Lack of Correlation Between Transconjunctival O₂ and Cerebral Blood Flow during Carotid Artery Occlusion

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The primary goal of monitoring during carotid endarterectomy is the early and accurate recognition of impending cerebral ischemia, thereby allowing corrective measures to be taken to lessen the likelihood of postoperative neurologic deficits. A recent report by Shoemaker and Lawner has advocated the use of the transconjunctival oxygen (PCO₂) monitor for that purpose, and states that PCO₂ correlates well with cerebral ischemia during temporary occlusion of the carotid artery.¹ We compared the transconjunctival oxygen monitor (Orange Medical Instruments, Costa Mesa, California) to two standard methods of assessing intraoperative cerebral ischemia: intrarterial ¹³³Xe regional cerebral blood flow (rCBF) studies and continuous 16-channel electroencephalogram.²

METHODS

After obtaining institutional approval and informed consent, 18 patients scheduled for elective carotid endarterectomy were studied. Anesthesia was induced with thiopental, 3–5 mg/kg iv, and maintained with isoflurane (0.25–0.5% expired) and 50% N₂O in oxygen. Arterial blood pressure was maintained at the upper limits of each patient's normal preoperative range by altering the anesthetic level. Paco₂ was maintained between 38–42 mmHg, as confirmed by periodic arterial blood gas sampling before and during carotid artery occlusion. Continuous values were available for arterial blood pressure, ECG, 16-channel electroencephalogram (EEG), bilateral PCO₂, and end-tidal CO₂, O₂, and isoflurane by mass spectrometry. Measurements of ¹³³Xe rCBF were made prior to carotid occlusion, immediately after occlusion, and following restoration of flow.

For this study, the occlusion value for ¹³³Xe rCBF was compared to the maximal change in ipsilateral PCO₂ within 5 min of carotid occlusion, and EEG changes were noted.

To avoid making the assumption that our population followed a Gaussian distribution, a simple ranking system was used to describe the data and Kendall's Rank Correlation Coefficients were calculated to measure the relationships.

RESULTS

Within 3 min of carotid occlusion, ipsilateral PCO₂ decreased in all patients, from ~1 to ~28 mmHg. Occlusion rCBF measurements ranged from 2 to 27 ml·100 g⁻¹·min⁻¹.

Fourteen of the 18 patients (77%) had occlusion rCBF of ≤18 ml·100 g⁻¹·min⁻¹. Five of the 18 patients (27%) had EEG changes of ischemia within 2 min of carotid artery occlusion (fig. 1). All five of these patients had oc-
The palpebral conjunctiva presents a unique opportunity to noninvasively monitor a capillary bed separated only 2–5 cell layers from the sensing device, across which little barrier to the diffusion of oxygen exists. The transconjunctival (PC$_{O_2}$) monitor, like the transcutaneous monitor, measures tissue oxygen tension which reflects both the O$_2$ content of the arterial blood and the blood flow to the particular tissue in question. Assuming a steady metabolic state, if arterial content remains constant, then any change in tissue oxygen should be secondary to a change in flow to that tissue.

The hypothesis that the PC$_{O_2}$ monitor would be useful in monitoring for cerebral ischemia is based on the assumption that, since the internal carotid artery is the primary source of oxygenated blood to both the conjunctiva and the cerebrum, changes in O$_2$ tension in one (the conjunctiva) should reflect changes in the other (the cerebrum).

Our failure to demonstrate any correlation between ΔPC$_{O_2}$ and occlusion rCBF or EEG changes during carotid occlusion can be explained by closer study of the vascular anatomy of the head and neck. The conjunctival capillary bed is supplied primarily by the ophthalmic branch of the internal carotid artery. However, it also receives variable supply from the external carotid artery via anastomoses with the superficial temporal, middle meningeal, and external maxillary arteries, thereby potentially participating in a rich network of extracranial collaterals.

Our findings agree with Shoemaker and Lawner in that, with occlusion of the carotid artery, ipsilateral PC$_{O_2}$ fell in all patients. However, when monitoring patients during carotid occlusion, the important question is not whether there has been a decrease in perfusion, but rather, is that change a significant one? Does it represent cerebral ischemia? Is a shunt needed: if so, with what degree of urgency?

In this study, the information provided by the PC$_{O_2}$ monitor did not consistently reflect cerebral ischemia as documented by EEG changes, nor as measured by $^{185}$Xe rCBF studies. Therefore, we conclude that the PC$_{O_2}$ monitor is an inadequate device for the detection of cerebral ischemia during carotid endarterectomy.
The concept of critical cerebral blood flow is important and can be defined as that flow below which cerebral metabolic demand exceeds cerebral blood flow and ischemia ensues. Based on work done during halothane anesthesia, the value for critical cerebral blood flow has been accepted to be \(<18 \text{ ml} \cdot \text{100 g} \cdot \text{min}^{-1}\). Utilizing essentially the same anesthetic techniques as in those earlier studies, we have shown what appears to be a much lower value for critical cerebral blood flow using isoflurane. In our study, five of six patients with occlusion rCBF \(< 8 \text{ ml} \cdot \text{100 g} \cdot \text{min}^{-1}\) exhibited EEG changes, while no EEG changes were seen when occlusion rCBF was above that value. We suggest that this apparent decrease in critical rCBF in humans under isoflurane anesthesia may be related to the possible cerebroprotective properties of isoflurane.

In summary, this study demonstrated no correlation between transconjunctival PCO2 and cerebral ischemia during carotid occlusion. A lower critical rCBF was noted during isoflurane anesthesia in man than has been reported during halothane administration in similar circumstances.

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The Influence of Hypothermic Cardiopulmonary Bypass on Neuromuscular Transmission in the Absence of Muscle Relaxants

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Cardiopulmonary bypass attenuates neuromuscular blockade from d-tubocurarine and alcuronium\(^{1,2}\) and augments neuromuscular blockade from atracurium\(^{3}\) and vecuronium,\(^{4}\) although all of these drugs are nondepolarizing muscle relaxants. One reason these conflicting findings cannot be explained satisfactorily is the lack of control data.\(^{4}\) Therefore, we examined the impact of hypothermic cardiopulmonary bypass on neuromuscular transmission in the absence of muscle relaxants.

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MATERIALS AND METHODS

This study was conducted according to the institutional standards of clinical research. Ten patients, ASA classes III and IV, undergoing open-heart surgery under nitrous oxide–fentanyl anesthesia were investigated. After induction of anesthesia with fentanyl, 1–2 mg, and fentanyl, 0.5–1.0 mg iv, the trachea was intubated following topical anesthesia without the aid of a muscle relaxant. The administration of nitrous oxide was discontinued on initiation of cardiopulmonary bypass.

Further details on anesthesia, cardiopulmonary bypass, and monitoring were described previously.\(^{4}\) Neuromuscular transmission was assessed by simultaneous recording of the compound electromyogram (EMG) of the right thenar\(^{4}\) and twitch tension of the right adductor pollicis muscle\(^{5}\) in response to supramaximal train-of-four stimulation of the ulnar nerve at the wrist (pulse width 0.2 ms). The changes in the size of the EMG action potentials (measured from peak to peak) and in twitch tension secondary to cooling and rewarming were related to naso-