Percutaneous cervical sympathetic block to treat cerebral vasospasm and delayed cerebral ischemia: a review of the evidence

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ABSTRACT

Delayed cerebral ischemia (DCI) affects 30% of patients following aneurysmal subarachnoid hemorrhage (aSAH) and is a major driver of morbidity, mortality, and intensive care unit length of stay for these patients. DCI is strongly associated with cerebral arterial vasospasm, reduced cerebral blood flow and cerebral infarction. The current standard treatment with intravenous or intra-arterial calcium channel antagonist and balloon angioplasty or stent has limited efficacy. A simple treatment such as a cervical sympathetic block (CSB) may be an effective therapy but is not routinely performed to treat vasospasm/DCI. CSB consists of injecting local anesthetic at the level of the cervical sympathetic trunk, which temporarily blocks the innervation of the cerebral arteries to cause arterial vasodilatation. CSB is a local, minimally invasive, low cost and safe technique that can be performed at the bedside and may offer significant advantages as complementary treatment in combination with more conventional neurointerventional surgery interventions. We reviewed the literature that describes CSB for vasospasm/DCI prevention or treatment in humans after aSAH. The studies outlined in this review show promising results for a CSB as a treatment for vasospasm/DCI. Further research is required to standardize the technique, to explore how to integrate a CSB with conventional neurointerventional surgery treatments of vasospasm and DCI, and to study its long-term effect on neurological outcomes.

BACKGROUND

Aneurysmal subarachnoid hemorrhage (aSAH) is a severe disease that affects six to nine per 100,000 people per year and results in significant morbidity and mortality.1 Brain injury at the time of aneurysm rupture is a significant cause of these poor outcomes, but cerebral artery vasospasm and delayed cerebral ischemia (DCI) are more insidious causes of neurologic injury, disability, and death. DCI is defined as the sustained neurologic deterioration that is not secondary to hydrocephalus, infection, or metabolic disarray.2 Vasospasm and DCI occur 3–14 days following aSAH, and the pathophysiologic basis of vasospasm and DCI are multifactorial and incompletely understood. It is likely that impaired cerebral perfusion secondary to arterial vasospasm, microvascular thrombosis, and cortical spreading depolarization result in significantly reduced cerebral perfusion.3 If cerebral perfusion is reduced significantly, cerebral infarction may occur, which may lead to permanent neurologic deficits and even death. Therefore, significant efforts are harnessed to identify patients at an increased risk of vasospasm and DCI.

Between 50% and 67% of patients with aSAH develop large cerebral artery vasospasm,4 but more than half of patients with arterial vasospasm do not develop neurologic symptoms. It is likely that these asymptomatic patients sustain sufficient cerebral perfusion to preserve neurologic function, whereas symptomatic patients with DCI likely suffer from more significant reductions in cerebral perfusion. Interestingly, studies have shown that endovascular and pharmacological treatment of large artery vasospasm have only a marginal impact on improving long-term neurological outcomes,5 6 which indicates that vasospasm is likely not the predominant cause of DCI.7 In support of this notion, the pattern of cerebral infarction due to DCI is often diffuse and frequently localizes to regions without proximal artery vasospasm.8

Limits of current treatments for vasospasm and DCI

Therapies for the treatment of vasospasm and DCI include medical and endovascular interventions. Medical therapy currently consists of permissive hypertensive therapy and administration of calcium channel antagonists.9 Oral nimodipine is the only class I, level of evidence grade A, therapeutic agent recommended by the American Heart Association/American Stroke Association for prophylactic treatment after subarachnoid hemorrhage. Permissive hypertension is another commonly performed therapy that carries a class I, level of evidence grade B recommendation.10 Nimodipine has a very modest effect on the development of arterial vasospasm, but it does lead to improved clinical outcomes when administered for 21 days after presentation with aSAH.

More recently, intrathecal administration of nimodipine has been explored as a treatment for vasospasm and DCI. This emerging treatment effectively prevents angiographic cerebral vasospasm, but the effect may be limited to vessels in close proximity to the drug administration or release site. Furthermore, this treatment has not yet been shown to improve long-term clinical outcomes.11
New devices and techniques

Other pharmacologic treatments have also failed to improve clinical outcomes significantly. For example, clazosentan is a selective endothelin-A antagonist that held promise as preventative treatment for DCI. The multicentric CONSCIOUS-1 randomized control trial showed that despite significant reductions in angiographic cerebral vasospasm, clazosentan did not decrease morbidity and mortality or improve functional outcome. Recently, two double-blind, placebo-controlled phase 3 studies in Japan demonstrated that clazosentan significantly reduced the combined incidence of vasospasm-related morbidity and all-cause mortality post-aSAH, but has not yet been approved for treatment globally. The use of other pharmacologic treatments such as intravenous magnesium, statins, and intravenous milrinone also showed no long-term benefit in patients with aSAH. In aggregate, these studies strongly suggest the pathophysiological mechanism behind DCI is likely to extend beyond pure large vessel vasoconstriction.

Endovascular therapy (EVT) treatment of vasospasm and DCI consists of trans-arterial endovascular procedures that aim to improve cerebral artery vasospasm and cerebral perfusion. The two most performed EVT treatments are (1) trans-arterial vasodilator infusion and (2) cerebral artery angioplasty. Vasodilator infusion is commonly performed with calcium channel blocking medications (nicardipine and verapamil) or milrinone. These medications are infused through arterial catheters that are commonly positioned within the cervical internal carotid artery or vertebral artery, proximal to the regions of cerebral artery vasospasm. This treatment does result in an improvement in cerebral artery vasospasm, but these medications have a relatively short half-life and may result in significant hemodynamic instability during infusion.

More aggressive EVT treatments of vasospasm include angioplasty using a balloon microcatheter or stent-retriever device, which are often performed in conjunction with trans-arterial vasodilator infusion. These techniques require microcatheter and device placement within the cerebral arteries, which carries an increased risk of an ischemic stroke and vessel dissection or laceration. Angioplasty is effective in arterial dilatation, but it is limited to the treatment of proximal cerebral arteries, and it does not directly treat the more distal arterial bed, which may also be affected by vasospasm.

Overall, although the use of EVT treatment strategies is reasonable in patients with symptomatic cerebral vasospasm, as suggested by the most recent guidelines, future prospective and randomized studies are warranted to demonstrate the impact of these therapies on clinical outcomes. New treatment strategies that improve cerebral blood flow (CBF), cerebral perfusion, and long-term clinical outcomes are sorely needed.

Sympathetic innervation, CBF regulation, and a potential treatment for vasospasm and DCI

The cervical sympathetic trunk is located on both sides of the cervical spine and runs along the prevertebral fascia posteriorly and the longus colli muscle anteriorly. It is composed of the superior, the middle, the stellate, and inconstant vertebral ganglia (figure 1). The cervical and cerebral arteries are well innervated by sympathetic nerves. Sympathetic trunk stimulation results in constriction of these arteries, which, in turn, reduces CBF. By contrast, sympathetic trunk inhibition or ablation results in vasodilatation and improved CBF.

Following aSAH, there is a massive activation of the sympathetic nervous system, which likely contributes to the development of vasospasm in these patients. The cerebral arteries have rich sympathetic innervation in the large cerebral arteries as well as within the smaller arteries and arterioles, which raises the possibility that sympathetic stimulation in aSAH patients may directly cause vasospasm that impacts the macro- and microcirculation of the brain. It also follows that sympathetic nerve blockade may release the vasoconstricting stimulus and improve CBF and cerebral perfusion (figure 2).

In 1936, Leriche described that a stellate ganglion block reversed cerebral vasoconstriction in a patient with cerebral vasospasm due to aSAH. More recently, Kim et al demonstrated in an animal model that superior cervical ganglion stimulation resulted in 20–30% reduction in mean ipsilateral CBF and that prior injection of lidocaine to the cervical ganglion inhibited the effects of this stimulation and restored normal cerebral perfusion. However, sympathetic nerve block techniques are not widely performed for the treatment of vasospasm and DCI.

We conducted a review on cervical sympathectomy to treat vasospasm and DCI in humans after aSAH to define the state of the literature on this topic and to identify knowledge gaps to guide future studies. Table 1 summarizes the characteristics of the studies included in this review.

Evidence that CSB is effective in treating vasospasm

Experimental models of aSAH demonstrated that CSB results in vasodilatation due to a reduction in sympathetic tone in the cerebral arteries. Tregridgi et al performed a study in which patients with symptomatic vasospasm underwent a baseline digital subtraction angiography (DSA) followed by a CSB. After
Evidence that a prophylactic CSB prevents DCI and improves clinical outcomes

The early evidence that CSB leads to cerebral artery dilatation and increased cerebral perfusion suggests that this technique may lead to a reduced risk of DCI and an increased likelihood of favorable clinical outcomes. Several single arm studies suggest that CSB leads to an immediate improvement in neurologic status and favorable rates of long-term clinical outcomes.

Treggiari et al. found an immediate improvement in neurologic status in DCI patients, which included complete reversal of neurological deficits in six out of nine patients. Jain et al. reported an improvement in Glasgow Coma Score 30 min after CSB and favorable long-term neurologic outcomes, as measured by the Glasgow Outcome Scale, in 73% of treated patients 6 months after aneurysm rupture. Samagh et al. found neurologic improvement in 25% of patients receiving a CSB. Zhang et al. were the first to administer a CSB prophylactically, before the occurrence of DCI, in patients undergoing ruptured aneurysm treatment by neurosurgical clipping. The authors randomized patients to either standard medical therapy or ultrasound-guided CSB the day of surgery and on post-operative days (PODs) 2, 4, and 6. They measured velocities with TCD in the MCA in the basilar artery (BA), markers of inflammation and of brain injury the day before surgery and on PODs 1, 3, and 7. Velocities in the MCA and BA were the same at baseline in both groups and significantly increased from POD 1 to POD 7; nevertheless, these increases were significantly lower in the group of patients receiving a CSB (P<0.05). The increase in velocities peaked at POD 7, with increases higher than 100% in the group not receiving a block, while in the CSB group the average increase was 50% compared with baseline. At the 6 months follow-up, 54% of patients in the CSB group had a favorable clinical course as measured by the Glasgow Outcome Scale compared with 32.6% in the control group (P=0.001). However, the authors never diagnosed vasospasm and DCI. They detected increased TCD velocities compared with baseline values, which were higher in patients who did not receive a CSB, but they never distinctly diagnosed vasospasm by non-TCD imaging or DCI by clinical symptoms, which is a significant limitation of this study.

These studies are promising and suggest that CSB may lead to improved short- and long-term neurologic outcomes. However, most of these studies have a small sample size, and did not correlate imaging biomarkers of vasospasm and cerebral perfusion to clinical outcomes. Therefore, further research is needed to confirm if a CSB consistently improves neurologic outcomes and by which mechanisms.

CSB technical considerations

The studies described above have varied slightly in the CSB technique, which introduces uncertainty as to the optimal method for performing the sympathetic block. Most authors report doing astellate ganglion block, which is commonly performed at the level of the sixth or seventh cervical vertebrae, whereas others have described a superior cervical ganglion block. These technical differences may seem relatively minor, but the anatomy and sympathetic innervation of the cerebral arteries is variable, which may impact the efficacy of the CSB. For example, there is evidence that the anterior cerebral circulation is innervated by sympathetic postganglionic fibers that originate from the superior cervical ganglion, whereas the posterior cerebral circulation derives its sympathetic innervation by postganglionic fibers originating from the stellate ganglion. Therefore, the anatomic targeting of the CSB may selectively impact the anterior versus posterior circulation.

CSB, patients underwent a repeated DSA to assess for changes in cerebral artery caliber. Interestingly, DSA studies done after CSB did not demonstrate a significant difference in arterial diameter but detected a decreased filling time of the proximal intracranial carotid arteries on the blocked side and a reduced circulation time compared with baseline and compared with the opposite side. These results suggest that CSB may induce small artery vasodilatation or other changes that improve CBF and cerebral perfusion. Pileggi et al. performed a similar study and detected distal circulation vasodilatation on CTA studies performed after CSB (Figure 3). However, single-phase CTA is insensitive to cerebral perfusion given the technique’s lack of temporal resolution, which is a limitation of these studies.

Samagh et al. insonated the middle cerebral artery (MCA) with transcranial Doppler (TCD) before and after CSB and observed a statistically significant reduction in velocities after CSB. Subjects in this study also underwent digital subtraction angiography, which demonstrated a significant increase in the mean vessel diameter measured at the mid-M1 segment of the MCA and at the mid-A1 segment of the anterior cerebral artery, and a decreasing trend in mean parenchymal filling time and mean venous sinus filling time after the sympathetic block, which also indicates vasodilatation of the microcirculation. Other studies have found reduced velocities in the MCA after a CSB, which is consistent with vasodilatation of this arterial bed after treatment.

Collectively, these data suggest that CSB results in vasodilatation of the cerebral arteries. It remains uncertain whether the CSB vasodilatory predominantly affects proximal or distal arteries of the cerebral circulation.

An improvement in cerebral artery caliber is expected to increase CBF and cerebral perfusion. However, only one case report in humans has directly measured cerebral perfusion changes after CSB. In this study, authors compared cerebral perfusion pre- and post-CSB on CT perfusion (CTP) studies. They found that CSB resulted in an improvement in cerebral perfusion, which is consistent with animal studies. However, this study is confounded by the intra-arterial administration of a vasodilator before the CSB. In summary, the available evidence consistently points to cerebrovascular dilatation, preferentially in distal beds, with variable changes in large vessel diameter.

Figure 2 Digital image of the circle of Willis at digital subtraction angiography pre- (left side) and post- (right side) treatment showing the expected vasodilatation in the territory of the middle cerebral artery after a sympathetic block.
<table>
<thead>
<tr>
<th>First author, year</th>
<th>Type of study</th>
<th>Number of patients, age</th>
<th>Block technique</th>
<th>Vessel diameter measurement</th>
<th>Time of vessel diameter measurement</th>
<th>Findings</th>
<th>Neurological changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treggiari et al., 2003</td>
<td>Prospective, observational</td>
<td>9 41±17</td>
<td>Fluoroscopy</td>
<td>DSA</td>
<td>To diagnose CAV and repeated after the block</td>
<td>Arterial delay, from 0.9±0.6 to 0.2±0.3 s (P&lt;0.05) and parenchymal defect from 2.5±1.5 to 1.1±1.5 s (P&lt;0.05) after the block. Unchanged vessel diameter</td>
<td>Clinical improvement with complete symptom resolution in 6 patients</td>
</tr>
<tr>
<td>Jain et al., 2011</td>
<td>Prospective, no control</td>
<td>15 45.5±13.6</td>
<td>Surface landmarks using anterior paratracheal approach</td>
<td>TCD</td>
<td>Before the block, 10 and 30 min, 2-6,12 and 24 hours after the block</td>
<td>Ipsilateral to CSB: mean MCA velocity, from 134 (8, SE) to 110 (6) at 6 hours and 113 (6) cm/s (P&lt;0.001) at 24 hours after the block; mean ACA velocity, from 106 (6) to 94 (5) at 6 hours and 96 (5) cm/s (P&lt;0.001) at 24 hours after the block. Contralateral to CSB: mean MCA velocity, from 96 (4) to 85 (4) cm/s (P&lt;0.001) at 6 hours; mean ACA velocity from 74 (5) to 69 (5) cm/s (P&lt;0.001) at 24 hours after the block.</td>
<td>Improvement in GCS after 30 min (p=0.002); 73% of patients had favorable outcome as measured by GOS at 6 months after SAH</td>
</tr>
<tr>
<td>Wendel et al., 2020</td>
<td>Retrospective</td>
<td>37 49.9±11.1</td>
<td>Surface landmarks using anterior paratracheal approach</td>
<td>TCD</td>
<td>CBFV before the block, and at 2 and 24 hours afterwards</td>
<td>Ipsilateral MCA velocity: from 160±28 cm/s to 127±34 at 2 hours, to 137±38 cm/s at 24 hours after the block corresponding to a 20% and 15% reduction</td>
<td>24 of the 37 patients significantly disabled (GOS ≤3) at ICU discharge, and moderately disabled (GOS 4) at 6 months follow-up</td>
</tr>
<tr>
<td>Pileggi et al., 2021</td>
<td>Retrospective</td>
<td>10 Mean age 50.2 years, range 37–66</td>
<td>Fluoroscopy</td>
<td>DSA</td>
<td>Before the block in all patients, and in selected patients after the block</td>
<td>Angiographic vasodilation on the distal circulation</td>
<td>Average clinical outcome measured by modified Rankin Scale at discharge was 3 indicating moderate disability</td>
</tr>
<tr>
<td>Zhang et al., 2021</td>
<td>Randomized controlled trial</td>
<td>5G (n=50) 51.4±2.22. Non-SGB (n=52) 53.7±1.74</td>
<td>Ultrasound</td>
<td>TCD in the MCA and basilar arteries</td>
<td>The day before surgery (POD 0) and on POD 1–3</td>
<td>MCA and basilar arteries’ velocities were higher on POD 1 and POD 3 compared with POD 0 in both groups, but this increase was lower in the SGB group (P&lt;0.05)</td>
<td>Better neurologic prognosis score and neurological function 6 months after surgery in patients who received an SGB</td>
</tr>
<tr>
<td>Samagh et al., 2022</td>
<td>Prospective, observational</td>
<td>20</td>
<td>Ultrasound</td>
<td>TCD and DSA</td>
<td>Before and after the block</td>
<td>TCD: reduction in ipsilateral systolic MCA velocity (from 150±22 to 140±24 cm/s, P=0.005), mean MCA velocity (from 120±23 to 108±25 cm/s, P=0.025), and Lindegaard ratio (from 2.57±0.46 to 2.34±0.50, P=0.022) after a CSB. DSA: increase in the mean vessel diameter measured at the mid-M1 segment of ACA (from 0.109±0.049 cm to 0.124±0.0517 cm, P=0.002), mid-A1 segment of MCA (from 0.174±0.046 cm to 0.189±0.0415 cm, P=0.003); decreased mean parenchymal filling time (from 3.18±0.37 s to 3.13±0.36 s, P=0.163) and venous sinus filling</td>
<td>Neurological improvement in 5 (25%) patients</td>
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ACA, anterior cerebral artery; aSAH, aneurysmal subarachnoid hemorrhage; CAV, cerebral arterial vasospasm; CBV, cerebral blood flow velocity; CBFV, cerebral blood flow velocity; CSB, cervical sympathetic block; DSA, digital subtraction angiography; GCS, Glasgow Coma Scale; GOS, Glasgow Outcome Score; ICU, intensive care unit; MCA, middle cerebral artery; POD, post-operative day; SAH, subarachnoid hemorrhage; SGB, stellate ganglion block; TCD, transcranial Doppler.
Cadaveric and imaging studies have shown a considerable anatomic variation in the localization of the cervical sympathetic ganglia compared with the transverse processes of the cervical vertebrae with respect to the most common ones reported. In practice, it is challenging to selectively perform a CSB at a specific ganglion, which is partially influenced by variable anatomy. In theory, if a CSB is accurately performed and the local anesthetic is delivered within the plane deep to the prevertebral fascia, between the longus capitis and longus colli muscles, the local anesthetic will diffuse to the nearby sympathetic trunk which is contained within this space.

An injection midway at the sympathetic trunk, at C6 at the level of the middle cervical ganglion, offers multiple advantages. The middle cervical ganglion lies in proximity to the anterior tubercle of C6, which is the more prominent cervical tubercle and therefore easier to visualize on ultrasound (figure 4). Anesthetic injection at the C6 level should block all the preganglionic fibers ascending from the spine such that sympathetic synapses located more cranially at the level of the superior cervical ganglion will be blocked. Thus, a CSB performed at the C6 level should effectively block sympathetic innervation of the anterior circulation. In addition, there is a high likelihood that local anesthetic administered at the C6 level will diffuse inferiorly to the C7 level, where the stellate ganglion is located. Therefore, a C6 CSB will also likely inhibit the stellate ganglion, such that the postganglionic fibers that enervate the posterior circulation will be blocked along with the anterior circulation.

By contrast, a selective superior cervical ganglion block may spare the fibers that enervate the posterior circulation. Recently, Kim et al21 showed that stimulation of the superior cervical ganglion results in a global reduction of ipsilateral perfusion, as measured by CTP, that is isolated to the anterior circulation territory. The posterior circulation was therefore not affected by this CSB of the superior cervical ganglion. There is a need for additional studies to determine how CSB anatomic location influences cerebral perfusion in the anterior versus posterior circulation of the brain.

**Modality of performing the technique**

CSB may be performed using a variety of anatomic and image-guided techniques. Anatomic landmark, ultrasound guidance, fluoroscopy, or open surgical resection may be used for CSB localization, and the way the CSB is performed may influence the efficacy of the technique. Ultrasound imaging has revolutionized the field of regional anesthesia and led to improved efficacy and safety of CSB due to improved real-time visualization of the anatomy being targeted by the procedure. In addition, ultrasound allows for the CSB procedure to be safely performed at the bedside in the intensive care unit, with rapidity and minimal equipment, supplies, costs, and risks. Although there are no data that have compared the efficacy of the different execution modalities of a CSB, anesthesiologists today prefer to use ultrasound guidance to perform nerve blocks as there is evidence that nerve blocks performed by ultrasound guidance are superior in terms of efficacy and fewer minor complications reported.

**Drug type, drug volume and drug delivery modality**

It is likely that the dose and delivery modality of the CSB anesthetic influences the efficacy and durability of the block. However, how these variables influence the impact of CSB on vasospasm and DCI has not been well described. Previous studies of CSB in aSAH patients are heterogeneous in terms of drug type, dose and volume. Studies used either 5–10 mL of 0.3% ropivacaine, or 8–10 mL of 0.2% ropivacaine, or 0.375% and 0.5% ropivacaine. Some authors added clonidine as an adjuvant to the local anesthetic to prolong the effect of a single shot injection, and one group of authors used an indwelling catheter infusing 0.2% ropivacaine at 5 mL/hour to prolong the effect of the block.

In addition, how the manner of drug delivery by a single injection versus a continuous infusion via an indwelling catheter influences vasospasm and DCI is poorly described. Two prior
studies describe placement of an indwelling periangiomatic catheter for continuous CSB, which may reduce the need for sequential CSB procedures given the relatively short half-life of the local anesthetic medications used in these procedures.

Whether infusion catheter placement is advantageous in vasospasm and DCI reduction or increases the risk of the CSB technique in aSAH patients requires further study.

Unilateral versus bilateral CSB
Vasospasm and DCI affect the entirety of the cerebral circulation, although vasospasm is often more severe in the region immediately adjacent to the ruptured aneurysm. The diffuse nature of vasospasm and DCI suggests that performing a bilateral CSB may be a superior technique, although unilateral versus bilateral CSB has not been directly compared in this patient population. However, bilateral stellate ganglion blocks may increase the risk of a profound bradycardia due to blockade of the cardioaccelerator fibers that originate from the stellate ganglia. A block attempting to be selective for the middle/superior cervical block will not have this side effect. Additional safety and feasibility studies performing bilateral CSB in patients with ruptured cerebral aneurysms are warranted.

CSB complications
In general, CSB complications are minor and self-limited. A study on 2000 stellate ganglion blocks performed using anatomic landmarks found no serious complications. Nevertheless, technical complications such as injury to the nearby nerves, such as the vagus nerve or the brachial plexus roots, and nearby viscera, such as the trachea and esophagus, during insertion of the needle have been reported. The use of ultrasound minimizes these risks due to real-time visualization of the needle. Non-specific complications of nerve blocks include infections, puncture of the dura, and intravascular injections of local anesthetic leading to toxicity, the latter of which is easily avoidable with ultrasound guidance.

Roadmap for future studies
The available evidence for CSB as an effective preventative and therapeutic treatment for vasospasm and DCI is promising. However, this understudied field remains in its infancy and requires more rigorous scientific exploration. Future studies should focus on (1) CSB technique standardization, (2) improving our understanding of how CSB improves cerebral perfusion in humans, and (3) providing stronger evidence that the technique improves immediate and long-term neurologic outcomes.

Additional knowledge is also required to understand how CSB might be integrated with conventional EVT treatment of vasospasm and DCI. Does CSB compliment or replace intra-arterial vasodilator infusion and angioplasty? Can CSB be used to reduce significantly the likelihood of developing vasospasm and DCI when performed shortly after presentation with aSAH? Well-conceived prospective studies are needed to address these pressing questions.

CONCLUSIONS
CSB is a minimally invasive, safe technique that may provide a new treatment for vasospasm and DCI in patients who have suffered cerebral aneurysm rupture. The initial studies described in this review suggest great promise for CSB, but there is significant work to do before the technique can be more widely performed in aSAH patients.


Moore DC, Bridenbaugh LD. The anterior approach to the stellate ganglion use without a serious complication in two thousand blocks. JAMA 1956;160:158–62.


New devices and techniques

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