Lumbar disc disease can interfere with the spread of contrast material and presumably local anesthetic in the lumbar epidural space. Epidurography performed with metrizamide in our Pain Clinic has demonstrated failure of contrast material to diffuse upward past the level of a bulging lumbar disc, and to run out the foramina just below the level of disc abnormality. Likewise, several authors[5-7] have shown failure of contrast material to reach the area surrounding nerve roots affected by lumbar disc disease. In their series of 600 epidurograms, Luyendyk and van Voorthuisen[7] found that contrast material failed to reach the affected nerve foot in 33 per cent of patients with uncomplicated disc prolapse and that contrast did not rise in the epidural space above the affected disc in 4.9 per cent of cases. If water-soluble contrast material failed to reach these areas we presume that local anesthetics would find a barrier to spread as well.

In our case, we feel that scarring around the right L5 nerve root, perhaps in conjunction with the epidural venous engorgement of pregnancy,[8] prevented downward spread of local anesthetic through the right side of the epidural space. Injection below the level of obstruction allowed bilateral spread of anesthetic through the lower lumbar and sacral segments, producing sensory blockade of all segments, including the damaged L5 root on the right. This phenomenon has been fairly common in our experience with epidural steroid injections for sciatica. When injections of steroid and local anesthetics above the level of disc herniation do not produce analgesia in the affected dermatome, repeating the injection at a lower interspace, or via the caudal route will usually be successful.

In conclusion, our case illustrates that unblocked segments may be due to barriers to epidural diffusion caused by intervertebral disc disease and that the placement of a second catheter on the other side of the unblocked segment can produce adequate analgesia.

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Carotid Sinus Syndrome: Intraoperative Management Facilitated by Temporary Transvenous Demand Pacing

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Carotid sinus syndrome is an uncommon entity in which symptomatic cardiovascular instability results from minimal mechanical stimulation of the carotid sinus baroreceptors. Surgical denervation of these receptors may be undertaken in patients with disabling symptoms; however, cardiovascular instability may be a major intraoperative problem. We report a case in which the elective, preoperative placement of a transvenous demand pacemaker was essential to the management of a patient undergoing surgical denervation of the carotid sinus.

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Report of a Case

A 64-year-old man was admitted for surgical denervation of his right carotid sinus, after prolonged asystole was documented electrocardiographically during massage of the right carotid sinus. He had a six-month history of progressively more frequent and severe syncopal episodes upon turning his head to the right or raising his right arm. A right carotid endarterectomy had been performed three weeks prior to the onset of his first syncopal symptoms. Noninvasive assessment of the cerebral circulation by oculoplethysmography revealed normal ocular pressures bilaterally. Apart from the syncopal episodes, there were no

other symptoms suggestive of transient ischemia and the neurological evaluation revealed no deficits. The medical history was significant only for a myocardial infarction three years previously, with neither postinfarction dysrythmias nor congestive heart failure. Direct myocardial revascularization had been performed one week after the carotid endarterectomy, with complete relief of his previous anginal symptoms.

On the evening prior to surgery, the patient was admitted to the Intensive Care Unit where peripheral intravenous and percutaneous radial arterial catheters were inserted and the standard lead 2 of the EKG was monitored. A transvenous demand pacemaker was inserted via a cut-down in the left antecubital fossa and good pacing capture was noted at 0.5 mA with a heart rate of 100 beats/min. With the pacer turned off, right carotid massage decreased arterial blood pressure from 126/80 to 00/00 mmHg and eight seconds of asystole were observed. This resolved spontaneously to a sinus rhythm with a rate of 85 beats/min. With the demand pacer set at 60 beats/min and 0.5-mA stimulus intensity, right carotid massage resulted in a decrease in blood pressure to 73/43 mmHg with a paced heart rate of 60 beats/min. Left carotid message produced no changes in blood pressure or heart rate.

In the operating room arterial blood pressure and the precordial lead V_5 of the EKG were monitored continuously. Anesthesia was induced with intravenous diazepam and meperidine, with nitrous oxide 70 per cent administered via face mask. After paralysis was achieved with 0.1 mg/kg pancuronium, the trachea was intubated. The EKG showed a stable sinus rhythm and rate; cervical plexus block was performed with 10 ml of 0.5 per cent bupivacaine with 1:100,000 epinephrine. Ventilation was controlled and normocapnea maintained as documented by serial analysis of arterial blood gases.

Scarring from the previous carotid endarterectomy made surgical dissection difficult. Despite repeated injections of 1 per cent lidocaine into the carotid sinus by the surgeons, periodic hypotension and bradycardia were noted, resolving with cessation of surgical traction on the sinus. During the dissection, the carotid artery was inadvertently entered with an acute blood loss of approximately 1,500 ml. Temporary control of the hemorrhage was obtained by compression of the carotid artery. This resulted in a decreased heart rate to a paced ventricular rate of 60 beats/min, and a decreased arterial blood pressure to 70/40 mmHg. Rapid crystalloid infusion and a phenylephrine infusion were instituted and the blood pressure increased to 80/60 mmHg, with the heart rate remaining paced at 60 beats/min. There was no EKG evidence of atrial activity. Increasing the pacer stimulus frequency to 90/min immediately restored the blood pressure to 130/80 mmHg while the arterial laceration was repaired with the carotid artery cross-clamped. After further dissection, the denervation was completed and the demand rate of the pacer was decreased to 60 beats/min. A spontaneous sinus rate of 72 beats/min returned and the blood pressure was stable at 120/80 mmHg without vasoactive drugs. The trachea was extubated in the operating room with a grossly normal neurological examination. No further hemodynamic instability was observed, and carotid massage failed to produce either bradycardia or hypotension 24 hours later, at which time the pacemaker was removed.

Discussion

Carotid sinus syndrome is caused by an exaggeration of the normal physiologic function of the carotid baroreceptors. Several reviews have summarized the anatomy and physiology of these structures.[1-3] Stimulation of these receptors by massage in normal subjects results in a slight bradycardia and a minimal decrease in blood pressure. In 1933, Weiss and Baker reported several patients whose syncopal symptoms could be reproduced by lateral neck massage.[5] Franke defined criteria for carotid sinus hypersensitivity as asystole lasting longer than three seconds or a depressor response of greater than 50 mmHg below baseline blood pressure following 20 seconds of carotid sinus massage.[5] Symptomatic patients with these responses are classified as having the carotid sinus syndrome. Although the syndrome is uncommon, it is a known complication of carotid endarterectomy and also is associated with tumors in the neck and vascular disease in general.[6-8]

Two distinct types of cardiovascular responses may be noted with carotid sinus hypersensitivity. The cardioinhibitory reflex is mediated through the vagus nerve. Vagal efferent stimulation causes SA nodal suppression or AV nodal block, and a subsequent bradycardia.[3-9] The vasodepressor reflex is mediated by inhibition of the sympathetic vasomotor center, with a resultant decrease in systemic vascular resistance and in blood pressure.[6] These two reflex components may exist separately or in conjunction, and either may result in syncope. Walter *et al.*[3] noted that 80 per cent of patients with carotid sinus syndrome had only the cardioinhibitory component, 10 per cent had only the vasodepressor component, and 10 per cent had both components.

Distinguishing the variant of carotid sinus hypersensitivity is crucial. Treatment by permanent pacemaker implantation or with anticholinergic drugs may give symptomatic relief with the cardioinhibitory type of syncope, but will be ineffective in the vasodepressant or combined types.[6,10,11] Ventricular pacing or atropine administration during carotid massage will unmask the vasodepressor component of the carotid sinus syndrome, and these maneuvers have been recommended as part of the initial evaluation of carotid sinus syndrome.[3]

The treatment of carotid sinus syndrome may be pharmacologic, electrical, or surgical. Anticholinergic agents and pressor drugs are limited in their utility by cardiovascular and central nervous system side-effects, and are rarely effective with the vasodepressant or combined variants. Permanent transvenous pacing has been employed since Voss and Magnin reported the technique in 1970[13] and avoids many of the problems posed by long term drug therapy.[14-19] Pacing has complications, and does not treat the cardiodepressant component of the sinus reflex. Since the majority of patients have only the cardioinhibitory type of carotid syncope, implantation of a permanent demand pacemaker is the initial treatment of choice for most patients with carotid sinus syndrome. In patients whose vasodepressor response is refractory to such ventricular pacing, surgical ablation of the carotid sinus receptors may be attempted.[20-22]

Surgical management is often complicated by intraoperative dysrhythmias and hypotension. Injection of the

sinus with local anesthetics prior to dissection has been recommended in order to attentuate the carotid sinus reflex.[8,20] This decreases cardiovascular instability in some cases, but may be unreliable, as in our case. Local anesthetic blockade of the sinus reflex may also make the perioperative evaluation of the completeness of denervation difficult.[22] Postoperative hypertension has been reported after bilateral carotid sinus denervation,[23] although this is relatively uncommon and of short duration.

This case presents several of the problems associated with the surgical management of carotid sinus syndrome. The patient demonstrated a combination of both components of the carotid sinus relfex. In addition, he had both coronary and extracranial cerebral vascular disease, conditions frequently associated with carotid sinus syndrome.[6,12] Although he had an apparently successful carotid endarterectomy of the involved side and had an uncomplicated myocardial revascularization procedure, we felt that his limits of tolerance for prolonged hypotension or tachycardia might be critically narrow. Intraoperative elicitation of the carotid sinus reflex, initially by the surgical stimulation of dissection and later with pressure during control of the hemorrhage from an inadvertant carotid arteriotomy, led to bradycardia and hypotension despite direct infiltration of the sinus with local anesthetic. The importance of both cardiac and vascular components of this reflex was demonstrated by our inability to restore normal blood pressure even after rapid crystalloid and phenylephrine infusion until the heart rate was raised by ventricular pacing. Atropine, epinephrine, or isoproternol could have been used to elevate heart rate, but we were concerned that the resultant uncontrolled tachycardia might lead to myocardial ischemia in this patient. Additionally, in the absence of a pacer, the asystole secondary to prolonged sinus stimulation might have required cardiopulmonary resuscitation for distribution of these cardiac stimulant drugs before they could be effective. The ability to precisely control heart rate with the pacemaker and systemic vascular resistance with the phenylephrine infusion permitted good control of systemic blood pressure while minimizing the risks of myocardial ischemia during replacement of the blood loss.

In summary, the intraoperative management of carotid sinus syndrome requires attention to the potential cardiovascular instability caused by surgical stimulation. We conclude that preoperative evaluation should include identification of the presence of both cardioinhibitory and vasodepressor reflex components. A high probability of associated coronary vascular disease should be considered. Intraoperatively, local anesthetic blockade of the sinus may be helpful but is not reliable. Intraoperative use of a transvenous demand pacemaker (inserted preoperatively) enabled us to reduce the risk of pharmacologic overshoot in this high risk patient and to gain precise control of heart rate at a time when cessation of carotid sinus stimulation was not possible.

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