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Correspondence

Oxygen administration can reverse neurological deficit following carotid cross-clamping

Editor—We read with interest Stoneham and Martin's recent case report[1] describing two patients who became neurologically obtunded during awake carotid surgery. They found that administration of oxygen 100% through a tight-fitting anaesthetic face mask and circle-breathing system reversed the neurological deficits, so that surgery could be completed without the need for shunting.

Two common mechanisms cause neurological deficits during carotid surgery: cerebral hypoperfusion, which is usually reversible; and macro-embolization, which is often irreversible. Most patients, undergoing loco-regional carotid surgery, will tolerate the cross-clamp phase without difficulty, however, in those patients who do become obtunded, a clear strategy needs to be agreed in advance by both anaesthetist and surgeon. We handle deficits that occur within the first 90 s by declamping the artery and allowing the deficit to recover. The operation is then continued under general anaesthesia and the carotid shunt can be inserted in a controlled fashion. A deficit that occurs more than 90 s after cross-clamp, but before the carotid arteriotomy (trial clamp for 5 min), is relatively straightforward to manage. Clamps are temporarily released, normal neurology restored, clamps are then reapplied allowing a shunt to be inserted before the patient becomes obtunded a second time. The most difficult situation, and that described in Stoneham and Martin's report[1] is when the patient deteriorates after the arteriotomy has been made, as clamp release is no longer an option. In this situation, the patient often begins to deteriorate in a fairly subtle fashion, then develops mild focal neurological deficits before finally becoming profoundly obtunded. These subtle neurological changes should be picked up at an early stage and the options of pharmacologically augmenting the blood pressure or increasing the $F_{I_{O_2}}$ are likely to be beneficial. If the deterioration in cerebral function is slow, this implies only a modest imbalance between the regional cerebral oxygen delivery (rCOD) and regional cerebral oxygen consumption (rCOC). Any strategy that improves rCOD or decreases rCOC may reverse the deficit. $P_{a_{O_2}}$[1] and blood pressure[2] are important determinants of rCOD. Cerebral function is altered by hypoxia. When $F_{I_{O_2}}$ is decreased to 75% of normal, complex task performance is altered; at 65% short term memory is impaired; at 50% judgement is altered; unconsciousness occurs with $F_{I_{O_2}}$ between 30–40% of normal.[3]

Stoneham and Martin[1] did not measure $P_{a_{CO_2}}$ at the time the patient was obtunded, but they speculate that that there might be 10% decrease in ventilation leading to an increase in $P_{a_{CO_2}}$ of up to 0.5 kPa, causing a right shift in the oxygen dissociation curve, improving rCOD. The effect of $P_{a_{CO_2}}$ on rCOD to the hypoxic 'normal' brain at altitude has been studied and suggests other mechanisms also come into play.[4 5] A rise in $P_{a_{CO_2}}$ causes an increased rate and depth of respiration, increased cerebral blood flow, and right shift of the oxygen dissociation curve. These mechanisms improved rCOD, and this was confirmed with near infrared cerebral spectroscopy. Interestingly there appears to be a synergistic effect with a combination of supplemental oxygen and carbon dioxide giving the largest improvement in both $P_{a_{O_2}}$ and cerebral oxygenation. There are animal data suggesting the hypoxic hypercarbic brain is less susceptible to hypoxic neuronal damage than the hypoxic hypocarbic brain.[6]

These case reports give important insights into the options as to how to manage patients with hypoperfusion deficits during loco-regional carotid surgery. Further studies into the manipulation of cerebral oxygen delivery during carotid endarterectomy are indicated. Clinical measurement of the balance between rCOD and rCOC is difficult, as patients undergoing awake carotid surgery rarely have jugular venous lines inserted. Cerebral near infrared spectroscopy, whilst having limitations in terms of absolute measurements,[7] may give continuous non-invasive assessments of the rCOD–rCOC balance.

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Editor—As the increase in oxygen carrying capacity achieved by the administration of oxygen 100% with a close-fitting anaesthetic face mask in a patient with a normal haemoglobin and who is not desaturated ($\Delta P_{a_{O_2}} \times 0.00310$) is miniscule, how might these neurological deficits have been reversed?[1] By inducing an acute lipid shift? If so, what was the trigger? Free radicals? If so, might adverse effects have been detected had a prospective study been performed in which the neurocognitive tests used in an earlier study in your institution[8] were conducted preoperatively and 3 months later?

'Detailed cognitive assessment, using a battery of tests, shows some impairment in as many as 80% of patients [having open heart surgery] at the time they are discharged from hospital, which persists in around a quarter of them at six months'.[8] As neurocognitive impairment in patients undergoing conventional and off-pump coronary artery bypass grafting in your institution were similar it was 'speculated that the effects of surgical injury and anaesthesia might be as important as the use of cardiopulmonary bypass in causing impairment'. Hyperoxia might be a cause despite these two case reports.[9]

In a patient who develops an extradural haematoma after a head injury, mild concussion may be followed by a lucid interval after which neurological symptoms and even death from coma may develop many hours later. I would be very wary of accepting these two case reports as evidence in support of the view that increasing $F_{I_{O_2}}$ to 1.0 during carotid artery surgery is universally beneficial. There are many possible causes of secondary brain injury in these circumstances.

'The introduction of routine intraoperative shunting and patch closure [in your institution], as well as allowing surgical trainees to perform supervised CEAs, has not affected perioperative morbidity and mortality rates or long-term outcome'.[9] Your results[10] appear, however, to fall far short of those routinely achieved in the US.[11] This difference, if real, could be technical. If so, using awake carotid surgery to reduce the need for shunts is unlikely to be a satisfactory solution. The difference might alternatively or additionally be a reflection of differences in the standards of anaesthetic practices in the UK and US.

Is mental functioning during awake neurosurgery, including intracranial revascularization, a reliable proxy for metabolic monitoring?[12] I suspect not because it has required detailed cognitive assessment to reveal the extent of the impairment after cardiac surgery and many neurocognitive disturbances appearing

after head injuries and brain surgery might have been avoidable events.[13]

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Editor—Thank you for the opportunity to reply to correspondence about our recent case report concerning cerebral oxygenation during awake carotid surgery.[1] I am not aware of specific evidence supporting Imray and colleagues' differential approach to the management of cerebral hypoxia after cross-clamping depending on the speed of onset of the symptoms. For patients developing neurological deficits within 90 s of cross-clamping, other vascular teams would choose to keep the patient awake and to insert a shunt electively, keeping general anaesthesia as a 'last option'. We use a trial cross-clamp period of just 2 min, as most neurological deficits present within that time.[14] Others do not use a trial cross-clamp period at all, proceeding directly to arteriotomy after cross-clamping (Dr R. Telford, personal communication).

The Pa_{CO_2} data that Imray and colleagues refer to are very interesting. One can only speculate about how important this was as a contribution to the improvement in cerebral oxygenation in our case report. Whatever the cause, I agree that non-invasive cerebral oximetry may provide the easiest technique of further investigation. Our pilot studies using cerebral oximetry so far do suggest that cerebral oxygenation is improved by supplemental oxygenation.

The thread of Dr Fiddian-Green's letter is more difficult to follow. He believes that standards in anaesthesia and surgery in the UK fall below those of the USA. He reaches this conclusion by comparing surgical data from Oxford going back to the mid-1970s with current data from the USA. In fact, over 75% of carotid surgery in Oxford is now performed awake. Routine shunting has been abandoned, using the awake patient's neurological state as the prime indicator of the need for shunting. Eversion endarterectomy, without patch closure is now performed by the majority of our surgeons.

Dr Fiddian-Green considers the increase in oxygen-carrying capacity of the blood when changing the $F_{I_{O_2}}$ from 0.3 to 1.0 to be 'miniscule'. Simple calculations (as detailed in the discussion section of our report) show that the increase was in fact an unexpected 8% of the total oxygen content—which could well have made a significant impact in a group of neurons close to their ischaemic threshold.

One must be cautious comparing cerebral hypoxia after carotid cross-clamping with the cognitive impairment that occurs after cardiac surgery or the neurological effects of raised intracranial pressure after an extradural haematoma. The pathophysiology and timescale in each case is different. The cerebral symptoms during awake carotid endarterectomy develop very acutely—a cerebrovascular accident is evolving before one's very eyes. We are not advocating the administration of oxygen 100% to all patients—rather, this becomes an addition to the anaesthetist's armamentarium in the management of this fascinating but challenging group of patients.

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1 Stoneham MD, Martin T. Increased oxygen administration during awake carotid surgery can reverse neurological deficit following carotid cross-clamping. *Br J Anaesth* 2005; **94**: 582–5
2 Stoneham MD, Warner O. Blood pressure manipulation during awake carotid surgery to reverse neurological deficit after carotid cross-clamping. *Br J Anaesth* 2001; **87**: 641–4
3 Blass JP, Gibson GE. Consequences of mild grade hypoxia. *Adv Neurol* 1979; **2**: 229
4 Imray CH, Walsh S, Clarke T, *et al*. Birmingham Medical Research Expeditionary Society. Effects of breathing air containing 3% carbon dioxide, 35% oxygen or a mixture of 3% carbon dioxide/35% oxygen on cerebral and peripheral oxygenation at 150 m and 3459 m. *Clin Sci (Lond)* 2003; **104**: 203–10
5 Imray CH, Brearey S, Clarke T, *et al*. Cerebral oxygenation at high altitude and the response to carbon dioxide, hyperventilation and oxygen. The Birmingham Medical Research Expeditionary Society. *Clin Sci (Lond)* 2000; **98**: 159–64
6 Simon RP, Niiro M, Gwinn R. Brain acidosis induced by hypercarbic ventilation attenuating focal ischaemic injury. *J Pharm Exp Ther* 1993; **267**: 1428–31
7 Pattinson K, Clutton-Brock T, Imray C. Validity of near-infrared cerebral spectroscopy. *Anaesthesia* 2004; **59**: 507–8
8 Fiddian-Green RG. eLetter re: Taggart D. About impaired minds and closed hearts. *Br Med J* 2002; **325**: 1255–6
9 Fiddian-Green RG. eLetter re: Khaw KS, Wang CC, Ngan Kee WD, Pang CP, and Rogers MS. Effects of high inspired oxygen fraction during elective Caesarean section under spinal anaesthesia on maternal and fetal oxygenation and lipid peroxidation. *Br J Anaesth* 2002; **88**: 18–23
10 Hussain T, Senaratine JW, Green FR, Collin J, Hands L, Morris PJ. Vascular surgical society of great britain and ireland: twenty-three years of experience of carotid endarterectomy. *Br J Surg* 1999; **86**: 690
11 Cowan JA Jr, Dimick JB, Thompson BG, Stanley JC, Upchurch GR Jr. Surgeon volume as an indicator of outcomes after carotid endarterectomy: an effect independent of specialty practice and hospital volume. *J Am Coll Surg* 2002; **195**: 814–21
12 Fiddian-Green RG. eLetter re: Czosnyka M, Pickard JD. Monitoring and interpretation of intracranial pressure. *J Neurol Neurosurg Psychiatry* 2004; **75**: 813–21
13 Fiddian-Green RG. eLetter re: Smith M. Editorial I: Neurocritical care: has it come of age? *Br J Anaesth* 2004; **93**: 753–5
14 Lawrence PF, Alves JC, Jicha D, Bhirangi K, Dobrin PB. Incidence, timing, and causes of cerebral ischemia during carotid endarterectomy with regional anesthesia. *J Vasc Surg* 1998; **27**: 329–34; discussion 335–7

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Spinal endoscopy for chronic sciatica

Editor—We congratulate Dr Dashfield and colleagues[1] on a well conducted clinically relevant study. Caudal epidural placement of steroid and spinal endoscopic placement of steroid were found to be effective in patients with sciatica of 6–18 months. Superior, but not significantly superior results were found in the caudal epidural group. The conclusion must be that putting a patient through the longer, more uncomfortable, more costly and potentially more hazardous procedure of spinal endoscopy is difficult to justify on symptomatic grounds. We would tentatively accept this conclusion, but wish to emphasize some important caveats.

We refer particularly to a prospective observational study,[2] two prospective case series,[3 4] two retrospective evaluations[5 6] and a randomized double-blind controlled trial,[7] which have shown positive results with spinal endoscopy in patients with chronic low-back pain with radiculopathic leg pain who had previously obtained inadequate pain relief with traditionally placed caudal or lumbar epidural steroids. The reasons for this discrepancy are relevant to those who manage these patients on a day-to-day basis.

Firstly, all of these studies[2–7] involved different populations from those of the Dashfield and colleagues' study where no patients had undergone back surgery and mean symptom duration was