could be adequate to diagnose ischaemia and provide the answer to the 'shunt or not shunt' clinical question, this device would be a valuable tool, indeed.

We recently were involved in the care of a patient who developed signs of intraoperative cerebral ischaemia during awake carotid endarterectomy performed under cervical plexus block. Some of our 'awake' carotid endarterectomy procedures are converted to a general anesthetic if the patient's mental status deteriorates or if the regional anesthetic is insufficient. Therefore, we use the PSA 4000 with the PSArray[2] (PSA) (Physiometrix Inc., North Billerica, MA, USA) as an adjunctive monitor to better titrate the depth of anesthesia. In these cases the PSA is used during the entire procedure. The PSA, like the BIS, uses the EEG and a complex algorithm to derive the Patient State Index (PSI), a number from 0–100 that is intended to be a guide to anesthetic depth. Unlike the BIS, which uses a single channel monopolar EEG to derive the BIS value, the PSA utilizes four channel (F_p1, F_pz1, Cz, Pz), multi-regional, multi-frequency, power and coherence relationships and its own unique algorithm to arrive at the PSI value. There have been case reports that the PSA, like the BIS, can detect global cerebral ischaemic events.[2] Often, even focal cerebral hypoperfusion manifests with large regional or even global EEG changes.[3] Additionally, there are some data that suggest that as few as two channels are necessary to detect cerebral ischaemia during carotid endarerectomy.[4]

Our patient was a 56-yr-old male with severe left internal carotid artery stenosis (80–99% stenosis) and moderate right internal carotid artery stenosis (50–79% stenosis). After placing a cervical plexus block and until the time of carotid cross-clamp, the patient was awake and alert, with a PSI value of 95–98. The monitor was set to display the raw EEG waves during the case, which were predominantly alpha and beta waves on both the left and right. Shortly after left carotid cross-clamp, the patient's mental status acutely deteriorated and his right hand grip strength was markedly diminished. Interestingly, the PSI value dropped to 85 after 2 min and the raw EEG showed delta and theta waves on both the left and right—a dramatic change from just moments before. Haemodynamics were unchanged.

It would appear that the BIS, as Deogaonkar and colleagues state, lacks the sensitivity to adequately detect cerebral ischaemia and detect the restoration of cerebral electrical activity once perfusion is reestablished. This monitor was certainly not designed for such a purpose. However, PSA may offer advantages over the BIS in having multi-channel analysis and the ability to compare raw EEG from the left and right hemispheres, thus the potential for the PSA to detect cerebral ischaemia may be much higher. Further study with the PSA in this area may be worth pursuing.

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Editor—Thank you for the opportunity to respond to the letter regarding our article.[1] We are interested to see Culp and colleagues confirm our observation that the BIS does not detect unilateral (ipsilateral) ischemia during awake carotid endarterectomy. It is interesting that more detailed EEG analysis by the PSA device might be more sensitive than BIS. Whichever form of monitoring is utilized, there is nothing more sensitive than the patient's neurological status during awake endarterectomy. It is for this reason that the awake procedure offers the highest sensitivity and specificity for ischaemia while no other monitoring procedure has achieved this sensitivity. We have previously reported the specificity and sensitivity of jugular venous oxygen saturation monitoring during awake carotid endarterectomy.[5] Of all the forms of intraoperative monitoring that have been compared with ischaemia in awake patients during carotid endarterectomy the SJV02 had the highest sensitivity and specificity. Transcranial Doppler has also been evaluated but it is not tolerated well in awake patients because of the need to maintain pressure on the temporal bone constantly throughout the procedure.

Careful studies using sophisticated electrophysiology as used in the case reported by Culp and colleagues need to be evaluated in a large series of patients before they can be universally accepted.

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Safety of transtracheal jet ventilation in upper airway obstruction

Editor—We read with interest the article by Chandradeva and colleagues,[1] who reported the use of percutaneous transtracheal jet ventilation (PTJV) in the emergency management of two patients with severe upper airway obstruction. We agree that this is an extremely useful technique for this situation but would like to suggest that the method they employed could be further enhanced by the use of an automated jet ventilator with end-expiratory pressure monitoring linked to a pause function. We have used this technique in two situations similar to those described in their article.

Case 1, a 64-yr-old man undergoing tracheostomy because of worsening episodes of stridor secondary to a vocal cord palsy after radiotherapy for malignant neck lymphadenopathy. He was given a small dose of midazolam and then an inhalational induction was attempted using sevoflurane in oxygen 100%. During induction the patient developed worsening stridor, and became restless. His arterial oxygen saturation began to fall, at which point the anaesthetist abandoned the gas induction and called for help. Stridor and hypoxia persisted despite discontinuing the volatile agent. At this point a second anaesthetist inserted a 14G Ravussin jet ventilation catheter (VBM, Germany) through the cricothyroid membrane under local anaesthesia, and the patient's lungs were oxygenated with PTJV. We used an automatic jet ventilator (Mistral model, Acutronic, Switzerland), and delivered oxygen ($F_{I_{O_2}}$=1) with initial settings of: driving pressure=1 bar (15 psi), frequency=30 min^{-1}, and pause pressure=20 cm H_2O. This led

to a rapid improvement of his oxygen saturation and a decision was made to continue with inhalational induction. This proceeded with difficulty, but on the occasions when upper airway patency was reduced, the ventilator detected a rise in the end-expiratory pressure and paused to limit lung over-inflation. I.V. anaesthesia was administered to complete the induction, and the patient was intubated orally once asleep and after neuromuscular blocking agents had been given. Like Chandradeva and colleagues,[1] we found that PTJV maintained good oxygenation, and that the gas emerging through the glottis also assisted with successful tracheal intubation.

Case 2, an 88-yr-old man presented with stridor secondary to bilateral vocal cord palsy for tracheostomy and examination under anaesthesia. Because of diminished cervical spine mobility, a good view at direct laryngoscopy could not be predicted confidently. A 14G jet ventilation catheter (as above) was inserted through the cricothyroid membrane under local anaesthesia. Tracheal placement was confirmed from the presence of a regular capnograph waveform, and jet ventilation started with the patient awake. The ventilator was initially set at: driving pressure=1.5 bar (22.5 psi), frequency=30 min^{-1}, and pause pressure=25 cm H_2O. Anaesthesia was induced i.v., and bag mask ventilation confirmed after which the patient was intubated conventionally with an 8.0 mm cuffed oral endotracheal tube. The jet ventilation catheter was removed, and the tracheostomy completed uneventfully. In common with the first case, effective oxygenation was achieved with jet inflation pressures limited to 1.5 bar, and end-expiratory pressures limited to levels conventionally believed to be safe.

As Chandradeva and colleagues acknowledge, the incidence of barotrauma during PTJV remains a concern, but the actual risk of this complication has not been estimated precisely. PTJV using a jet ventilator that incorporates end-expiratory pressure monitoring is now well described for elective surgery to the larynx.[2] Although peak inflation pressures remain unknown, the measured end-expiratory pressure has been shown in a separate study to correlate well with pulmonary distension.[3] In their clinical study employing pressure limited PTJV in elective patients, Bourgain and colleagues reported an incidence of 1% for pneumothorax.[2] In emergency scenarios, where there is severe compromise to the upper airway, intrathoracic pressures and volumes during unrestrained PTJV would be unknown, and the risk of barotrauma is likely to be higher. Although these are, to an extent, speculations we believe that on *a priori* grounds, using a dedicated jet ventilator with a pause pressure alarm facility can limit these risks as far as possible, and in the future may come to represent best practice.

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Editor—Thank you for the opportunity to respond to the letter from McLeod and colleagues concerning our paper.[1] They suggest that the delivery of jet ventilation with an automated jet ventilator with end-expiratory pressure monitoring linked to a pause function could enhance the safety of PTJV by minimizing the risk of barotrauma in severe upper airway obstruction. We agree with their statement and would like to thank them for making a contribution to the safety of PTJV in airway obstruction.

There is another potential benefit of employing automated jet ventilation. Studies in experimental models that simulate normal airway diameter and lung compliance using a driving pressure of 3.5 bar (50 psi) have demonstrated that the gas flow through 20, 16, and 14G cannula is ~400,[4] 500,[5] and 1600 ml s^{-1},[4] respectively. As the 1-s tidal volume with a 14G cannula is equal to 1600 ml, the inspiratory time should be limited to less than 1 s (e.g. 0.5 s) and the inspiratory:expiratory (I:E) ratio should be ~1:3 to allow adequate time for deflation or exhalation and thereby avoid air trapping and barotrauma. In order to meet these ventilatory requirements, it could be argued that automated ventilation is safer than the manual delivery, especially in anxious resuscitation situations.

In the cases reported by McLeod and colleagues, the airway obstruction appeared to be caused by vocal cord palsy but in our cases the acute airway obstruction was attributable to severe supraglottic oedema. We feel that it is important to take this difference into account as the driving pressure that is required to open up airway obstruction attributable to supraglottic oedema could be higher. However, the optimum driving pressure or more importantly the optimum intratracheal pressure that is required is not known. McLeod used a driving pressure of up to 1.5 bar (22.5 psi) via 14G transtracheal cannula whereas we employed 3 bar (44 psi) via a 14G cannula in severe supraglottic oedema. Patel[6] reported a driving pressure of 3.5 bar (50 psi), which was delivered manually via varying sizes of cannulae (12G, 16G, and 6 F) in 23 patients when there was a cannot intubate and difficult to ventilate' situation and did not encounter barotraumas in the case series. Although barotrauma is a potential complication and a serious concern among the users of PTJV, the reported incidence of this complication, if driving pressure is less than 4 bar,[4] remains low.[2 6] When in doubt, it seems prudent to start with the driving pressure at a low level, increasing it as dictated by the clinical response.

We wish to emphasize that the application of PTJV should be considered as a rescue and temporary manoeuvre to oxygenate the patient while a more secure permanent airway is being established at the earliest opportunity. It is our experience that tracheal intubation is aided by virtue of inducing high intratracheal pressure and its effect on the glottic area. If direct laryngoscopic tracheal intubation during the PTJV fails, a definitive airway should be established by means of surgical or percutaneous dilatational tracheostomy or fibreoptic-aided intubation without delay. Whether automated or manually delivered PTJV is used, great care should be exercised at all times to minimize the risk of barotrauma and the duration of the PTJV should be kept to a minimum.

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