Editor—Many thanks to Dr Bourgain and colleagues for their interest in our article. First, we would like to apologize for mis-attributing the statement to the authors. The correct reference in our article\(^1\) was to 11 not 1 and it is not a direct quotation.\(^4\)

We believe our survey provides sufficient evidence to raise concerns over the use of transtracheal ventilation using a high pressure source without control of airway pressure, but a survey lacking denominators cannot confirm or refute its lack of safety. In the absence of large randomized controlled trials comparing techniques, properly designed prospective cohort data collection may be sufficient. It is possible that the 4th National Audit Project of the Royal College of Anaesthetists\(^5\) which will, from September 2008, prospectively identify major airway complications throughout the UK, will provide such information.

What our survey does illustrate is that the reality in the UK is that in the majority of hospitals, transtracheal procedures can only be performed using manual techniques. Assuming these techniques are only used for laryngeal surgery, we conclude that only 15% of respondents performed transtracheal techniques electively and only 7% have access to high-frequency jet ventilation. We agree the manual technique is ‘suboptimal’. However, if our experience is typical, it is likely that financial pressures prevent purchase of the more expensive equipment and suboptimal techniques are likely to be the norm in the UK for some time to come.

As inferred in our paper, we agree that all anaesthetists should be familiar with transtracheal ventilation techniques. Our survey suggests, at least in the UK, that experience is unlikely to be achieved during elective ENT lists. Other solutions are necessary.

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Effects of glyceryl trinitrate on cerebrovascular autoregulation

Editor—We read the article by Moppett and colleagues\(^1\) with great interest. There is a paucity of data on cerebrovascular effects of vasoactive drugs in awake patients without intracranial pathology and we are grateful to the authors for providing such data. However, we would like to highlight two aspects regarding the glyceryl trinitrate (GTN) part of their study.

First, the CO\(_2\) reactivities measured in this study are very high. We would expect reactivities in the range of 15–30% per kPa.\(^2\) Such high CO\(_2\) reactivity makes the study of changes in blood flow velocity in response to changes in arterial pressure very difficult, as small changes in CO\(_2\) will have marked effects on the flow velocities. Table 1 suggests that a shift to a lower end-tidal CO\(_2\) occurred between the measurements performed before and during infusion of GTN. This would have led to improved autoregulation. Secondly, assuming intact autoregulation, the decrease in flow velocity in response to the arterial pressure decrease induced by GTN is surprisingly large. If the static rate of autoregulation\(^3\) is calculated based on the median given in Table 1, the result is 37%, that is, in contrast to the measurements of dynamic autoregulation performed by the authors, static autoregulation is clearly disturbed. This discrepancy is unexpected in the investigated group of patients.

In our opinion, based on the presented data, it cannot be concluded that GTN did not affect cerebrovascular autoregulation in this study.

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Editor—Many thanks to Dr Steiner and colleagues for their interest and thoughtful comments on our study.\(^1\)

In response to the end-tidal CO\(_2\) values, the individual changes in ‘resting’ end-tidal values with and without GTN were mostly <0.3 kPa, and occurred in both directions. We cannot exclude undetected changes in Pa\(_{\text{CO}_2}\) due to GTN effects on pulmonary blood flow. We would therefore suggest that, within the limits of the study, any effects of changes in end-tidal CO\(_2\) were small and unlikely to affect autoregulation significantly.

We agree with Dr Steiner that the decrease in flow velocity in parallel with a GTN-induced decrease in arterial pressure can be interpreted as a reduction in static
autoregulation. However, two factors suggest that this may not be the case. First, neither we nor other authors found changes in tests of dynamic autoregulation, and it would be an interesting finding to demonstrate significant uncoupling of dynamic and static tests of autoregulation. Secondly, GTN produces vasodilation of the basal cerebral arteries, which will result in lower flow velocities for the same cerebral blood flow. The theoretical advantage of dynamic techniques is that the confounding effects of GTN are present throughout the test thereby avoiding this problem.

We would therefore caution against claiming changes in static autoregulation without confirmatory evidence that cerebral blood flow has in fact decreased.

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Recombinant activated factor VII for acute subdural haematoma in an elderly patient taking fondaparinux

Editor—Acute subdural haematoma is one of the most common neurosurgical emergencies. Anticoagulants may be a contributing factor. Emergency treatment aims to normalize haemostasis to limit haematoma expansion, as rapidly as possible. We report a case of subdural haematoma in a patient on treatment with fondaparinux which was antagonized by recombinant activated factor VII (rFVIIa).

A 76-yr-old man receiving fondaparinux 7.5 mg once a day for prevention of stroke due to atrial fibrillation was admitted with cranial trauma after a fall. His Glasgow coma scale (GCS) was 15/15, and there were no focal abnormalities on initial clinical examination. One hour later, his level of consciousness had decreased (GCS 11/15). A brain CT scan revealed an acute subdural haematoma. Two hours later, his neurological status seriously worsened (GCS 7/15) with unilateral mydriasis. A repeat brain CT scan revealed further bleeding with massive oedema and transtentorial herniation. It was decided to evacuate the haematoma as an emergency after administration of rFVIIa (90 µg kg⁻¹) to antagonize the fondaparinux. A craniotomy and clot evacuation was performed and haemostasis was successfully and easily achieved. By 12 h, intracranial pressure had increased to >40 mm Hg and a CT scan showed diffuse, massive oedema associated with hemispheric ischaemia. The patient died at 24 h from refractory intracranial hypertension.

Fondaparinux (Arixtra®) is the first synthetic selective factor Xa inhibitor. As with any anticoagulant therapy, there is a risk of bleeding complications. However, there is, as yet, no recommended antidote. rFVIIa has been reported to be effective in reversing anticoagulant effect of fondaparinux. In healthy volunteers, rFVIIa normalized haemostasis for at least 6 h after a single dose of 90 µg kg⁻¹. These results have been confirmed in vitro. We found only one case report of the use of rFVIIa, in a patient with haemorrhagic shock after fondaparinux 2.5 mg who was successfully treated with rFVIIa (90 µg kg⁻¹), combined with anti-fibrinolytic (tranexamic acid 15 mg kg⁻¹), which reduced bleeding within 1 h. In our case, advanced age and reduce renal clearance may have been risk factors for bleeding. We think that rFVIIa administration contributed to limit the bleeding during neurosurgery by antagonizing fondaparinux, and facilitating surgical haemostasis. It is possible that earlier administration may have avoided the subdural haematoma expansion we observed. Safety of rFVIIa is still controversial. One of the potential risks is arterial or venous thrombosis. In volunteers, a single dose of 90 µg kg⁻¹ did not result in an overshoot of coagulation. A clinical study comparing rFVIIa with placebo in intracerebral haemorrhage did not report any increased frequency of adverse events in the rFVIIa group, but another showed an increased frequency of serious thrombotic events. In our case, it is possible that the hemispheric ischaemia demonstrated by the CT scan at 12 h was the result of a prolonged cerebral herniation, or the result of a vessel thrombosis caused by rFVIIa administration.

Anticoagulant therapy may expose patients to serious side-effects. The risk–benefit assessment is particularly important for new anticoagulants such as fondaparinux, for which the management of bleeding complications is poorly documented. The use of rFVIIa as an antidote may be effective in life-threatening bleeding. As with other anticoagulant therapy and related intracranial haemorrhage, the timing of treatment has a major effect on the capability of rFVIIa to limit development of a haematoma.