small sample size does not favour this regimen. The present case has also made us question the prevalence figures for serious spinal-space infections after extradural catheterization. Is it really as low as < 1,500? Kvalsvik and Givvold, during 1 year, had two severely traumatized patients who developed extradural abscesses during extradural catheterizations of 11–14 days’ duration. This would imply that the number of unrecorded cases may be substantial.

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Editor,—We think that the case described by Andersen and Sörensen well emphasizes the importance of a thorough consideration of the potential benefits and risks of a central neuraxial regional anaesthetic procedure in the potentially or overtly infected patient. Further aspects of the controversies about regional anaesthesia in the potentially infected patient are highlighted in the American Society of Regional Anaesthesia (ASRA) newsletter, February 1998, pp 4–6. A recent article in the official journal of the Swedish Medical Association1 underlines the necessity of meticulous hygiene when performing spinal/extradural catheterization, with special emphasis on the mandatory use of good-quality face masks to avoid iatrogenic meningitis. This article also focuses upon the difficulties of reaching the correct diagnosis in certain patients with spinal-space infections. This supports the suspicion of Andersen and Sörensen that the prevalence of these infections is underestimated. The type of dressing (water-impermeable or not) used by the anaesthesiologist to achieve good fixation of the extradural catheter might also be important.

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Use of laryngeal mask during tonsillectomy

Editor,—The laryngeal mask is becoming widely used for airway maintenance during tonsillectomy. The following case report documents a complication of such use which is potentially serious.
A healthy 17-yr-old male was scheduled for tonsillectomy. Anaesthesia was induced with propofol 200 mg and fentanyl 100 µg and the airway was maintained using a size 4 flexible laryngeal mask airway for spontaneous ventilation. After insertion of a Boyle-Davis gag, the airway was assessed with reference to the capnograph and chest movement, and found to be unobstructed. Coincident with removal of the first tonsil however, the airway became totally obstructed, as indicated by cessation of carbon dioxide emission, a tracheal tug with paradoxical respiration and development of arterial hypoxaemia. A quick assessment of the placement of the laryngeal mask revealed that the pilot tubing had become entrapped and severed by the snare used to remove the tonsil, and the cuff of the laryngeal mask had therefore totally deflated. In the face of brisk bleeding from the tonsillar bed, the mask was withdrawn following suction and a new mask inserted. This passed easily and the airway was re-established. At the end of surgery, the mask was withdrawn and direct laryngoscopy revealed only minimal soiling of the larynx and trachea with blood. Subsequent recovery from anaesthesia was uneventful.

Recent studies have suggested that the laryngeal mask airway has advantages over formal tracheal intubation for maintenance of the airway during adenotonsillectomy, with good surgical access and no increase in the incidence of aspiration of blood into the trachea.1 Included in the complications recorded are laryngeal spasm after placement of the mask; obstruction of the airway after insertion of the Boyle-Davis gag, which has a reported incidence in the region of 20%2 3; and occasional airway problems during recovery and removal of the mask.4 However, there does not seem to be a report of an airway complication during surgery resulting from inadvertent cutting of the pilot tubing by the surgeon.

The reason that this complication is more likely with the laryngeal mask is that, whereas the insertion of the pilot tubing into a 9 mm RAE profile tracheal tube (Malinkrodt) is approximately 4 cm behind the top incisors, the insertion of the pilot tubing of all laryngeal masks is into the cuff itself. This increases its predisposition to loop behind the tonsil where it is hidden from view during surgery. It is now my practice to tape the pilot balloon to the main tubing well proximal to its point of insertion, and not only to assess respiration but also to visually inspect the airway intra- orally after insertion of the Boyle-Davis gag.

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Method for reinserting the connector after cutting paediatric tubes

Editor,—The letter by Preis and Preis1 has suggested a practical solution to the potentially fatal problem of damage to the connector while inserting it into the cut end of a paediatric tracheal tube. We would like to report the method we practise to avoid damage to the connector or the tracheal tube itself.

The tracheal tube is cut so as to make a short bevel. The connector is introduced at an angle to the tube, that is, directly facing the bevel (fig. 1). The bevel provides a wider area for initial insertion of the connector. Lubrication by a dab of soluble jelly at the tip of the connector facilitates its insertion. The connector is now brought into the longitudinal axis of the tube while maintaining a firm pressure when it gently slides into the tube (fig. 2). The last manoeuvre facilitates the insertion of the connector without damaging either the tracheal tube or the connector. As a result the connector can be inserted further into the tube. Visual inspection down the long axis of the tube detects any damage to the tube or the connector.

This method eliminates the need for using a special paediatric airway adaptor or any dilatational instrument for its purpose.

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1. Preis CA, Preis IS. Obstruction of the lumen of a plastic 15 mm connector inserted into a cut paediatric tube—Is it possible to avoid this damage? British Journal of Anaesthesia 1997; 79: 692.
more insertion length is lost. The key issue, of course, is that the connector be inserted far enough into the tube so that the connector/tube bond, which is guaranteed by our technique,1 is not compromised.

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1. Preis CA, Preis IS. Obstruction of the lumen of a plastic 15 mm connector inserted into a cut paediatric tube—Is it possible to avoid this damage? British Journal of Anaesthesia 1997; 79: 692.

Figure 1 Connectors inserted into 3.5 mm internal diameter paediatric tubes cut at: (A) an angle of about 45°, (B) straight across, (C) an angle of about 60°.