Case Report

Increased oxygen administration during awake carotid surgery can reverse neurological deficit following carotid cross-clamping

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We describe the management of two patients undergoing awake carotid surgery who developed signs of cerebral ischaemia following cross-clamping of the internal carotid artery. Administration of oxygen 100% with a close-fitting anaesthetic facemask reversed the neurological deficit, avoiding the need for insertion of an internal carotid artery shunt. Thus, the incidence of shunt insertion, which is reduced by the use of regional rather than general anaesthesia, could be reduced further by supplementary oxygenation. The possible mechanism and implications are discussed.

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It remains controversial whether regional or general anaesthesia for carotid endarterectomy (CEA) has the better outcome. There is some evidence from non-randomized studies that regional anaesthetic techniques are associated with less morbidity and mortality.1 Certainly, regional anaesthesia for CEA has become more popular in the UK in recent years.2 The reasons for this may include cultural factors and the personal choice of surgeons and anaesthetists, but there is little doubt that an awake patient provides real-time assessment of cerebration, contra-lateral motor power, and speech. This is clearly advantageous during the period of internal carotid artery cross-clamping, allowing timely intervention when required.3

Pharmacological augmentation of arterial pressure can reverse neurological deficits that develop following carotid cross-clamping during awake carotid surgery.4 The mechanism postulated to explain this effect is an increase in ‘driving pressure’ across the Circle of Willis from the vertebral and contra-lateral internal carotid arteries. In this case report, this intervention failed to reverse the neurological changes in two patients undergoing awake CEA. However, increasing the inspired oxygen concentration did reverse the neurological deficit, allowing surgery to conclude successfully.

Case reports

A female patient of 78 yr with visual disturbances and 70% stenosis of the left internal carotid artery presented for carotid endarterectomy. She was hypertensive, with mild angina on exercise, which had been treated successfully by angioplasty 2 yr previously. Six months before this reported procedure, she had undergone right carotid endarterectomy uneventfully. During the first procedure she tolerated carotid cross-clamping without developing symptoms or signs of cerebral ischaemia.

On this occasion, her unpremedicated arterial pressure, recorded on the ward and in the anaesthetic room, was approximately 160/85 (mean 115 mm Hg). Perioperative monitoring consisted of: 5 lead ECG; invasive arterial pressure; arterial blood gases; oxygen saturation; and end-tidal respiratory gases estimated from within a standard variable-performance medium concentration (MC) oxygen face-mask. No other cerebral monitoring technique was used. The patient was fully conscious, without sedation, throughout the procedure.

Vascular access, placement of deep and superficial cervical plexus blocks, surgical incision, and dissection proceeded uneventfully. Analysis of a sample of arterial blood, taken with the patient breathing oxygen 2 litre min⁻¹ via an MC mask revealed that the arterial partial pressure of oxygen (\(P_{aO_2}\)) was 16 kPa, and the arterial partial pressure of carbon dioxide (\(P_{aCO_2}\)) was 5.1 kPa. As the MC mask is known to deliver 0.25–0.3% \(F_{iO_2}\), this \(P_{aO_2}\) is consistent with the patient having no or negligible pulmonary shunt fraction.5

The patient’s neurological status was unchanged following a period of 2 min trial cross-clamping. The surgeons...
therefore proceeded to eversion carotid endarterectomy rather than the standard surgical technique. However, 10 min after carotid cross-clamping, the patient became slightly confused, following which there was a gradual loss of consciousness until, 3 min after carotid incision, the patient was aphasic and unconscious, responding to painful stimuli with grimacing only.

This is an unusual time for presentation of a neurological deficit. Commonly this happens either immediately following carotid cross-clamping or later, when it is often associated with relative hypotension. However, at this stage, the patient’s arterial invasive arterial pressure was 175/90 mm Hg, with sinus rhythm, rate 52 and oximetric oxygen saturation (S\textsubscript{p\textsubscript{o}}\textsubscript{2}) reading 100%. With a ventilatory frequency (measured from the end-tidal carbon dioxide monitoring) of 13 to 15, the end-tidal oxygen concentration varied from 28–40% (estimated from within the MC mask).

Whilst an unresponsive patient, available options were limited. Whilst the surgeons prepared to insert an internal carotid artery shunt (considered by some surgeons to be more difficult using the inversion endarterectomy technique\textsuperscript{8}), pharmacological augmentation of arterial pressure was considered and rejected as arterial pressure was already elevated. Instead, conversion to general anaesthesia was planned.

The MC mask was exchanged for an oro-nasal anaesthetic facemask administering oxygen 100%, 6 litre min\textsuperscript{-1} via a circle breathing system. The plan was pre-oxygenation with oxygen 100%, intubation with a laryngeal mask airway, followed by spontaneous ventilation until the end of the procedure using a propofol infusion to maintain anaesthesia. However, after only 1 min of pre-oxygenation, the patient became more responsive. Over the next 3 min, her conscious level improved such that she became orientated in space and time, with normal speech and contra-lateral motor power. Although the dynamic change in P\textsubscript{a\textsubscript{o}}\textsubscript{2} was not recorded, assuming no change in V/Q shunt and an F\textsubscript{1\textsubscript{o}}\textsubscript{2} of 1.0, the P\textsubscript{a\textsubscript{o}}\textsubscript{2} may be estimated to be above 80 kPa.\textsuperscript{9}

Eversion endarterectomy was completed successfully without further neurological problems with a total cross-clamp time of 28 min. Her postoperative course was uneventful. She was discharged home on the second postoperative day.

A male patient of 78 yr with a 70% stenosis of the left ICA presented for left CEA under regional anaesthesia following a dysphasic stroke 6 months earlier. Anaesthetic and surgical techniques were as described for the previous case. Oxygen 2 litre min\textsuperscript{-1} was administered via an MC mask. The patient became confused and dysphasic 5 or 6 min after carotid cross-clamping. Cautious incremental administration of metaraminol (total dose 2 mg) elevated his arterial pressure to 185/85 without change in the neurological status. However, administration of oxygen 100% from a circle breathing system via an anaesthetic facemask reversed the neurological deficit completely. The MC mask was replaced, whereupon the patient became restless, anxious, and frightened over 2 or 3 min. These feelings disappeared once oxygen 100% was administered again. The operation thereafter proceeded uneventfully.

Discussion

There are two interesting features of these case reports, which merit further discussion. The first is to explain how increasing the inspired oxygen concentration could have improved the patients’ neurological status. The second is the intriguing possibility that the administration of oxygen 100% becomes another therapeutic option to be tried when a patient undergoing awake carotid surgery develops neurological symptoms following carotid cross-clamping.

To explain the first of these, it is relevant to calculate the oxygen content of the blood at the time the patient first lost consciousness. Before carotid cross-clamping, the patient was breathing oxygen at 2 litre min\textsuperscript{-1} via an MC mask.\textsuperscript{10} Her P\textsubscript{a\textsubscript{o}}\textsubscript{2} measured from arterial blood gas analysis, was 16 kPa. With a measured preoperative haemoglobin concentration of 135 g litre\textsuperscript{-1}, the oxygen content of the blood (C\textsubscript{a\textsubscript{o}}\textsubscript{2}) is calculated simply as follows:\textsuperscript{5}

\[
C_{a\textsubscript{o}\textsubscript{2}} = \alpha P_{a\textsubscript{o}\textsubscript{2}} + (S_{a\textsubscript{o}} \times [Hb] \times 1.31)
\]

Where: \(\alpha\) is the solubility coefficient of oxygen in blood= 0.228 ml litre\textsuperscript{-1} kPa\textsuperscript{-1}; \(P_{a\textsubscript{o}\textsubscript{2}}\) = arterial partial pressure of oxygen (kPa); \(S_{a\textsubscript{o}}\) = arterial haemoglobin oxygen saturation (%); \([Hb]\) = blood haemoglobin concentration (g litre\textsuperscript{-1}); and 1.31=Huffner constant (oxygen-binding capacity of haemoglobin) (ml g\textsuperscript{-1}).\textsuperscript{11}

Substituting the figures for this case results in a calculated oxygen content of 186 ml litre\textsuperscript{-1}. It is reasonable to assume that, although it was not directly measured during the period of cerebral hypoxia, increasing the F\textsubscript{1\textsubscript{o}}\textsubscript{2} from 30 to 100% would increase the P\textsubscript{a\textsubscript{o}}\textsubscript{2} to approximately 80 kPa. Thus, using the same equation (1), the oxygen content of the blood would have increased to: (0.228×80)+\((1.35\times135)\times1\)= 200.5 ml litre\textsuperscript{-1}, an increase of 14.5 ml litre\textsuperscript{-1} (8%). The question is whether this increase in oxygen content could account for the dramatic improvement in neurological condition?

The steps by which \(P_{O2}\) decreases from air to the mitochondria are known as the oxygen cascade.\textsuperscript{5} Included are factors such as: dilution of inspired oxygen by water vapour and carbon dioxide in the alveoli; the alveolar/arterial \(P_{O2}\) difference; haemoglobin carriage of oxygen; the haemoglobin-oxygen dissociation curve; oxygen consumption; and oxygen diffusion through tissues. Thus, the \(P_{O2}\) within the mitochondria may be as low as 0.5 kPa, although there is considerable variation between different areas of the brain.\textsuperscript{12}

Eventually, under hypoxic conditions, oxygen tension reaches the ‘critical \(P_{O2}\)’, below which oxidative cellular phosphorylation fails. This is the start of the neurotoxic cascade,\textsuperscript{13} which ultimately, ends in cell death. In isolated
mitochondria, the critical $P_{O_2}$ is below 0.13 kPa, although as a result of the barrier to oxygen diffusion presented by the proteinaceous nature of cytoplasm, this is more likely to be 0.5–1.3 kPa in intact cells.

An increase of 8% in blood oxygen content would presumably raise mitochondrial $P_{O_2}$ by a similar amount. This could be enough to take it above the critical level so that oxidative phosphorylation in ischaemic cerebral neurones could restart. Of course, this hypothesis is, with current technology, impossible to prove. $P_{O_2}$ varies immensely in tissues and within cells. Factors include distance from capillary supply, diffusion through cellular and extracellular substrates and the oxygen partial pressure gradients. Little is known of precise neurocellular oxygen dynamics but inherently it would appear obvious that a significant increase in the gradient could rescue a cell from a critical oxygenation state to one which allows oxidative processes to occur normally. Indeed, one could even speculate that the time it took for the neurological deficit to develop (more than 5 min in both cases) indicates that the pre-event mitochondrial $P_{O_2}$ must have been very close to the critical level.

Shift of the haemoglobin–oxygen dissociation curve may also have been advantageous in this case. Two separate factors may contribute here. First local tissue ischaemia causes anaerobic metabolism, leading to a local increase in [H⁺]. This decreases pH and shifts the oxygen dissociation curve to the right, thereby helping to unload oxygen to the tissues. At low $P_{O_2}$ values, the oxygen dissociation curve is steep, and large amounts of oxygen are liberated per unit drop in $P_{O_2}$. Secondly, if we assume the $P_{a_O_2}$ increased from 16 to 80 kPa, alveolar ventilation may decrease, leading to a rise in $P_{a_O_2}$. Eventually, ventilation may decrease by 10%, leading to an increase of up to 0.5 kPa in $P_{a_O_2}$. This will also tend to right-shift the oxygen dissociation curve, helping to unload oxygen to the tissues.

Of course, the duration of ischaemia is also critical here. For cerebral tissues, irreversible hypoxia may result after just a few minutes of ischaemia. In this case, the duration of the neurological deficit was itself only 1 or 2 min.

This phenomenon could be investigated by looking at the influence of oxygen 100% on the $P_{a_O_2}$ of blood in the internal carotid artery above the carotid cross-clamp, and on jugular venous $P_{O_2}$. It would be interesting to know what the decrement in distal carotid $P_{a_O_2}$ associated with detectable neurological deficit is. Near infrared spectroscopy has previously been used to monitor cerebral oxygenation during carotid endarterectomy and angiography, but the technique is global and does not offer the sensitivity that is required to elucidate oxygen status changes in the most oxygen-sensitive brain areas.

The management of patients developing neurological deficit during awake carotid endarterectomy is controversial. The options available depend on the experience of the anaesthetist, the conscious level of the patient, and the degree of neurological deficit. There is one major decision to make—whether to attempt to reverse the neurological deficit or to convert to general anaesthesia, which is the fall-back option. This must obviously be considered a priority if the patient’s airway is compromised because of reduced conscious level or seizure activity. However, there have been previous descriptions of pharmacological intervention to reverse the neurological deficit, in particular elevation of arterial pressure. The mechanism hypothesized to explain this is via an increase in perfusion pressure across the Circle of Willis. Exponents of general anaesthesia for carotid surgery have also described deliberate hypertension to 25% above baseline as a preventative measure to maintain cerebral perfusion.

Given that the aim of any such intervention is to increase the $P_{a_O_2}$ of blood in the ipsilateral cerebral cortex, then increasing the oxygen content of the blood to as high a level as possible may be considered as an additional intervention to be tried. For patients undergoing carotid endarterectomy—which is a preventative, not curative, operation—to suffer a perioperative stroke is a disaster. The administration of supplementary oxygen is another therapeutic possibility, which may be added to the list of options when cerebral ischaemia occurs. At the very least it may ‘buy time’ in terms of the integrity of the cortical neurones.

References


