Cervical Sympathetic Block to Reverse Delayed Ischemic Neurological Deficits After Aneurysmal Subarachnoid Hemorrhage

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Background and Purpose—The purpose of the present study was to evaluate the feasibility and safety of a locoregional cervical sympathetic block to improve cerebral perfusion in patients suffering from cerebral vasospasm after aneurysmal subarachnoid hemorrhage.

Methods—Nine consecutive patients with symptoms of delayed ischemic deficits, induced by angiographically confirmed cerebral vasospasm, were treated with the injection of locoregional anesthesia to block the ascending cervical sympathetic chain at the level of the superior cervical ganglion. Neurological status was recorded before and after the procedure, and cerebral angiography was performed before and after the procedure.

Results—No complications occurred in this short series. The procedure appeared to be simple and safe. Horner’s signs appeared within 12/11006 0.1 minutes and lasted for an average of 6.3/11006 4 hours. In all patients, improved cerebral perfusion was detected at the confirmatory angiography but without change in vessel caliber. One patient died of the complications of the initial hemorrhage, and 2 died of the consequences of the severe vasospasm despite maximal medical treatment. In all the other cases, the neurological status promptly returned to normal within 48 hours after the locoregional treatment.

Conclusions—Patients with mild to moderate symptoms seem to benefit greatly from transient ipsilateral cervical sympathetic block. This simple technique may be helpful when used as an adjunct to the standard therapy to improve cerebral perfusion. (Stroke. 2003;34:961-967.)

Key Words: anesthesia, local ■ autonomic nerve block ■ cerebrovascular disorders ■ human ■ vasospasm, intracranial

Cerebral vasospasm as a complication of subarachnoid hemorrhage (SAH) seriously aggravates the vital prognosis, with a 1.5- to 3-fold increase in mortality in the first 2 weeks after SAH. In survivors, it is the major cause of disability, with a 25% decrease in excellent outcome.1-4 In the presence of cerebral vasospasm, current management consists of hypervolemic, hypertensive, hemodilutional (HHH) therapy and neuroradiological procedures.5-12 The results of these treatment options have been hampered by some technical limitations and the occurrence of medical complications and have demonstrated a variable effect on outcome.13-19 Therefore, new approaches toward improving cerebral blood flow are under continuous investigation.

The cerebral vasculature, in particular the pial vessels, is densely supplied with noradrenergic sympathetic nerve fibers mainly originating in the superior cervical ganglion, accompanying the carotid artery, and projecting into the ipsilateral hemisphere.20-22 Intracerebral vessels constrict in response to cervical sympathetic stimulation and dilate when these fibers are interrupted. Efflux and reuptake of the neurotransmitter may be prevented by sympathectomy.

In the present early report, we describe for the first time the use of a simple locoregional anesthesia at the superior cervical ganglion to prevent worsening and reverse neurological symptoms in patients suffering from cerebral vasospasm. We postulated that, during symptomatic vasospasm, the anesthetic-induced chemical interruption of the ascending cervical sympathetic chain might improve cerebral perfusion for an extended period of time in the carotid territory and, as a result, relieve neurological symptoms.

Subjects and Methods

The study was approved by our Institutional Ethical Committee for Human Subjects and was a prospective open intervention trial over a 1-year period. Consent was obtained from the patient or, in case of inability to cooperate, from a family member.

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Inclusion Criteria
Patients suffering from aneurysmal SAH and developing symptoms of vasospasm were considered for inclusion in the study. Symptomatic vasospasm was defined as follows: new onset of focal neurological deficit or deterioration in the level of consciousness after exclusion of other possible causes of deterioration, such as rebleeding, hydrocephalus, surgical complications, cerebral edema, electrolyte disorder, infection, or seizure. Cerebral angiographic confirmation of the presence of either proximal or distal vasospasm, compatible with the clinical presentation, was also required for eligibility. Locations of vasospasm considered for treatment were either proximal on the main segments of cerebral arteries, more distal to the first segment of the internal carotid branches, or both.

Exclusion Criteria
Exclusion criteria were as follows: asymptomatic patients with vasospasm demonstrated incidentally at angiography; refusal of consent; history of allergy to local anesthetic agents; and coagulation disorders with a prothrombin time <70% and/or platelet count <70x10^9/L.

Postoperative Management
All patients underwent early aneurysm treatment and were treated in the postoperative period with a standardized protocol that included immediate postoperative intensive care monitoring, normotension and avoidance of hypotensive events, fluid resuscitation to maintain normovolemia (defined as a positive fluid balance >500 mL/d), and spontaneous hemodilution at 0.3 hematocrit. All patients received oral nimodipine for 3 weeks. Analgesia was provided as needed, and sedation was avoided if possible unless signs of intracranial hypertension were present. Patients were assessed hourly for vital and neurological signs during the first 3 postoperative days. Serum electrolytes, plasma protein, and fluid balance were monitored daily. Patients presenting with good neurological status were equipped only with an arterial line to monitor blood pressure. Invasive monitoring was initiated only if more aggressive treatment was required on the basis of clinical symptoms. When there was clinical suspicion of vasospasm, the diagnostic evaluation to rule out vasospasm included the performance of a cerebral CT scan, and, in the absence of ventricular dilation or another focal brain lesion explaining the clinical symptoms, an emergency angiographic study was performed within 1 hour or less. At this point, the decision was made to enroll the patient in the study.

Study Protocol
After inclusion of the patient in the study and while the patient was still in the angiography suite, locoregional anesthesia to block the superior cervical ganglion was administered with the use of the technique described below. Before performing the cervical block and after its onset, a senior neurosurgeon (A.R.) assessed the neurological status. Subsequently, a second angiography was performed for comparison with the initial examination. Before the neuroradiologist proceeded with the second angiography, hemodynamic variables, blood gases, and temperature were recorded to ensure that the values were similar to those recorded during the initial examination, and, if not, they were corrected accordingly. If the angiography demonstrated an improvement in the cerebral circulation and/or relief of neurological symptom was observed, the treatment was considered satisfactory. In case the treatment effect was deemed insufficient, additional endovascular procedures including balloon angioplasty, intra-arterial papaverine injection, or a combination of both procedures was performed as appropriate, according to the usual care protocol. After the completion of the protocol, the patients were transferred to the surgical intensive care unit for continuous monitoring of mean arterial blood pressure, heart rate, right atrial or pulmonary artery occlusion pressures if a pulmonary artery catheter was in place, and temperature. Neurological assessment was performed hourly during the 48 hours after the procedure. Survivors were followed after hospital discharge, and 3-month outcome was recorded in all patients.

Digital Subtraction Angiography Evaluation Technique
Cerebral angiography was performed by a transfemoral approach with a high-resolution biplane digital subtraction technique (BV 3000, Philips, PMS). A bolus injection of a total of 30 mL of contrast material (Iopamiro 300, Bracco) was infused at the level of the ascending aorta through a 5F pigtail catheter via an automatic pump and coordinated with the acquisition of the images that were centered on the head in the anteroposterior projection with a moderate craniocaudal inclination (Town projection). The contrast infusion started automatically 2 seconds after the beginning of the image acquisition process. Delivery rate was 15 mL/s during 2 seconds. Sequences were obtained with 2 images per second during the first 10 seconds and then 1 image per second during the following 10 seconds. The following semiquantitative aspects were considered and evaluated at each of the angiographic phases (arterial, capillary, and venous): arterial caliber, velocities of arterial filling with comparison of left with right side, capillary stain (parenchymal) defects, cortical supply, and venous stasis. Vasospasm was classified as mild in the presence of <50% vessel caliber reduction, as moderate if vessel diameter reduction was 50%, and as severe if the reduction was >50%. We assessed filling velocities during the arterial phase by estimating the time from the start of the injection of the radiological contrast to the initial filling of the anterior cerebral arteries or the middle cerebral arteries, whichever occurred first (arterial delay). The parenchymal phase was defined as the time between the arterial and the venous phases. The characteristics of this phase were quantified as the intensity of opacification observed at the nadir of the capillary phase. Parenchymal defects were defined as an incomplete capillary staining resulting in a poor or absent visualization of any parenchymal area and were graded from 0 (normal) to 4 (absence of perfusion). We also recorded the time from initiation of the venous filling until drainage of the transverse sinus.

To ensure unbiased interpretations of the radiological imaging, a 2-block randomization was generated to assign blindly the preblock and postblock sequence of the angiography. Blinding was achieved by accurate concealment of patient identification, examination date, and time, with the exception of the time in seconds (equal in both examinations) to allow for calculation of the circulation time. A neuroradiologist (J.B.M.) blinded to the sequence of the angiography, patient identification, clinical status, and outcome interpreted the images after study completion.

Cervical Sympathetic Nerve Block Technique
According to individual anatomy, 2 approaches were considered, either lateral or medial to the neck vascular structures. After injection of a local anesthetic to the skin, a 25-gauge spinal needle 3.5 inches in length (89 mm) was advanced slowly until the tip was visualized in the anteroposterior projection to be close to a line halfway between the line formed by the lateral border of the vertebral bodies and the line formed by the transverse processes. In lateral projection, the needle tip was directed close to the line formed by the anterior contour of the vertebral bodies. Before the injection of the anesthetic solution, 1 mL contrast material was injected under fluoroscopic control to verify that the needle position was close to the prevertebral fascia and the neural sheath at the C2-C3 level (Figure 1). A 12- to 15-mL solution of bupivacaine 0.5% containing 50 μg of clonidine was then introduced in a single slow injection.

During the performance of the superior cervical ganglion block, the patient’s neurological status was monitored continuously, along with arterial blood pressure, surface ECG, oxygen saturation, and temperature. Fifteen minutes were allowed for the block to take effect, during which peripheral sympathetic signs were monitored to recognize the onset of a Horner’s sign (ipsilateral miosis, ptosis, anhydrosis, and conjunctival hyperemia). If sympathetic block was not established, the protocol was aborted, and routine procedures resumed.

Statistical Analysis
The end point of the study was clinical improvement of neurological symptoms and cerebral perfusion improvement assessed by cerebral
angiography. Continuous variables were compared before and after the cervical block with the use of the Wilcoxon signed rank test if the distribution was nonnormal or the 2-tailed paired t test. Dichotomous variables were analyzed before and after the procedure with the McNemar test for paired binary data (exact probability).

Results
During the study period, 42 patients with a ruptured intracranial aneurysm and 14 with an unruptured intracranial aneurysm were admitted and treated at Geneva University Hospital. Nine consecutive patients presented postoperative symptomatic vasospasm confirmed by cerebral angiography, and all were included in the study. Patients’ baseline demographic characteristics are shown in Table 1. The group included 4 patients harboring an aneurysm on the anterior cerebral artery complex, 3 on the carotid–posterior communicating artery complex, and 2 on the middle cerebral artery bifurcation. The arterial territory demonstrating vasospasm could explain the clinical symptoms, with the exception of 1 case presenting diffuse cerebral arterial spasm but unilateral symptoms. In this case, the block was performed on the side corresponding to the clinical manifestations. In patient 5, a bilateral block was performed under general anesthesia.

In all cases, the block was technically simpler when we used an anterior approach with the needle passing just medial to the neurovascular sheath of the carotid-jugular compartment. The average amount of anesthetic solution injected was $12.6 \pm 1.6 \text{ mL}$. Signs of cervical sympathetic block presented on the blocked side $12 \pm 0.1 \text{ minutes after the anesthetic injection}$. Ptosis was observed in 5 patients, miosis in 7, and hyperemia in 2; in 2 patients clinical signs were not assessable because of general anesthesia. The average duration of sympathetic block signs was $6.3 \pm 4 \text{ hours}$. Hemodynamic and oxygenation variables before and after the block were stable: mean arterial pressure was $85 \pm 6 \text{ mm Hg}$ before and $82 \pm 10 \text{ mm Hg}$ after ($P=\text{NS}$), heart rate was $75 \pm 25 \text{ bpm}$ before and $79 \pm 25 \text{ bpm}$ after ($P=\text{NS}$), and oxygen saturation was $99 \pm 1\%$ before and after the block.

The blinded neuroradiologist correctly identified the pre-block and post-block sequence with which the cerebral angiographies were performed with no exceptions. The post-block control angiography was performed $19 \pm 0.1 \text{ minutes after the injection of the anesthetic solution}$. An improvement in cerebral perfusion was confirmed in all patients in the postblock angiography (Figure 2). The examination demonstrated decreased filling time of the proximal intracranial carotid arteries on the blocked side and reduced circulation time compared with baseline and compared with the opposite

Table 1. Baseline Clinical Characteristics of 9 Consecutive Patients Included in the Study (Mean±SD)

<table>
<thead>
<tr>
<th>Age, y</th>
<th>41±17</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (F/M), n</td>
<td>8/1</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>74±26</td>
</tr>
<tr>
<td>Height, cm</td>
<td>167±10</td>
</tr>
<tr>
<td>WFNS grade, n</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>3</td>
</tr>
<tr>
<td>II</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>0</td>
</tr>
<tr>
<td>IV</td>
<td>0</td>
</tr>
<tr>
<td>V</td>
<td>3</td>
</tr>
<tr>
<td>Fisher grade, n</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>1</td>
</tr>
<tr>
<td>III</td>
<td>4</td>
</tr>
<tr>
<td>IV</td>
<td>3</td>
</tr>
<tr>
<td>Admission GCS</td>
<td>11.2±4.8</td>
</tr>
<tr>
<td>Time from hemorrhage to surgery, d*</td>
<td>0.5±0.8</td>
</tr>
<tr>
<td>Postsurgical GCS</td>
<td>13.4±3.2</td>
</tr>
<tr>
<td>Time from SAH to delayed ischemic deficit, d*</td>
<td>9.4±3.3</td>
</tr>
</tbody>
</table>

WFNS indicates World Federation of Neurological Surgeons’ classification for subarachnoid hemorrhage (SAH); GCS, Glasgow coma scale.

*One case: unknown date of bleeding.
side (Table 2). Distally, a nearly complete opacification of previously hypoperfused (defect) regions was observed. The delay in venous drainage was reduced, but this was not significant. However, at the selective catheterization, the vessel diameter was unchanged compared with baseline (Table 2).

In 6 conscious patients, immediate clinical improvement with complete symptom resolution was observed (Table 3). Three of the comatose patients had an increase in the Glasgow Coma Scale score by 1 point. However, 2 patients died despite the procedure and aggressive hypodynamic treatment with catecholamines. Patient 5, who presented with poor neurological status with severe intracranial hypertension, also underwent balloon angioplasty as an ultimate attempt to improve cerebral perfusion. Despite angiographic improvement and maximal treatment to maintain an adequate cerebral perfusion pressure, this patient died soon after the neuroradiological procedure.

No major complications attributable to the procedure were observed. Three patients complained of transient changes in voice and 1 of swallowing discomfort. One patient complained of headache lasting 12 hours after the procedure. Mean arterial pressure remained unchanged during the 24 hours after the procedure. In survivors, the 3-month outcome was excellent in 4 patients, 1 patient had mild residual paraphasia, and 1 patient developed multiple sclerosis with pure spinal cord involvement, unrelated to the preceding SAH.

### Table 2. Angiography Characteristics Before and After the Onset of the Cervical Sympathetic Block (Mean±SD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Angiography Before Cervical Block</th>
<th>Angiography After Cervical Block</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial delay, sec†</td>
<td>0.9±0.6</td>
<td>0.2±0.3*</td>
</tr>
<tr>
<td>Parenchymal defect‡</td>
<td>2.5±1.5</td>
<td>1.1±1.5*</td>
</tr>
<tr>
<td>Venous delay, sec§</td>
<td>2.5±2.6</td>
<td>1.9±1.7</td>
</tr>
<tr>
<td>Percent reduction in M1 caliber</td>
<td>57±16</td>
<td>56±11</td>
</tr>
<tr>
<td>Percent reduction in M2 caliber</td>
<td>55±12</td>
<td>54±16</td>
</tr>
</tbody>
</table>

*P<0.05.
†Time from the start of the contrast injection to the initial filling of the anterior cerebral artery.
‡Parenchymal opacification defect: 0: normal; 1: <0.5 cm; 2: 0.5–1 cm; 3: ≥1 cm; 4: no perfusion.
§Time from the beginning of the venous filling until the drainage of the transverse sinus.

Discussion

The present study shows that a noninvasive procedure of temporary chemical sympathectomy can contribute to improve cerebral perfusion in patients presenting symptoms of cerebral vasospasm. This technique was effective in reversing mild to moderate symptoms but was insufficient in treating very severe or advanced cases. However, because the morbidity related to vasospasm may be associated with mild symptoms, an effort should be made to correct even mild deficits with an approach free of adverse effects. The potential benefits for the patients are reduction of vasospasm-related morbidity and decreased exposure to aggressive treatments of interventional neuroradiology and/or HHH therapy. Because this technique is devoid of systemic effects, it may help to reduce the need for extreme hemodynamic end points to obtain reversal of symptoms, which is of particular concern in elderly patients. The treatment effect, either as alternative or complementary intervention, may be more sustained compared with the short-lived action of intra-arterial papaverine when dealing with distal and diffuse vasospasm. Moreover, the treatment can be repeated without the requirement of additional cerebral angiography.

The rationale for this procedure is that the blockage of sympathetic nerve activity or reversal of overactivity may dilate intracerebral vessels and improve cerebral blood flow, whether or not a sympathetic disorder contributes, among other mechanisms, to critically reduce cerebral blood flow. The vasodilatation induced by cervical sympathectomy has been shown to improve regional cerebral blood flow in healthy volunteers. Indeed, stellate ganglion block induced a definite ipsilateral increase in cerebral blood flow at cerebral
TABLE 3. Description of Symptoms Before and After the Onset of the Cervical Sympathetic Block

<table>
<thead>
<tr>
<th>Patient ID</th>
<th>Symptoms Description</th>
<th>Patient ID</th>
<th>Symptoms Description</th>
<th>Other Procedures</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Global aphasia</td>
<td>After Block</td>
<td>Complete resolution</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>Aphasia, right superior limb paresis, right-side numbness</td>
<td>Complete resolution of aphasia, paresis and numbness</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Confusion and dysphoria, expressing a frontal syndrome</td>
<td>Complete resolution of the frontal syndrome</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Right inferior limb paresis, bradypsychism</td>
<td>Complete resolution</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Decrease GCS from 14 to 5, severe hyperventilation</td>
<td>Transient improvement in GCS to 12, followed by a secondary worsening and death 4 days later</td>
<td>Balloon angioplasty and coiling of another aneurysm</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Confusion and dysphoria, due to a frontal syndrome</td>
<td>Complete symptom resolution within 12 hours</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Global aphasia</td>
<td>Complete symptom resolution within 48 hours</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Decrease GCS from 5 to 3</td>
<td>No improvement, life support withdrawal</td>
<td>Rebleeding, new clipping</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Decrease GCS from 12 to 9, right hemiparesis</td>
<td>Transient improvement of right hemiparesis, subsequent worsening 6 days later, loss of consciousness, and withdrawal of life support</td>
<td>No</td>
<td></td>
</tr>
</tbody>
</table>

scintigraphy. Since we did not observe a change in the caliber of major vessels, the increased distal cerebral perfusion possibly occurs as a consequence of a decrease in peripheral resistance. The improved distal perfusion is the same mechanism advocated for the action of calcium antagonists such as nimodipine.

Yasargil performed surgical ablation of the periadventitial sympathetic plexus from the arterial wall during craniotomy for intracranial aneurysmal clipping, thereby producing chronic denervation, and showed a low incidence of postoperative vasospasm. However, this technique is potentially dangerous because dissection of the adventitial layer may produce a weakening of the arterial wall, leading to the delayed formation of new aneurysms at this location. It is reasonable to assume that, compared with chronic denervation, acute sympathectomy obtained by a single injection of a local anesthetic agent has no effect on the endothelial modification (thickening of the wall, inflammatory changes) that can be observed as a delayed manifestation of cerebral vasospasm and that is probably modulated by disorders in the prostaglandin equilibrium.

No major complications were observed in this short series. In a previous report, no morbidity was reported when superior ganglion block was performed for other purposes, such as enhancement of hypothalamic function (eg, resumption of menses, cure for insomnia, stabilization of body temperature). Self-limited minor complications reported in relation to cervical ganglion block procedures include partial involvement of brachial plexus or recurrent nerve involvement, headache, nausea, sensation of warm face, fullness of ear, stuffy nose, and soreness of shoulder, neck, and jaw with facial hot flashes. Non-specific complications related to peripheral block procedures include hematoma at the site of injection, intravascular injection (which should be prevented by repeated aspiration and slow injection), and eventually puncture of the dura (injection should be performed slowly for early recognition). Local anesthetic toxicity is observed with acute intravascular injections, involves mainly the cardiovascular and central nervous system, and depends to a large extent on the rate of uptake by the circulation from the site of administration. In the present series all these potential events were prevented by the use of a small-caliber spinal needle and contrast-enhanced fluoroscopy control before injection of the solution. With the exclusion of allergic reactions and toxicity due to high dosages, local anesthetics are unlikely to cause side effects. The second agent added to the injected solution was clonidine. This α2-agonist prolongs the duration of the peripheral block and, at the dose administered, is not expected to cause any systemic effect.

In conclusion, cervical sympathetic block is a minimally invasive and simple technique that was demonstrated to be effective in improving distal cerebral perfusion and increasing cerebral circulation time, leading to resolution of mild to moderate symptoms of delayed ischemic deficits. Although the treatment effect alone is insufficient to restore adequate perfusion in severe and advanced cases, cervical sympathectomy may contribute to improve cerebral perfusion when used as an adjunct to standard procedures or when standard procedures are not applicable or contraindicated. A larger-scale trial that includes a control group comparison should be undertaken to firmly establish the value of the cervical block. Information obtained from such research would be useful to learn more about the role of the block within HHH therapy or endovascular treatment algorithms.

References

Sympathetic Block for Vasospasm

Vasospasm of the cerebral vessels is a major cause of neurological morbidity in patients who survive aneurysmal subarachnoid hemorrhage (SAH). Although the clinical and radiographic diagnosis of this condition has been well established, extensive investigation has failed to positively elucidate the pathophysiology and molecular basis for the development of this condition. Treatment by endovascular angioplasty and hypertensive, hypervolemic, hemodilution (HHH) protocols is not always effective, and, as a result, patients may suffer permanent cerebral ischemia. In the accompanying article, Treggiari et al report their results of a prospective study of the use of locoregional cervical sympathetic block in the treatment of symptomatic vasospasm after aneurysmal SAH. In their series, 9 of 42 consecutive patients with surgically treated aneurysmal SAH developed delayed neurological deficits in the presence of angiographically proven vasospasm. All underwent a cervical sympathetic block with a mix of 0.5% bupivacaine and 50 μg clonidine. Six conscious patients experienced immediate resolution of their symptoms, and each of 3 comatose patients improved their Glasgow Coma Scale score by at least 1 point. Postblock angiographic images demonstrated decreased filling time of the proximal internal carotid arteries and reduced circulation time on the injected side compared with preblock and contralateral images. A blinded neuroradiologist correctly identified the preblock and postblock images with no exceptions. There were no major complications attributable to the procedure. The authors conclude that cervical sympathetic block is a minimally invasive technique that is effective in the treatment of post-SAH symptomatic vasospasm.

Activation of the sympathetic system leading to an elevation in circulating catecholamines has been well documented in patients after SAH. This activation has been implicated in the development of cerebral vasospasm, yet there is no unequivocal linking evidence. Percutaneous blockade of the sympathetic chain at the superior cervical ganglion interrupts the innervation of adrenergic cerebral nerve fibers and can exclude any potential detrimental effect sympathetic hyperactivity may have in the occurrence and evolution of vasospasm.

The development of effective alternative approaches with reduced potential morbidity for the treatment of vasospasm is an important clinical issue. Through solid statistical testing and prospective analysis of cases, the authors of the accompanying article demonstrate that although the caliber of the involved


vessels does not change with the block, several independent angiographic characteristics such as arterial delay and the presence of parenchymal defects improved significantly. These angiographic changes correlated well with clinical outcome, with complete resolution of the neurological deficits immediately after sympathetic block in 6 of 9 patients.

This is a well-organized and well-performed study that appears to demonstrate the value (effectiveness and safety) of a relatively simple technique in the treatment of cerebral vasospasm. Despite the small total number of symptomatic patients, statistical significance in the treatment effects was observed.

A randomized comparison of this treatment with control saline injections or untreated groups, along with its role within HHH or endovascular treatment algorithms, needs to be further investigated to firmly establish its value.

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