Our experience suggests that the lightwand technique, no matter which induction method is selected, provides an efficient and effective technique for tracheal intubation of the child with an abnormal upper airway. It may be employed successfully as a planned technique when a difficult intubation is anticipated, or as an alternative when other methods, such as blind nasal intubation or fiberoptic laryngoscopy, have unexpectedly failed.

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Cerebral and Hemodynamic Effects of Lidocaine Accidentally Injected into the Carotid Arteries of Patients Having Carotid Endarterectomy

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Sudden EEG slowing or seizure activity during surgery to relieve carotid artery stenosis may signify the onset of cerebral ischemia. If these EEG alterations occur during manipulation or instrumentation of the carotid artery, the diagnosis of (and therapy for) cerebral thromboembolus should be considered. In the following two case reports the sudden onset of EEG slowing (and in one case epileptiform EEG activity) accompanied by hypertension immediately followed an attempt to inject the carotid sinus with lidocaine. Because the EEG changes reversed spontaneously and there were no detectable neurologic sequelae in either case, the neurologic and hemodynamic alterations were presumed not to involve thromboembolus; instead the diagnosis of intraarterial lidocaine injection was made.

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CASE REPORTS

Case 1. A 64-yr-old, 107-kg man was scheduled for a right internal carotid endarterectomy to relieve a 99% stenosis of the proximal vessel. Carotid angiography also revealed diffuse atherosomatous disease of the left common and internal carotid arteries and an absence of right to left cross flow. Neurologic history was significant for blurring of vision and leftsidedness upon standing, with these symptoms increasing in frequency during the three months prior to surgery. The patient had never experienced seizures. Medical history included cigarette smoking for 25 pack-years and the use of hydrochlorothiazide for 15 yr for the treatment of hypertension. The patient had had one previous general anesthetic for gynecotomary surgery without complications. Physical examination was remarkable only for the presence of a left carotid bruit. The preoperative EEG, chest roentgenogram, blood chemistries (SMA-12), and cell counts were normal.

Following premedication with pentobarbital 100 mg and atropine 0.4 mg im, the patient was taken to the surgical suite where monitoring of the 16-lead EEG, ECG, arterial blood pressure measured directly, and O2 saturation by pulse oximetry were begun prior to anesthetic induction. The awake EEG showed 10 Hz alpha activity posteriorly, which was of slightly lower frequency periodically on the right. Anesthesia was induced with iv thalidomine 400 mg, vecuronium 20 mg, and lidocaine 100 mg. After tracheal intubation and controlled ventilation to maintain normocapnia (measured by mass spectrometry), anesthesia was maintained with isoflurane 0.5–0.8% inspired and nitrous oxide 50% in oxygen. Intraoperative blood pressure was maintained in the patient’s preoperative range of 140–170 mm Hg systolic. A Nat gamma scintillation detector was positioned over the right parietal cranium for measurement of regional cerebral blood flow (rCBF) of the middle
eventfully with no new neurologic deficits. The postoperative EEG was normal with resolution of the preoperative posterior asymmetry. Upon evaluation six weeks after hospital discharge the patient had no signs or symptoms of cerebral dysfunction.

Case 2. A 49-yr-old, 84-kg man was scheduled for a right common and internal carotid endarterectomy. Preoperative neurologic history was significant for two episodes of amaurosis fugax in the two months prior to surgery. Past medical history included hyperlipidemia, cigarette smoking, hypertension, and osteoarthritis of both knees. His medications included isosorbide dinitrate, clofibrate, hydrochlorothiazide, and trimetazidine. The patient had an uncomplicated myocardial infarction two years prior to surgery but had no subsequent cardiac symptoms. The patient had three uncomplicated prior general anesthetics for right knee surgery. Physical examination revealed bilateral carotid bruits and an abdominal aortic bruit. Visual field examination was grossly normal, but retinal artery pressures were decreased bilaterally, the left more than the right. Neurologic examination was otherwise normal. Carotid angiography demonstrated complete occlusion of the left internal carotid artery. The right common carotid artery had a long 50% stenosis that extended into the right internal carotid artery. The right carotid circulation contributed to the blood supply of both cerebral hemispheres, whereas the left external carotid artery did not contribute to the blood supply of either hemisphere. Chest roentgenograph and blood cell counts were normal. Aside from elevated cholesterol and triglyceride levels, the serum chemistries tested were normal. The EEG was consistent with an old inferior myocardial infarction; there was no evidence of ongoing ischemia or strain.

Following premedication with meperidine 50 mg and atropine 0.4 mg im the patient was taken to the surgical suite where preoperative monitoring of the EEG, EEG, and blood pressure (via a radial arterial cannula) was begun. The preanesthetic EEG showed normal symmetric 8 Hz alpha background rhythm. Anesthesia was induced with thiopental 500 mg and pancuronium 10 mg iv. The trachea was intubated, and ventilation was controlled. Anesthesia was maintained with halothane 0.5–1.0% inspired and N2O 50% in O2. The EEG consisted of well-developed 12–14 Hz rhythmic activity bilaterally with reduced amplitude in the left parasagittal region when compared to the right. Prior to carotid artery crossclamping a 25-gauge ½ inch needle was inserted by the neurosurgeon into the carotid sinus, and when no blood was aspirated, approximately 4 ml of lidocaine 1% was injected. Immediately thereafter the EEG exhibited marked slowing and reduction in activity bilaterally, which in both cases was more pronounced on the right (lower four channels in figs. 1A and 1B). After the onset of slowing there were several episodes of synchronous spikes as well as polyspikes maximal in the frontal regions (fig. 1B). The EEG slowing and intermittent activation resolved over the ensuing ten minutes, with the EEG returning to the anesthetized pattern seen prior to lidocaine injection.

With the onset of EEG disturbances the heart rate initially decreased from 80 to 50 beats/min but increased to 120 beats/min within 3 min. The sinus tachycardia persisted for ten minutes and then converted to a nodal rhythm at a rate of 80 beats/min. The systolic blood pressure acutely increased from 160 to 200 mmHg and returned to baseline values within 30 min. 133Xe CBF measurement during EEG slowing was 30 ml·100 g−1·min−1, a decrease from 49 ml·100 g−1·min−1 prior to the onset of EEG abnormalities. The CBF after carotid occlusion was 20 ml·100 g−1·min−1 and right-sided EEG slowing was noted six minutes later, becoming progressively more pronounced and bilateral. Shunt placement ten minutes after carotid occlusion resulted in resolution of the EEG abnormalities, first on the right and then on the left. CBF measurements after shunt placement and subsequent shunt removal were 75 ml·100 g−1·min−1 and 90 ml·100 g−1·min−1, respectively. Arterial blood gas tensions and pH were normal during the period of EEG alterations, and the remainder of the intraoperative course was remarkable only for occasional relapses into nodal cardiac
rhythm. The patient awakened uneventfully from anesthesia and had no apparent neurologic deficits. Follow-up examination four weeks after surgery detected no abnormalities.

**DISCUSSION**

Manipulation of the carotid artery during endarterectomy may produce stimulation of carotid sinus receptors residing in the carotid bifurcation, resulting in alterations of arterial blood pressure and heart rate. Because many patients with carotid occlusive disease may also have generalized peripheral vascular occlusive disease and occlusive coronary artery disease, intraoperative arterial blood pressure and heart rate fluctuations should be kept to a minimum. Injection of the carotid sinus with local anesthetic is one method to attenuate these fluctuations in cardiovascular function and is frequently performed by neurosurgeons at this institution. Potential complications of this technique include stimulation of the sinus during anesthetic injection, dislodging atheromatous plaques into the carotid lumen either during manipulation of the carotid artery or by the needle used to inject local anesthesia, or, as seen in the two reported cases, injection of local anesthetic into the cerebral vasculature.

During attempted local anesthetic blockade of the carotid sinus the anesthetic may enter the cerebral vasculature by several routes. First, the injecting needle tip may accidentally enter the carotid lumen. Although aspiration failed to withdraw blood in both of the reported cases, it is possible that the needle lumen was obstructed or against a vessel wall at the time of aspiration. Although the cerebral angiogram suggested 99% occlusion of the right carotid artery in case 1, it is possible that adequate flow was present to permit passage of the injected local anesthetic into the cerebral circulation. Another possibility is that the local anesthetic was injected into the carotid vasa vasorum, entering the internal carotid artery by retrograde flow. Finally, perhaps the distal internal carotid artery had contributions from collateral channels of the ascending pharyngeal artery. With very high grade stenosis of the proximal internal carotid system, this vessel may become an important source of collateral flow to the ipsilateral cerebral hemisphere. § Injection of this artery as it passes in close proximity to the carotid sinus could thus result in spread of local anesthetic to the cerebral circulation.

The intraoperative EEG changes in the two reported cases were temporally related to lidocaine injection of the carotid sinus. Both patients experienced a temporary decrease in cerebral electrical activity, and in the second case, brief bursts of potentially epileptogenic spikes were observed. The cerebral angiography results provide an explanation for the fact that EEG changes with lidocaine injection were unilateral in case 1 and bilateral in case 2. In the former case there was no cross flow between hemispheres, whereas in case 2 there was right to left cross flow. In case 2 a decrease in CBF coincided with the depression of the EEG.

The cerebral alterations following lidocaine CNS toxicity are dose-dependent. Small doses of lidocaine produce parallel decreases in CBF, metabolism, and electrical activity, whereas larger doses produce seizure activity. Unlike the seizures associated with organic epilepsy and most epileptogenic agents, lidocaine seizures result in only small increases in CBF and metabolism when compared with prelidocaine control values. Increasing the lidocaine dosage even further results in burst suppression or an isoelectric EEG accompanied by large decreases in CBF and metabolism. Due to a diminished volume of distribution, the dose of lidocaine required to produce CNS toxicity following arterial injection is small when compared to reported iv toxic doses. As little as 1 mg of lidocaine injected into the carotid circulation of humans can result in intraarterial levels as high as 30 μg/ml, levels that produce toxic manifestations in Rhesus monkeys. A diminished volume of distribution may also explain why seizures have occurred with probable intraarterial injection of low doses of local anesthetics for dental procedures.

In both of our cases EEG alterations were accompanied by hypertension, although one patient had EEG evidence of cerebral depression and the other had EEG evidence of seizure activity. In one case a cardiac rhythm disturbance was also observed. This hemodynamic pattern cannot be attributed to a direct effect of lidocaine on the cardiovascular system because the lidocaine doses were much smaller than the doses expected to cause hemodynamic alterations. Furthermore, when iv lidocaine is given to humans and laboratory animals in quantities sufficient to produce EEG depression and seizures, there is concomitant cardiovascular depression that persists throughout the seizure episode. The cardiovascular depression associated with cerebral–toxic doses of iv lidocaine is probably due to a direct effect of lidocaine on the heart. In contrast, in the two cases reported here the hemodynamic changes were likely due to the influence of lidocaine on cerebral autonomic function.

Intraoperatively, lidocaine-induced changes in cerebral activity should be differentiated from embolic stroke or other forms of cerebral dysfunction so that appropriate therapy can be initiated. The diagnosis of a lidocaine-mediated effect would include a temporal relationship between the lidocaine injection and the signs and symptoms of cerebral dysfunction. CBF monitoring may offer little assistance in distinguishing regional cerebral depression following lidocaine injection from that following

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ischemia. However, should seizure activity occur, the normal or only slightly increased regional CBF associated with lidocaine toxicity may differ from ischemia-induced seizures. The EEG is most useful in confirming the diagnosis of lidocaine toxicity. Intraarterial injection of small boluses of lidocaine results in short-lived, spontaneously reversible changes in electrical activity. This is in contrast to intraoperative cerebral ischemia-induced alterations of embolic origin, which usually result in regional changes in cerebral electrical activity of longer duration. The distribution of EEG changes following local anesthetic injection depends on the area of brain supplied by the injected artery. In case 1 the EEG alterations occurred over most of the ipsilateral hemisphere, a pattern that should appear in most patients. However, if the injected vessel supplies blood to both hemispheres, as seen in case 2, the less common pattern of sudden, bilateral EEG alterations will occur. The use of hemispheric EEG changes versus isolated, regional EEG changes as a criterion to distinguish between carotid injections of lidocaine versus thromboembolus, respectively, also will be hindered if a limited number of EEG electrodes are used and those electrodes are placed only over the area at greatest risk for embolic ischemia.

Until a diagnosis of lidocaine toxicity is made, cerebral EEG alterations should be treated as if they were embolic in nature. Therapy should include maintenance of adequate oxygenation and ventilation and maintenance of blood pressure in the normal to slightly elevated range. In both reported cases arterial blood pressure acutely increased for unexplained reasons following injection of lidocaine and no further therapy was needed. Because lidocaine-induced seizures are self-limited and associated with nearly normal CBF and metabolism, measures to alter these parameters are usually unnecessary. This is in marked contrast to the patient experiencing cerebral ischemia, in whom appropriate anesthetic management may include prolonged blood pressure elevation and barbiturate therapy to alter CBF and metabolism.

In summary, we report two cases in which transient EEG alterations consistent with severe cerebral dysfunction were observed during carotid endarterectomy. The proximity in time of the onset of EEG changes to the carotid sinus injection with lidocaine and the short-lived and transient nature of these changes suggest that they were secondary to lidocaine CNS toxicity and not ischemia. In addition, the appearance of bisynchronous spikes, an EEG change rarely observed with acute cerebral ischemia, would be more consistent with the known effects of lidocaine. In the absence of EEG monitoring and in the presence of general anesthesia and neuromuscular paralysis, similar events might go undetected or be manifested only by a change in heart rate or arterial blood pressure. These changes are easily confounded with those of mechanical stimulation of the carotid sinus. Although morbidity and mortality probably due to intraarterial injection of lidocaine into the cerebral circulation has been reported, it has occurred in patients without the advantages of preexisting airway support and monitoring. Under the latter circumstances adverse sequelae should not occur if proper attention is given to the patient's cardiovascular status and supportive measures are performed as needed. Neither of the patients reported here required additional therapy, and the cerebral dysfunction in both resolved spontaneously and without sequelae.

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