Correspondence

Posterior subgluteal approach to block the sciatic nerve: the controversy of ‘which came first, the chicken or the egg’ is alive

EDITOR:
We read the article by Di Benedetto and colleagues [1] describing the new approach to block the sciatic nerve with tremendous interest mixed with a sense of déjà vu. The technique claimed to be safe, easy to perform and causing minimal discomfort to the patient; needs to be lauded and authors complimented for the innovation.

However, we have some points to make. First, the dose of oral diazepam for premedication; ‘30 mg, 30 min before placing the block’. Thirty milligrams is a large dose considering the long half-life of diazepam and particularly because some of the patients in their study may have been in the late middle age or even beyond 60 yr (as suggested by the mean age of 44 ± 18 yr). Also, the 30 min time interval between the oral administration and placing the block is too short.

Secondly, the authors have very modestly proposed that ‘further studies are required to compare this new approach to other proximal approaches to the sciatic nerve, particularly the classical posterior approach of Labat modified by Winnie’. But haven’t the authors already done that and even published it nearly a year ago [2]? In fact, large chunks of text have been lifted ad verbatim from that article while the remaining matter has been culled to give it a different look. The only difference besides the number of patients in the subgluteus group in the earlier study [2] is the dose of oral diazepam, 10 mg, which is more acceptable than 30 mg. Why a three-fold increase in the dose of diazepam was needed is difficult to understand (unless it is a printing error). What has disappointed us the most is the fact that the authors have just concealed their earlier study [2], and we believe that even the publishers of the European Journal of Anaesthesiology would not be aware of it. It also needs to be clarified if the patients in subgluteus group in the earlier study were included in the present study. We leave it to the publishers to decide whether it is a case of duplicate publication?

In describing and proposing further studies after conducting and reporting comparative study with the commonly performed technique and even the continuous subgluteus nerve block [3], the authors surely have given a new look to the controversy: which came first, the chicken or the egg?

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References
A reply

EDITOR:
We all know that the sciatic nerve can be blocked at several levels along the so-called ‘sciatic line’, and a variety of approaches have been described to block the nerve along this line, with little innovation one from another. However, although we thank Drs Singh and Sharma for their interest [1] in our article on a subgluteal approach to the sciatic nerve [2], their suggestion of duplicate publication seems somewhat unwarranted for a research group that has published in the field of regional anaesthesia for over 15 yr. The first point raised concerns about the premedication dose of diazepam that we used. We would also be concerned giving diazepam 30 mg to elderly patients, and this is indeed our own typographical mistake, since they received diazepam 10 mg orally 30 min before block placement, as is routine in our department. We can only apologize for the error and thank Drs Singh and Sharma for pointing it out.

We do not agree that this is a case of duplicate publication but is somewhat related to the unpredictably different editorial history of different papers, coming from different studies produced by the same research group. Let us make just a few observations to clarify this. First of all, Drs Singh and Sharma should recognize that it is reasonable that a research group to focus its interest in one restricted area. Accordingly, it is easy to forecast that most of the publications produced by this research group will be focused on very similar topics. The scientific production of this group will vary little from paper to paper, but this does not mean that the authors are duplicating studies. Second, we must take into consideration the different editorial history of each manuscript, as well as the speed of the editorial process, which may change editorial history of each manuscript, as well as the speed of the editorial process, which may change considerably. The upshot in our case was that our ‘second’ randomized study was published in 2001, while our ‘first’ observational study was published in 2002. We are happy to open our records for inspection to anyone with a legitimate interest in this matter.

On the other hand, we consider that using ad verbatim the same description of the technique and method while writing the ‘second’ randomized study to be acceptable; we were always the same anaesthesiologists placing the same type of block for the same type of patients (even though they were different patients!). Our main fault was that we did not correct the proofs of the observational study [2] to include the paper about the second randomized study [5]; this was partly related to the ‘chicken and egg’ problem, and partly to the fact that extensive rewriting of the proofs at a late stage would be necessary – for this we can only apologize to the editorial team and readership of the European Journal of Anaesthesiology. At the same time we also thank Drs Singh and Sharma for their accuracy and constructive criticism [1].

To answer Drs Singh and Sharma’s joke, although we all know that the egg came from the chicken, it happens sometime in life that we first see the egg, and then only after notice the chicken: this does not reduce the clinical relevance of either the chicken or the egg.

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Editor:
The analgesic effects of µ-agonists have never been separated from their depressant effect on spontaneous
ventilation. Breath intervals have been shown to be a reproducible method of measuring the time-course
of opioid effect in anaesthetized patients breathing spontaneously \[1,2\]. After a single dose of intravenous
(i.v.) opioid, a plot of breath interval (observed – baseline) against time displays a curve very similar to
the concentration–time curve after a drug that requires absorption to the circulation (Fig. 1). There is a rise to
peak effect because of delays in the uptake of opioid to the brain and the time taken to elicit an effect at the
effect site. The duration of the altered breath interval observed after a patient has received an opioid corre-
sponds to the duration of dynamic opioid effect. Relief from pain cannot be measured during anaesthesia, has
considerable between-patient variation and can be recorded only intermittently. Breath intervals provide
a continuous measure of opioid dynamic effect during anaesthesia in patients breathing spontaneously. We
sought to discover if measurement of breath intervals provided a sufficiently sensitive method to distinguish
the different duration of action of fentanyl and alfentanil after single i.v. doses.

Ethics Committee approval and informed written
consent were obtained from patients admitted for knee replacement surgery. Patients received temazepam
20 mg before anaesthesia was induced with propofol 2–3 mg kg\(^{-1}\) i.v., and maintained with isoflurane
0.8% and nitrous oxide 67% in oxygen through a laryngeal mask. Sciatic and three-in-one femoral nerve
blocks were then performed using a nerve stimulator and bupivacaine 0.375% 20 mL into each nerve.

The analogue output of the carbon dioxide (CO\(_2\))
concentration from a Capnomac Ultima\(^\text{\texttrademark}\) (Datex,
Helsinki, Finland) was fed to an analogue-to-digital
converter sampling at 100 Hz. The time between
corresponding points on the down stroke of the
CO\(_2\)–time waveform on successive waves was recorded
as the breath interval. Patients were randomized
to receive either fentanyl 0.75 µg kg\(^{-1}\) or alfentanil
2.25 µg kg\(^{-1}\) by single injection i.v. at zero time.

Breath interval data were plotted against time (Fig. 1).
From the latter two-thirds of the elimination phase,
the slope of the regression line through the natural log-
arithm of breath interval – baseline gave the elim-
ation rate constant, \(k\). From this, the dynamic
elimination half-life of the opioid effect was calculated
using \(t_{1/2} = \ln(2)/k\). The mean effect time \([3]\) of fen-
tanyl in the body was also calculated from the ratio of
the area under the first moment curve (AUMC) to the
AUC. Both AUC and AUMC were extrapolated to
zero by dividing the last datum point by \(k\). The data
were analysed using MKMODEL Software \([4]\).

Statistical analysis was by ANOVA. Significance was
accepted when \(P < 0.05\).

Thirty-four patients were studied. Sixteen were
removed from the study because of inadequate block

References
1. Singh B, Sharma P. Posterior subgluteal approach to block
the sciatic nerve: the controversy of ‘which came first, the
chicken or the egg’ is alive. Eur J Anaesthesiol 2003; 20: 496.
subgluteal approach to block the sciatic nerve: description
of the technique and initial clinical experiences. Eur J
Fanelli G. A new posterior approach to the sciatic nerve
block: a prospective, randomized comparison with the clas-


Breath interval as a continuous measure of opioid effects of intravenous fentanyl and alfentanil

Figure 1.
Serial ‘difference in breath interval’ measurements for two patients
given fentanyl (●) or alfentanil (▲) i.v. at 0 min. Breath intervals
are shown after subtraction of baseline values.
(n = 8), respiratory depression (6), operation changed (1) and computer failure (1) – leaving nine patients in each group. There were no differences between fentanyl (F) and alfentanil (A) with respect to weight (mean ± SD): F, 66 (±8); A, 77 (±18) kg; height: F, 166 (±8); A, 165 (±11) cm; age: F, 68 (±17) (range 33–86); A, 61 (±19) (33–80) yr; or time from the induction of anaesthesia to the opioid dose i.v.: F, 32 (±14); A, 35 (±15) min. There were four male patients in each group.

Nine patients received fentanyl 0.71 (0.09) µg kg\(^{-1}\) and nine patients alfentanil 2.3 (0.2) µg kg\(^{-1}\). The opioid dynamic effects were significantly different for fentanyl and alfentanil: rate constant of elimination (k): F, 0.0628 (0.0405); A, 0.21 (0.133) min\(^{-1}\), P = 0.009; elimination half-life: F, 15.2 (8.56); A, 5.04 (2.91) min, P = 0.004; mean effect residence time (MRT): F, 23.7 (9.3); A, 7.0 (3.4) min, P < 0.001.

Baseline and peak values for breath intervals and end-tidal CO\(_2\) and the time from opioid injection are given in Table 1. There were no differences between the time to peak values of breath interval and CO\(_2\) for fentanyl (breath interval – CO\(_2\): −0.71 (−2.8 to 1.4) (mean (95% CI)) min, P = 0.48), or alfentanil (breath interval – CO\(_2\): −0.79 (−2.2 to 0.6) min, P = 0.25).

Breath intervals have proved sufficiently sensitive to distinguish the time-course of dynamic effects of fentanyl and alfentanil. Results obtained show a difference in both the mean residence time of fentanyl and alfentanil at the effect site, and the elimination half-life of the two drugs.

The kinetic and dynamic effects of fentanyl and alfentanil have been modelled from plasma drug concentrations and the EEG [5]. The times to EEG peak effects were 5 min for fentanyl and 1.5 min for alfentanil. These are similar to the times to peak effect by breath interval in this study of 5.3 (1.2) min for fentanyl and 1.9 (1.2) min for alfentanil (Table 1).

Modelling kinetic data to obtain the effect site concentrations, the peak effect site concentrations obtained occurred after 1.4 min for alfentanil and 3.6 min for fentanyl after single i.v. injections [6]. The results were similar to the dynamic peak effects measured in this study using breath interval.

The EEG simulation also produced k\(_{\text{eo}}\), a rate constant of equilibration with the effect site [5]. k\(_{\text{eo}}\) for fentanyl was 0.11 min\(^{-1}\), which was lower than that of alfentanil, 0.63 min\(^{-1}\). The lower k\(_{\text{eo}}\) for fentanyl damps the rise in fentanyl concentrations at the effect site when plasma fentanyl concentrations are at a peak. This may explain why doses of fentanyl in this study that were only one-third of doses of alfentanil produced the same maximum end-tidal CO\(_2\) concentrations: fentanyl 7.7 (1.2)%, alfentanil 7.5 (1.2)% (Table 1). In volunteers whose respiratory effects were measured by their response to inhaled CO\(_2\) at intervals over 60 min, a dose of alfentanil 20 µg kg\(^{-1}\) was equivalent to fentanyl 2 µg kg\(^{-1}\). The doses of fentanyl 0.75 µg kg\(^{-1}\) and alfentanil 2.25 µg kg\(^{-1}\) in this study were derived empirically during previous clinical practice, being doses which produced a measurable change in breath interval without apnoea, in anaesthetized patients breathing isoflurane and nitrous oxide. The higher k\(_{\text{eo}}\) of alfentanil will produce proportionately higher peak effect site concentrations than fentanyl after a single dose i.v. Limited by apnoea at the peak effect, we were obliged to use a relatively smaller dose of alfentanil. Equianalgesic doses would have produced apnoea in the alfentanil patients or a slight deviation of breath interval only in the fentanyl patients.

There were no differences between the times to peak breath interval and CO\(_2\) effect. This suggests both variables were affected by a common stimulus, the opioid, rather than by a change in one being consequent to the effect of the other.

Breath interval to measure opioid effect was successful during surgery only with an effective peripheral nerve block. Patients withdrawn because of respiratory

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**Table 1.** Baseline and peak breath interval and end-tidal carbon dioxide concentrations.

<table>
<thead>
<tr>
<th></th>
<th>Fentanyl (n = 9), mean (± SD), (95% CI)</th>
<th>Alfentanil (n = 9), mean (± SD), (95% CI)</th>
<th>Difference, mean (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline breath interval (s)</td>
<td>3.1 (1.0)</td>
<td>2.8 (0.5)</td>
<td>ns</td>
</tr>
<tr>
<td>Peak interval (s)</td>
<td>8.3 (6.4)</td>
<td>4.8 (0.8)</td>
<td>ns</td>
</tr>
<tr>
<td>Difference peak – baseline (s)</td>
<td>5.1 (5.6) (0.8–9.5); P = 0.026</td>
<td>1.9 (0.7) (1.4–2.5); P &lt; 0.0001</td>
<td>–</td>
</tr>
<tr>
<td>Time to peak breath interval (min)</td>
<td>5.3 (1.2)</td>
<td>1.9 (1.2)</td>
<td>3.4 (2.2–4.6); P &lt; 0.0001</td>
</tr>
<tr>
<td>Baseline CO(_2) (%)</td>
<td>6.5 (0.9)</td>
<td>6.6 (1.0)</td>
<td>ns</td>
</tr>
<tr>
<td>Peak CO(_2) (%)</td>
<td>7.7 (1.2)</td>
<td>7.5 (1.2)</td>
<td>ns</td>
</tr>
<tr>
<td>Difference peak – baseline CO(_2) (%)</td>
<td>1.2 (0.6) (0.7–1.7); P = 0.0003</td>
<td>0.8 (0.4) (0.5–1.2); P = 0.004</td>
<td>–</td>
</tr>
<tr>
<td>Time to peak CO(_2) (min)</td>
<td>6.0 (2.7)</td>
<td>2.7 (1.6)</td>
<td>3.3 (1.1–5.5); P = 0.0055</td>
</tr>
</tbody>
</table>

ns: Non-significant.
depression had elimination breath interval slopes no different from patients with complete data once spontaneous ventilation resumed. Dynamic effects measured by breath interval had a similar time-course to simulated effect site concentrations and peak opioid effects as reported previously.

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References


EDITOR:
Myelography and spinal anaesthesia are known to be a cause of intracranial hypotension resulting from liquorrhoea. However, spontaneous intracranial hypotension is a rare event and occasionally it is responsible for obstinate postural headache. We describe two patients with postural headache attributable to spontaneous intracranial hypotension, which was resolved by application of a cervical epidural blood patch with 10 mL autologous blood.

A 28-yr-old male in excellent health suddenly developed a headache accompanied by nausea. The headache started in the occipital region and worsened significantly on standing. Neurological examination revealed no abnormality and a lumbar puncture showed an opening pressure of zero cmH2O. Cranial magnetic resonance imaging, with the administration of gadorinium, revealed enhancement of the dura mater. Radionucleotide cisternography after lumbar puncture showed an extradural leak at the C6/C7 level. The patient remained in bed and received 2000 mL intravenous fluids for 7 days. Since the headache was not completely alleviated, an epidural blood patch – with 10 mL aseptic autologous blood at the C6/C7 interspace – was instituted. Nevertheless, the headache was not completely relieved, and further neck stiffness was noted after the blood patch. Because the headache was persistent for the following week, we suspected that previous lumbar punctures were responsible for the leakage of cerebrospinal fluid. Therefore, another epidural blood patch with 30 mL blood, at the L1/L2 interspace, was applied to close the extradural leak. This manoeuvre was effective and the headache and nausea abated. However, neck stiffness persisted for another two weeks, although it was not made worse by the second epidural blood patch. The patient was discharged five days after the second epidural blood patch.

Our second patient was a 41-yr-old male who was admitted to hospital because he experienced a two day history of orthostatic headache. He had no history of chronic headache, cranio cervical trauma or dural puncture. His headache had started suddenly in the occipital region when he extended his neck. Orthostatic posture worsened the symptoms, and induced nausea and severe pain confined the patient to bed. Neurological examinations revealed no abnormality and a lumbar puncture showed a depressed opening pressure (zero cmH2O). Cranial computed tomography showed an increased space between the brain and the cranium, and cranial magnetic resonance imaging, with gadolinium administration, revealed enhancement of the tentorium cerebelli.

Spontaneous intracranial hypotension treated with a cervical epidural blood patch

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Indium-111 radionucleotide cisternography demonstrated an extradural leak in the upper cervical region. From these results, we made a diagnosis of spontaneous cerebrospinal fluid leakage. The patient stayed in bed and received 3000 mL intravenous fluid per day for the following seven days. However, as the complaint continued for another one week, an epidural patch was planned and nine days after the onset of his headache, the patient received an epidural injection of 10 mL aseptic autologous blood at the C7/T1 interspace. His postural headache resolved completely by 24 h; however, he now complained of mild neck discomfort for several days. The patient was discharged three days after an epidural blood patch and has remained well.

Spontaneous postural headache was reported by Schaltenbrand [1], where it was called aliquorrea. This kind of headache usually persists for several months when patients receive only conservative treatment. While the exact aetiology for this syndrome has not yet been clarified, cerebrospinal fluid leakage through a thecal tear, reduced production of cerebrospinal fluid or by hyperabsorption of cerebrospinal fluid are listed as the pathogenesis [2]. An epidural blood patch is highly effective in the management of spontaneous intracranial hypotension [3] when a cerebrospinal fluid leak can be demonstrated by spinal imaging; the most probable mechanism for spontaneous intracranial hypotension is cerebrospinal fluid leakage through a thecal tear provoked by an external force or a meningeal diverticulum [3].

Previous reports described a relationship between a severe headache and postural change (hyperflexion of body) [3] or a strong blow to the head. In our case, severe headache developed immediately after retroflexion of the neck. What are the underlying systemic disorders? Four patients of spontaneous intracranial hypotension had findings suggestive of connective tissue disorders [4]. In addition, spontaneous intracranial hypotension has complicated Marfan’s syndrome, where arachnoid diverticula exist. On the other hand, spontaneous intracranial hypotension can occur in patients without any particular underlying disease. However, our cases are idiopathic since neither of the two patients had any special history of underlying disease.

Baker first reported a case of spontaneous intracranial hypotension where a headache was relieved by an epidural blood patch [5]. Recently, this treatment is recognized as a highly effective treatment for spontaneous intracranial hypotension [2,3]. The effectiveness of this manoeuvre is >80% (26/32 cases reported in English language publications). Other treatments, e.g. epidural saline infusion, oral corticosteroid administration [4], fluid intake [2,4] or caffeine [2], are less effective (effective rate 57%, 63%, the latter two treatments gave transient relief, respectively). In 10 patients with a meningeal diverticulum or focal spinal cerebrospinal fluid leakage, surgical intervention was most effective, especially in patients in whom multiple attempts at non-surgical treatment had failed, achieving 100% relief from the headache [4]. However, we should always bear in mind that this is traumatic therapy.

In our patients, a cervical epidural blood patch with 10 mL autologous blood was effective. However, in the first patient, we had to add a lumbar epidural blood patch to patch the fistula generated by the lumbar puncture. Despite the lumbar puncture being indispensable in the diagnosis of a spontaneous intracranial hypotension patient, we should pay attention to the fact that this process per se can trigger headache.

A cervical epidural blood patch is superior to a lumbar epidural blood patch because the quantity of blood required is smaller. Furthermore, this may decrease the possibility of meningitis or sepsicaemia. Our patients complained of mild neck discomfort only after the cervical injection. As De Rosaryo [6] has suggested, it is important to follow-up these patients regularly with special reference to the possibility of neurological deficits developing after this procedure. This is chiefly because of the anatomy of the cervical epidural space: a very narrow space between the ligamentum flavum and the dura mater. From our clinical experience, it appears to be safe and effective to control and use <10 mL blood. We recommend the very careful application of a cervical epidural blood patch to patients with persistent headache due to spontaneous intracranial hypotension, although we should always be mindful of possible complications such as a neurological deficit [7], septicaemia [8] and postdural puncture headache.

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References
Pharyngeal necrosis with the laryngeal mask airway

EDITOR:
The laryngeal mask airway has been used for periods of up to 10–24 h with no apparent problems [1–3]. Uvular necrosis of unknown origin has been reported following tracheal intubation [4], but to our knowledge, there are no other reports of pharyngeal necrosis associated with airway management.

A 53-yr-old female (weight 110 kg, height 163 cm) was scheduled for elective clipping of her left anterior cerebral communicating artery following a grade III subarachnoid haemorrhage. The patient was not considered to be at risk of aspiration. The airway was maintained with fibreoptic-guided intubation through a size 5 laryngeal mask airway under general anaesthesia using a size 7.5 mm endotracheal tube to minimize the cardiovascular stress response to laryngoscopy. Insertion of the laryngeal mask airway was easy and the cuff was inflated with 20 mL air. Fibreoptic intubation was accomplished at the first attempt. There was no significant change in heart rate or blood pressure (measurements ± baseline) during laryngeal mask airway insertion or intubation. A 14-FG nasogastric tube was passed into the stomach blindly behind the mask to permit enteral nutrition. The operation lasted for 8 h and the intraoperative course was unremarkable. Postoperatively, the patient was transferred to the intensive care unit for elective ventilation of the lungs. The laryngeal mask airway was left in situ since it was felt that attempts to remove it around the tracheal tube might result in accidental extubation or stimulation of the cardiovascular system jeopardizing the newly clipped aneurysm. Similarly, the cuff was not deflated since it was felt that the deflated cuff would make the position of the mask unstable and be more likely to cause movement of the endotracheal tube and stimulate the cardiovascular system. It was considered that the patient would probably be extubated within 48 h, but neurological function was slow to return and extubation and removal of the laryngeal mask airway did not take place until the eighth postoperative day. Shortly after extubation the patient developed acute respiratory distress and required reintubation. An area of necrosis, 0.75 cm in diameter, was seen at laryngoscopy in the midline on the posterior pharyngeal wall. This was treated with corticosteroids and debridement via a fibreoptic scope for one week. The patient subsequently recovered from respiratory failure and the pharynx healed completely. There were no further sequelae.

Animal studies have shown that tracheal mucosal injury is primarily related to the pressure exerted against the mucosa and, to a lesser extent, the duration of application of that pressure. There are no published data about the relationship between pharyngeal mucosal injury and the level/duration of mucosal pressure, but there is evidence that pharyngolaryngeal morbidity with the laryngeal mask airway is more common at high volumes [5] and following prolonged insertion [6]. The mean mucosal pressure exerted by the laryngeal mask airway is lower than the capillary perfusion pressure, but can occasionally exceed it at high cuff volumes, particularly against the posterior pharynx. Mucosal pressures may have been higher in our patient for several reasons: (a) the cuff was not deflated; (b) a large mask was used; (c) the patient was obese (there is some evidence that obese patients have a smaller pharynx than non-obese patients); and (d) there was a nasogastric tube in situ that might have created an area of high pressure. There are a lack of data about the use of the laryngeal mask airway for prolonged periods although procedures lasting more than 2 h have been commonly performed, and it has been used in 15 patients for between 4 and 8 h without problems [7]. There are two reports of laryngeal mask airway use in the intensive care unit for 10–24 h without apparent problems [1–3].

In retrospect, it would have been better to have removed the laryngeal mask since it is unlikely that accidental extubation would have occurred if an extender had been used and there is evidence that removal is unlikely to trigger a clinically significant cardiovascular stress response. Furthermore, the position of the accessory muscles of the tongue would have been preserved.

References

The laryngeal mask cuff is probably stable over the inflation range making it unnecessary to keep the cuff inflated. Unfortunately, this information was not available when the case occurred.

We conclude that there may be limits to the duration the laryngeal mask airway can be safely left in situ in terms of pharyngeal injury, particularly in the presence of a nasogastric tube. Animal studies are required to determine the extent of these limitations.

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References

More resistance than normal: equipment checks revisited

EDITOR:

We report a potentially dangerous failure of an epidural loss of resistance syringe that underlines the importance of the thorough checking of all anaesthetic equipment before use. In preparation for epidural catheter insertion into an adult patient using a Portex® Epidural Minipack, 16-G, System 1 (Portex Ltd, Hythe, UK), a careful dynamic check of the loss of resistance syringe revealed excessive resistance. On closer inspection, an accessory web of material was observed at the skirt end of the plunger (Fig. 1). The accessory web became folded back upon itself as the plunger was depressed, thus increasing the friction between the plunger and the barrel. Therefore, excessive force was required to depress the plunger of the loss of resistance syringe, which is normally a low-friction system. The syringe was returned to the manufacturer. The defect was due to a moulding fault compounded by an inspection error. It was the only reported failure in a production lot of nearly 11 000 units. A new moulding tool has since been commissioned (personal communication with Portex). This

![Faulty syringe](image1)

(a)

![Normal syringe](image2)

(b)

Figure 1.
(a) Faulty loss of a resistance syringe. The accessory web of material is seen as the irregularity on the skirt (distal) end of the plunger denoted by the arrow. (b) Normal syringe for comparison.
case demonstrates the importance of mandatory visual and dynamic checking of all anaesthetic equipment. Manufacturers and clinicians should co-formulate simple, standard checking routines, which are well publicized; indeed, many do this already.

The guidelines of the Association of Anaesthetists of Great Britain and Ireland for checking anaesthetic apparatus [1] are predominantly ‘airway-equipment’ oriented. We would strongly recommend that a greater emphasis is put upon the checking of non-airway-related anaesthetic apparatus in future editions of this document and in any other such guidelines on pre-use equipment checks. This would help reinforce any individual manufacturer’s advisory pre-use checks. Inevitably, despite high manufacturing standards, ‘non-airway’ anaesthetic equipment will occasionally prove to be faulty — with potentially dangerous consequences. We need to apply the same rigour to its checking as we do to airway-related equipment.

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