Hypocapnia Added to Hypertension to Reverse EEG Changes during Carotid Endarterectomy

ALAN A. ARTRU, M.D.,* HUGH G. MERRIMAN, M.D.†

Studies of patients undergoing carotid endarterectomy have reported that internal carotid mean blood pressure distal to clamping of the common carotid artery (stump pressure) and/or cerebral perfusion pressure (CPP) increased significantly when mean arterial blood pressure (MAP) was increased by 25–30% or when hypocapnia (\(P_{\text{CO}}\) \(\sim\) 25–29 mmHg) was induced at the time of carotid clamping. Based on these results, the usual recommendation for clinical practice is that MAP should be maintained at normotensive or mildly hypertensive values during carotid clamping. In contrast, because of theoretical considerations, the routine use of hypocapnia is not widely accepted and, instead, normocapnia is recommended.

We report a case of carotid endarterectomy where it was not possible to use a carotid bypass shunt. Carotid clamping produced acute and profound changes in the electroencephalogram (EEG). Elevation of MAP did not reverse the EEG changes. However, the EEG changes did reverse when hypocapnia was induced. This report may represent a case where hypocapnia provoked a beneficial "inverse steal."

CASE REPORT

A 59-yr-old woman with hypertension, exertional angina, a previous myocardial infarction, and a history of tobacco and alcohol use experienced periodic right upper extremity weakness, vertigo, and slurred speech and sought medical care. Her medications were hydrochlorothiazide, one tablet, daily; dipryridamole, 25 mg, twice daily; aspirin, one tablet, daily; nitrobid, 2.5 mg, twice daily; and nitroglycerin, 0.4 mg, as needed. She had no neurological deficit and was felt to have not suffered stroke or reversible ischemic neurologic deficit. Carotid duplex scan and angiography revealed 90% stenosis of the left internal carotid artery, and minimal stenosis of the right internal carotid and both vertebral arteries. The patient consented to a left carotid endarterectomy under general anesthesia.

On arrival to the operating room, four electroencephalogram (EEG) electrodes (gold, cup) were placed, two over the right hemisphere and two over the left. The placement sites for each hemisphere were \(P_3\) and \(P_4\). The EEG was monitored using a Beckman Acutrace® polygraph (Beckman Instruments, Inc., Fullerton, CA) with a bandpass of 0.3–75 Hz. Electrodes were adjusted to maintain impedances between electrode pairs at < 3 kΩ. Computer (Digital Equipment Corp. Minc-28®, Digital Equipment Corp., Marlboro, MA) analysis of the EEG was performed using a Compressed Spectral Array (CSA) program, which averaged the EEG power spectra of four epochs (1 epoch = 4 s) every 30 s. EEG power spectra were evaluated according to the power in the standard frequency bins: delta (1–4 Hz), theta (4–8 Hz), alpha (8–14 Hz), beta 1 (14–20 Hz), and beta 2 (20–32 Hz). Before induction of anesthesia, the patient's EEG was characterized by high delta activity and moderate theta, alpha, and beta 1 and 2 activity from both hemispheres.

After induction of anesthesia with fentanyl 350 µg, intravenously, and thiopental 200 mg, intravenously, the left superficial cervical plexus was infiltrated with bupivacaine (0.5%). General anesthesia was maintained with inhalation of isoflurane (0.4%) and nitrous oxide (50%) in oxygen, and intravenous infusion of fentanyl. Blood pressure, measured via left radial artery catheter, was maintained near preanesthetic values (130/70) by intravenous infusion of phenylephrine and ventilation was controlled (\(P_{\text{CO}}\) of 37 mmHg). Expired \(CO_2\) was determined continuously by mass spectroscopy. The gradient between expired \(CO_2\) and \(P_{\text{CO}}\) was 2–3 mmHg.

Before carotid clamping, blood pressure was about 120/70, expired \(CO_2\) was 38–39 mmHg, and the patient's EEG consisted of high levels of delta activity, moderate theta and alpha activity, and minimal beta 1 and beta 2 activity from both hemispheres. During the first 2 min after carotid clamping, the rate of phenylephrine infusion was increased to maintain blood pressure at 120–150/70–85 mmHg, and normocapnia was continued. EEG activity from the right hemisphere was unchanged, but there was a dramatic decrease of theta and alpha activity from the left hemisphere (fig. 1). Because preoperative angiography indicated that stenosis of the left internal carotid artery extended distally to the near the entry of the artery into the carotid canal, it was felt that insertion of a carotid bypass shunt was not possible, so stump pressure was not measured. At 2 min after carotid clamping, the rate of phenylephrine infusion was further increased and blood pressure increased to 130–185/70–100 in attempt to reverse the left hemisphere EEG changes, which were felt to indicate that the patient was at increased risk for neurological injury. Over the next 4 min, there was no improvement in left hemisphere EEG. Therefore, at 8 min after carotid clamping, hypertventilation was used to induce hypocapnia. Two and one-half minutes after the onset of hyperventilation, the alpha activity of the EEG increased and, by 4 min after the onset of hyperventilation (expired \(CO_2\) of 28–29 mmHg), the patient's EEG consisted of high delta activity, moderate theta and alpha activity, and minimal beta 1 and 2 activity, similar to that observed prior to carotid clamping.

The carotid endarterectomy was completed, and the patient was awakened from anesthesia. Neurological evaluation revealed no neurological deficit at the time of awakening or at 1 or 3 days postoperatively.

DISCUSSION

Recommendations that hypocapnia should be used during carotid endarterectomy generally are based on pre-
vious reports that stump pressure,\textsuperscript{1,2,3,5,6,7} CPP,\textsuperscript{2} and/or regional cerebral blood flow (CBF)\textsuperscript{8} were greater with hypocapnia (Paco\textsubscript{2} of 25–34 mmHg) than with normocapnia\textsuperscript{1,2,8} or hypercapnia\textsuperscript{1,3,6,7} during carotid endarterectomy in anesthetized patients. It was suggested that hypocapnia might benefit patients undergoing carotid endarterectomy by provoking an “inverse steal,” or increase in regional CBF to ischemic tissue, “presumably due to increased local driving pressures resulting from arteriolar vasoconstriction in normally reacting, supposedly more healthy brain areas.”\textsuperscript{9} Such a mechanism may have been responsible for the abrupt reversal of EEG changes associated with hypocapnia in the present case.

However, the results of some of these same studies cast doubt on the idea that hypocapnia improves regional CBF in ischemic tissue during carotid endarterectomy. In two studies, “CBF equivalent” was less with hypocapnia than with normocapnia,\textsuperscript{2} and CBF was less with hypocapnia than with hypercapnia.\textsuperscript{3} In contrast, Petru et al. reported that CBF and vessel diameter were not different with hypopcapnia than with normocapnia during traumatic internal carotid artery spasm in monkeys anesthetized with pentobarbital and nitrous oxide.\textsuperscript{10} In two other studies, the incidence of neurological complications was lower with hypoxia than with hypercapnia, although the differences failed to achieve statistical significance due to the small numbers of patients with complications.\textsuperscript{5,7} Thus, the previously reported results on stump pressure, cerebral perfusion pressure, CBF, vessel diameter, and neurological complications do not clearly indicate that hypocapnia should be either beneficial or detrimental to patients during carotid endarterectomy.

Recommendations that hypocapnia should not be used during carotid endarterectomy are often based on a previous report that cerebral ATP was less and cerebral lactate greater with hypocapnia induced by mechanical ventilation (Paco\textsubscript{2} ~ 20 mmHg) than with spontaneous ventilation (Paco\textsubscript{2} ~ 30 mmHg) when hypocapnia was begun 30 min following middle cerebral artery (MCA) clamping in monkeys anesthetized with pentobarbital.\textsuperscript{11} While this work of Michenfelder and Sundt\textsuperscript{11} is often cited in talks and articles dealing with anesthesia for carotid endarterectomy, there are several reasons why citing this work for that purpose may not be appropriate.

First, Michenfelder and Sundt induced hypocapnia 30 min after MCA clamping based on the premise that, under clinical conditions, the opportunity to induce hypocapnia within 30 min of such an ischemic event would rarely arise.\textsuperscript{11} It may not be appropriate to apply results of hypocapnia begun 30 min after MCA clamping to the case of carotid endarterectomy, where hypocapnia, if used, would more likely be induced very close to the time of carotid clamping. As regards this issue, others reported that brain infarct size at 2 h post-clamping was significantly less with hypocapnia (Paco\textsubscript{2} ~ 25 mmHg) begun 30–60 min before clamping than with normocapnia in pentobarbital anesthetized dogs, whereas infarct size at 4 h post-clamping was not different with hypocapnia (Paco\textsubscript{2} ~ 25 mmHg) begun 1 h after clamping compared to normocapnia in pentobarbital anesthetized monkeys.\textsuperscript{12,13} Second, the MCA clamping used by Michenfelder and Sundt was intended as a model of acute cerebral ischemia.\textsuperscript{11} In contrast, during carotid endarterectomy, clamping produces vascular occlusion against a background of years of slowly developing vascular stenosis in the ischemic area, possible stenosis of other major extracranial vessels, probable chronic cerebral ischemia, and regional and systemic responses to that chronic pathology. Third, Michenfelder and Sundt reported worsening of cerebral metabolites with hypocapnia induced by mechanical ventilation only when hypocapnia was compared to spontaneous ventilation.\textsuperscript{11} During carotid endarterectomy in anesthetized patients, mechanical ventilation to normocapnia, rather than spontaneous ventilation, is generally recommended.\textsuperscript{4} It is noteworthy that the study of Michenfelder and Sundt also included a group of monkeys whose lungs were mechanically ventilated to normocapnia. They reported no difference in cerebral ATP or lactate with hypocapnia induced by mechanical ventilation when hypocapnia was compared to mechanical ventilation to normocapnia.\textsuperscript{11}

![Fig. 1. Compressed spectral analysis (CSA) of the EEG is shown for 6.5 min before and 14.5 min after carotid clamping. The first arrow indicates the time of carotid clamping. Clamping was followed by a substantial decrease in theta and alpha activity over the left hemisphere. The second arrow indicates the time of further increase in the rate of phentolamine infusion. No improvement in the left hemisphere CSA tracing occurred over the following 4 min. The third arrow indicates the time of onset of hypocapnia (6 min after carotid clamping). The left hemisphere CSA tracing returned to the preclamping pattern soon after hypocapnia was induced. CSA traces are displayed at 30-s intervals with frequency (0–16 Hz) on the horizontal axis and power on the vertical axis. Actual time is displayed on the column in between the left and right hemisphere CSA traces.](image-url)
Other reasoning given for avoiding hypopcapnia during carotid endarterectomy include a leftward shift of the oxyhemoglobin dissociation curve (making oxygen less available to tissues) and the possibility of increased cerebral vascular resistance in collateral vessels supplying ischemic areas. There are no studies evaluating the practical importance of these theoretical considerations.

It should be emphasized that, in the present case, hypopcapnia was begun only after the usual recommendations for anesthesia for carotid endarterectomy, i.e., maintenance of blood pressure at normotensive or mildly hypertensive values and mechanical ventilation to normocapnia, failed to reverse the EEG changes that followed carotid clamping. While hypopcapnia was associated with reversal of the EEG changes, there is no proof that hypopcapnia caused the reversal, nor that neurologic changes might have occurred had hypopcapnia not been instituted. Having observed restoration of a preclamping EEG pattern in the present case, we did not consider it prudent to return to normocapnia or to decrease the blood pressure to preclamping values in attempt to determine which treatment, hypertension, or hypopcapnia, might have caused resolution of the EEG changes produced by carotid clamping. It should not be concluded from this case report that we recommend the routine use of hypopcapnia during carotid endarterectomy. We recommend consideration of hypopcapnia in cases where reliable monitoring of brain well-being is used by persons experienced in the technique, and usual anesthetic management is documented not to preserve brain well-being.

REFERENCES