Cervical Sympathectomy for Cerebral Vasospasm after Aneurysm Rupture

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Summary

The authors report a series of 11 cases of ruptured intracranial aneurysms followed by cerebral vasospasm manifesting neurological deficits. These patients were treated by cervical sympathectomy consisting of perivascular sympathectomy of the cervical internal carotid artery and superior cervical ganglionectomy on the same side as the cerebral angiospasm. Improvement of symptoms was observed following 13 procedures in 11 patients, within 10 hours in four cases, on the next day in five cases, and by the seventh day in two cases. In the remaining two cases no beneficial effect was observed. This technique was most valuable when it was done immediately after the appearance of the symptoms of infarction, before the development of irreversible brain damage.

Key words: aneurysm rupture, cerebral infarction, sympathectomy, ganglionectomy, vasospasm

Introduction

Cerebral vasospasm is observed angiographically as well as under direct vision on craniotomy in such conditions as head injury, intracranial inflammatory disease, and subarachnoid hemorrhage.^{4,7,8,12,14,16,31,39,41,51)} This phenomenon might be nature's defensive mechanism to prevent fatal bleeding from ruptured aneurysms. However, it may sometimes induce cerebral infarction characterized by paralysis, aphasia, and disturbances of consciousness.

In such cases the neurological deficits often cannot be controlled with vasodilators or other supportive medical cares. Thus the establishment of a clinical procedure for the management of cerebral vasospasm is one of the most urgent problems in practical neurosurgery.

There are many opinions as to the innervation of human cerebral arteries.^{11,13,25,35,50,53)} The electron-microscopic study by Sato and Suzuki³⁸⁾ identified numbers of myelinated nerves in the wall of human cerebral arteries, where vasospasm is commonly seen. In addition, degeneration of the adrenergic nerve elements in the ipsilateral cerebral arteries has been electron-microscopically demonstrated 28 to 48 hours after unilateral superior cervical ganglionectomy in animals.^{17,37)}

On the basis of these data, cervical sympathectomy was performed in 11 cases with ruptured aneurysm, symptoms of severe cerebral infarction and intracranial vasospasm demonstrated on carotid angiograms.

Technique

Perivascular sympathectomy of the cervical internal carotid artery associated with superior cervical ganglionectomy (Fig. 1) was carried out on the same side as the intracranial vasospasm. A skin incision should be made, avoiding a likely tracheostomy site when required. The internal jugular vein was retracted anterolaterally and perivascular denervation was performed starting 1 cm proximal to the carotid bifurcation and extending to the internal carotid as distal as possible, and including the beginning of the external carotid and the carotid body.

Fig. 1. Schematic illustration of a right cervical sympathectomy, showing the skin incision (*left*), pericarotid sympathectomy (*center*), and superior cervical ganglionectomy (*right*).

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Fig. 2. Intraoperative photographs of the right cervical sympathectomy (*left*) and pericarotid sympathectomy (*center*). Rt. vagus nerve (V), superior cervical ganglion (S), internal, external and common carotid arteries (IC, EC, CC). *Right*: The excised rt. superior cervical ganglion on the left, and pericarotid sympathetic plexus (P).

The superior cervical ganglion was excised completely as far as possible (Fig. 2). When this procedure was planned on both sides at once, anesthetic block of the nerve element was attempted prior to its excision for examination of a possible untoward effect on cardiopulmonary function.

Case Reports

Some illustrative cases are given in detail.

Case 1

On August 10, 1971, this 61-year-old woman felt a girdling pain in the occipital and neck region, followed by vertigo, blurred vision, weakness of the right leg and frequent vomiting. These symptoms disappeared on the next day. A spinal tap on August 31 revealed bloody cerebrospinal fluid (CSF); carotid angiograms demonstrated aneurysms of the right and left middle cerebral arteries. Cerebral vasospasm was not observed at this time (Fig. 3 *upper left*). On October 1, a ligature and a clip were successfully placed on the aneurysmal stalk bilaterally under hypothermic anesthesia at 27°C. The right and left middle cerebral aneurysms measured 6×5×5 mm and 3×3×4 mm respectively. A hematoma of 30 ml was also evacuated from the right Sylvian fissure.

Immediately after the direct aneurysm surgery, there was no neurological deficit nor disturbance of consciousness. However, the next day she suffered from left hemiparesis, and vasospasm of the beginning portion of the right

middle cerebral artery was revealed angiographically (Fig. 3 *upper right*). This symptom of cerebral infarction persisted for 17 days despite several supportive treatments with 20% mannitol solution, low molecular dextran, vasodilators, and stellate ganglion block.

On October 19, right cervical sympathectomy was carried out. On the next day, the motor paresis improved; angiospasm diminished on carotid angiogram (Fig. 3 *lower left*). Eight days later, the patient was discharged in good mental and physical conditions except for a right Horner's sign.

Case 2

On September 28, 1971, this 52-year-old woman had a severe bursting headache and lost consciousness for six hours. On examination, bloody CSF and an anterior communicating aneurysm were found (Fig. 4 *upper left*). The only neurological abnormality was neck stiffness.

On October 2, the aneurysm stalk was ligated through a bifrontal craniotomy under hypothermic anesthesia at 25.5°C. At craniotomy, arteriosclerosis and a hypoplastic horizontal portion of the right anterior cerebral artery were revealed; the aneurysm was irregular in shape and 10×8×8 mm to 2×3×2 mm in size. Postoperatively no neurological deficit was observed except for neck stiffness; however, the patient became aggressive, with illogical speech and pathological reflexes on the right side, and six days after aneurysm surgery she became completely aphasic. Left carotid angiography revealed diffuse vasospasm of the terminal portion of the internal carotid, anterior and middle cerebral arteries. Thalamic perforating branches were not visible at angiography (Fig. 4 *upper right*). A slight transient improvement of symptoms was observed, but she became hemiplegic. Stellate ganglion block caused only temporary improvement, and supportive treatment with vasodilator and low molecular dextran solution was not effective. On October 30, continuous ventricular drainage was instituted and left cervical sympathectomy performed. Seven hours after these procedures, the patient spoke several words logically and began to recover from the cerebral infarction. Angiography showed improvement of the vasospasm (Fig. 4 *lower right*).

Upper Left

Upper Right

Lower Left

Fig. 3. Case 1. Right carotid angiograms, lateral view. *Upper Left*: 44 days after aneurysm rupture. An aneurysm can be seen on the trifurcation of the right middle cerebral artery (*arrow*), with no vasospasm. *Upper Right*: Thirteen days after aneurysm stalk was successfully clipped. Right anterior and middle cerebral arteries and C₁ portion are noticeably spastic. *Lower Left*: Two days after the right cervical sympathectomy the spasm is diminishing.

Upper Left

Upper Right

Lower Left

Fig. 4. Case 2. Left carotid angiograms, anteroposterior view. *Upper Left*: Three days after the aneurysm rupture. The anterior communicating aneurysm can be seen with no vasospasm. *Upper Right*: Six days after the direct aneurysm surgery. The aneurysm has disappeared completely. Insufficient filling due to vasospasm of the carotid fork, anterior and middle cerebral arteries and thalamic perforating branches are noted. *Lower Left*: Six days after ventricular drainage and the left cervical sympathectomy. The vasospasm is diminished. Note also the improved visualization of the thalamic perforating branches.

Case 4

On October 24, 1971, this 48-year-old woman suffered from a severe diffuse headache, nausea, and vertigo. She was admitted three days later with a dull headache, a stiff neck, and in a delirious state. Examination revealed bloody CSF and a left middle cerebral aneurysm was demonstrated at angiography (Fig. 5 *upper left*). Nine days after admission, the patient began to speak illogically, and right hemiplegia was noted. The left carotid angiogram demonstrated marked vasospasm on the carotid fork (Fig. 5 *upper right*).

Continuous ventricular drainage was instituted and several hours later her speech and consciousness returned to nearly normal. Headache, neck stiffness, right hemiparesis, and papilledema were still noted. On October 31,

the patient became semicomatose despite patent and operating ventricular drainage. In spite of her desperate condition,^{45,46)} she was subjected to left frontal craniotomy followed by left cervical sympathectomy under normothermic anesthesia. An aneurysm measuring $5 \times 4 \times 4$ mm was clipped and a Sylvian fissure hematoma of 50 gm was evacuated. The next day, her consciousness improved and left carotid angiography showed abatement of the vasospasm. On the third postoperative day, she became alert and her motor paresis had disappeared. On November 12, vasospasm was no longer visible on the carotid angiogram (Fig. 5 *lower left*). She was discharged two weeks later in a very good condition except for a left Horner's sign.

Fig. 5. Case 4. Left carotid angiograms, antero-posterior view. *Upper Left*: Three days after the aneurysm rupture, showing the left middle cerebral trifurcation aneurysm (arrow), with no vasospasm. *Upper Right*: Eight days postrupture. Diffuse vasospastic narrowing of the anterior cerebral artery and carotid fork is seen; a portion of M_1 has been replaced with a collateral network. *Upper Left*: Complete disappearance of the vasospasm is shown 12 days after the left cervical sympathectomy. The arterial segments and the thalamic perforating branches are well visualized.

Case 7

On February 27, 1972, this 62-year-old woman suddenly groaned, vomited, and $1\frac{1}{2}$ hours later became semicomatose. The systemic blood pressure was 220/120 mm Hg. Examination on the following day showed neck stiffness. An anterior communicating aneurysm was demonstrated on carotid angiography. Bloody CSF with a pressure of 250 mm H_2O was noted on ventricular tapping, and continuous drainage was instituted. Two days later she was alert and well oriented.

On March 3, the aneurysmal stalk was successfully clipped and ligated under hypothermic anesthesia at $27.5^\circ C$. The right A_1 portion was very hypoplastic. Left cervical sympathectomy was also performed as a precaution against postoperative vasospasm. The day after surgery, the patient had no motor disturbance. On March 6, she became drowsy, with weakness of the left arm; the left A_1 portion appeared spas-

tic on angiography. Right cervical sympathectomy was carried out. Ten hours after this procedure, her consciousness seemed to have improved a little but the next day she was semicomatose and she died on March 9. At autopsy, marked brain swelling due to scattered infarction and softening in bilateral cerebral hemispheres were observed.

Case 10

On June 6, 1972, this 43-year-old woman suddenly vomited, and became unconscious for several hours. On the next day she was admitted to the hospital; bloody CSF was found at spinal tap. A left internal carotid-posterior communicating aneurysm was revealed on carotid angiograms. On June 21, she had a generalized tonic convulsion, resulting in right-sided hemiparesis and semicoma. At lumbar puncture, the CSF was clear; left carotid angiography revealed vasospasm with diffuse irregular narrowing at

the carotid fork and peripheral segments. Left cervical sympathectomy and tracheostomy were performed as emergency procedures. The next day her consciousness slightly improved but again deteriorated and she died on June 28, 1972. Autopsy revealed diffuse basal subarachnoid hemorrhage and marked swelling of the left hemisphere. The left internal carotid-posterior communicating aneurysm measured $7 \times 5 \times 5$ mm. On the cut surface infarction was noted at the left pallidum and parietooccipital cortices.

Case 11

This 36-year-old woman was admitted on October 2, 1972, three days after a subarachnoid hemorrhage. Right carotid angiograms taken four days after the hemorrhage revealed an aneurysm at the junction of the internal carotid and posterior communicating arteries, with vasospasm around the carotid fork (Fig. 6 *upper left*). She had been alert and had had no neurological signs except for moderate meningeal irritation until seven days after the hemorrhage when she became drowsy and developed progressive left hemiparesis. Right carotid angiography was repeated and showed more pronounced vasospasm in the same arteries than before (Fig. 6 *upper right*). Immediately after angiography the neck of the aneurysm was clipped, continuous ventricular drainage was instituted, and sympathectomy on the right side was carried out. Within six hours postoperatively the patient became alert and her hemiplegia improved markedly. On the third postoperative day she was no longer hemiplegic, and improvement of the vasospasm was noted on the angiograms (Fig. 6 *lower left*). She was discharged 14 days after the surgery without any neurological deficit except for right Horner's sign. Right carotid angiograms taken three months later showed entirely normal cerebral arteries (Fig. 6 *lower right*).

Summary of Cases

This series includes 11 cases operated on between September 1971 and October 1972. In two patients the procedures were bilateral. Clinical data are presented in Table 1. The interval between rupture of the aneurysm and cervical sympathectomy varied from five to 70

Fig. 6. Case 11. Right carotid angiograms, antero-posterior view. *Upper Left*: Five days after subarachnoid hemorrhage. An aneurysm is visualized at the junction of the internal carotid and posterior communicating arteries, with vasospasm in the carotid fork. *Upper Right*: Seven days after the hemorrhage when the patient became drowsy and hemiplegic. There is now pronounced vasospasm. *Lower Left*: Three days after the clipping of the aneurysm neck and cervical sympathectomy. C_1 and A_1 are less spastic and M_1 is rather dilated. *Lower Right*: Three months after surgery. The cerebral arteries now have an entirely normal appearance.

days. Symptoms of cerebral infarction as well as carotid angiographic evidence of vasospasm were observed prior to aneurysm surgery in Cases 4, 5, 6, 10, and 11. Of the cases in which symptoms of cerebral infarction appeared after the aneurysm surgery, all but one were alert; Case 3 was semicomatose. Ventricular drainage was performed in nine cases. In every case, symptoms of cerebral infarction due to vasospasm appeared three days or more after the rupture of the aneurysm.

In the cases where cerebral infarction appeared postoperatively, the vasospasm might

Table I. Clinical data of 11 cases manifested by symptoms of cerebral infarction due to vasospasm.
September 1971 through October 1972

Cases	Sign of cerebral infarction	Interval from aneurysm rupture to				Interval from sympathectomy to		Results and sequelae at discharge
		Sign of cerebral infarction	Vasospasm on carotid angiograms	Cervical sympathectomy	Direct aneurysm surgery	Ventricular drainage	Improvement of clinical sign	Improvement on carotid angiograms
1. 61-y-o F Blk. middle cereb. aneurysms	Lt. hemiparesis	53 days	65 days	70 days	52 days		1 day	<2 days
2. 52-y-o F Ant. commun. aneurysm	Aphasia Rt. hemiparesis	10 days	10 days	11 days	4 days	11 days	7 hrs.	<6 days
3. 38-y-o F Lt. int. carot. post. com. an.	Consciousness disturbance Rt. hemiplegia	3 days	7 days	7 days	2 days	1 day	3 days	3-14 days
4. 48-y-o F Lt. middle cereb. aneurysm	Illogical speech Rt. hemiplegia	7 days	8 days	10 days	10 days	8 days	1 day	<2 days
5. 36-y-o M Ant. commun. aneurysm	Lt. hemiparesis	6 days	13 days	13 days	10 days		6 hrs.	<2 days
6. 25-y-o M Lt. int. carot. post. com. an.	Consciousness disturbance Motor palsy	7 days	7 days	Lt. 7 days Rt. 15 days	9 days	7 days	Lt. 1 day Rt. ?	Lt. <2 days
7. 63-y-o F Ant. commun. aneurysm	Not marked	8 days	8 days	Lt. 5 days Rt. 8 days	5 days	1 day	Lt. none Rt. 10 hrs.	Lt. aggrav. Rt. ?
8. 37-y-o M Ant. commun. aneurysm	Monoparesis Mental disorder	6 days	7 days	35 days	2 days	2 days	7 days	<6 days
9. 41-y-o F Lt. int. carot. post. com. an.	Aphasia	18 days	17 days	19 days	9 days	19 days	1 day	<7 days
10. 43-y-o F Lt. int. carot. post. com. an.	Convulsion Rt. hemiparesis	9 days	10 days	10 days		10 days	1 day	<4 days
11. 36-y-o F Rt. int. carot. post. com. an.	Consciousness disturbance Lt. hemiparesis	7 days	5 days	8 days	8 days	8 days	6 hrs.	<3 days

have been caused by mechanical stimulation of the arterial wall,^{5,18)} intracranial bleeding,⁴⁴⁾ or the introduction of a so-called angiospasmogenic substance.^{7,19,20,36)} In Case 7 there was generalized cerebral arteriosclerosis, and prophylactic cervical sympathectomy was performed on the side of the dominant A₁ segment of the anterior cerebral artery; nevertheless the patient suffered from cerebral vasospasm leading to fatal cerebral infarction. Case 10 had an extremely severe infarction in one of the thalamic perforating branches manifested by convulsion. The sympathectomy should have been done immediately instead of six hours after the onset of infarction in such a fulminant case. The other nine cases improved following cervical sympathectomy, whereas other therapeutic efforts such as osmotherapy, low molecular dextran solution, vasodilator or stellate ganglion block had failed to have effects. Aphasia, motor palsy, or disturbance of consciousness diminished between six hours and seven days following the cervical sympathectomy. The angiographic evidence of the occurrence of spasmolysis is shown in Table 1. Angiographic visibility of the thalamic perforating branches as well as vasospasm on the carotid fork seemed to correlate significantly with cerebral infarction disorders.

Discussion

The existence of cerebral vasospasm has only recently been recognized, based on angiographic evidence or under direct visual evidence of craniotomy.^{1,4-6,8,10,12,14-16,18-20,23,30,31,34,41,42,47,48,51,52)} Not much is known of its fundamental mechanism, neurogenic participation, incidence, or so-called spasmogenic substance.^{7,19,20,47)} The search for a spasmolytic agent looks hopeful but it seems still a long way from clinical use.²⁹⁾

The therapeutic significance of cervical sympathectomy for cerebral ischemic disorders is equivocal.^{2,7,12,24-28,32,33,39,43,51)} Although, cervical sympathectomy was introduced as early as in 1952²⁴⁾ its application seems to have been limited to brain damage due to a certain organic vascular lesions, such as thrombosis or embolism.^{2,24,28,32,33)}

We feel neurogenic implication of this should be emphasized by both physiological

study^{3,9,21,22,39,40,49)} and recent electron-microscopic study of innervation of the cerebral arteries.^{36,38)}

In accordance with the view that the internal carotid area is innervated mainly by the superior cervical sympathetic nerve^{17,37,49)} and that stimulation of this nerve results in cerebral vasoconstriction,^{21,26,51)} we attempted therapeutic cervical sympathectomy in severe clinical cases with infarction following cerebral vasospasm. According to previous papers, cervical sympathectomy does not show blood flow increase in normal cerebral arteries, but this may be achieved in spastic vessels as in subarachnoid hemorrhage.

Prophylactic cervical sympathectomy in normal state may not be performed as a precaution against vasospasm because this may settle so-called "denervation hypersensitivity"^{11,51)} and may result in vasospasm as seen in Case 7.

This technique is still in a preliminary stage and should be proven further in a large series. It is our opinion that cervical sympathectomy is therapeutically valuable in some cases if done immediately after manifestation of cerebral infarction due to vasospasm and before cerebral disorder progresses to irreversible damage.

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