

# Cavernous sinus syndrome during balloon test occlusion of the cervical internal carotid artery

## Report of two cases

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✓ The authors report the occurrence of ipsilateral transient cavernous sinus syndrome during balloon test occlusion (BTO) of the cervical internal carotid artery (ICA) and discuss the involved pathomechanisms.

The authors reviewed their series of 129 BTOs of the ICA performed between 1989 and 1996. Two patients developed facial paresthesias and transient palsies of the third through sixth cranial nerves during test occlusion of the cervical ICA. The tests were performed prior to planned permanent carotid artery occlusion for the treatment of a neck sarcoma in one patient and a giant cavernous carotid artery aneurysm in the other. The patients' symptoms resolved with deflation of the balloon. When the balloon was subsequently inflated above the inferior cavernous sinus artery (ICSA), one of the patients complained of mild facial discomfort. There was no contralateral weakness or mental status change during test occlusion in either patient. Angiography demonstrated good filling of the ipsilateral intracranial circulation via collateral vessels of the circle of Willis.

In these two cases, the cranial nerves in the cavernous sinus were likely supplied by the ICA via the meningo-hypophyseal trunk and the ICSA. In each case, there was excellent blood supply to the ipsilateral cerebral hemisphere; however, there was probably inadequate retrograde filling of the cranial nerve collateral vessels located where the meningo-hypophyseal trunk and ICSA originated. These cases emphasize the importance of a patent external carotid artery-ICA connection for successful cervical carotid artery occlusion. Neurological examination during BTO was critical to interpret the clinical manifestations caused by the hemodynamic changes.

**KEY WORDS** • balloon test occlusion • carotid artery • cavernous sinus syndrome • endovascular therapy • neurological deficit

**B**ALLOON test occlusion (BTO) was introduced by Serbinenko<sup>7</sup> in 1974. This endovascular technique is commonly performed to evaluate the tolerance of the central nervous system to carotid artery occlusion. We reviewed our series of 129 BTOs of the internal carotid artery (ICA) that were performed from 1989 to 1996. Two patients developed ipsilateral transient palsies of the third through sixth cranial nerves during test occlusions. The tests were performed before planned permanent carotid artery occlusion for the treatment of a neck sarcoma in one patient and a giant cavernous carotid artery aneurysm in the other. The interpretation of this finding and its implications are discussed.

### Case Reports

#### Case 1

*History.* This 20-year-old man with rhabdomyosarcoma of the right parapharyngeal space and infratemporal fossa (Fig. 1) was admitted for BTO of the right ICA prior to

surgical resection. The patient complained of decreased hearing on the right side and occasional episodes of dizziness that were most often postural in nature.

*Examination.* On examination, the patient had a lobulated retromandibular mass extending to the mastoid process and a right-sided facial nerve paralysis, but no other neurological deficits. Right ICA angiography demonstrated filling of the anterior and posterior communicating arteries. Angiograms of the left vertebral artery and the left ICA did not show any abnormalities. There was no appreciable compression of the ICA.

*Balloon Test Occlusion.* In preparation for the test occlusion, intravenous heparin (5000 U) was administered and topical nitropaste was applied to the chest wall. A pediatric Swan-Ganz catheter (Edwards Laboratories Inc., Anasco, Puerto Rico) was placed and inflated in the right cervical ICA at the C1-2 vertebral level (Fig. 2, position 1). Heparinized saline was slowly infused through the distal lumen of the balloon catheter. After 10 minutes of ICA occlusion, the patient began to complain of pain in the

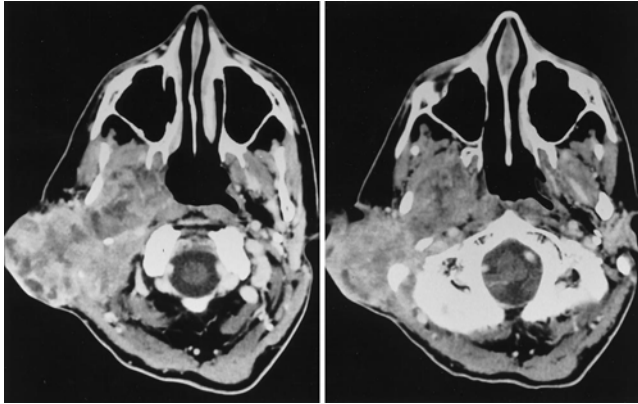


FIG. 1. Case 1. Contrast-enhanced computerized tomography scans demonstrating a large mass in the right parapharyngeal space and infratemporal fossa.

right orbit and face. Approximately 10 minutes later, after a total of 20 minutes of temporary occlusion, he developed a complete right-sided ophthalmoplegia. The balloon was deflated and the patient's symptoms resolved completely within 3 minutes.

To determine whether these transient deficits had developed as a result of diminished perfusion of the cavernous arterial branches from the ICA to the third through sixth cranial nerves, the test was repeated using a nondetachable silicone balloon (Interventional Therapeutics Corp., Fremont, CA) placed just above the origin of the inferior cavernous sinus artery (ICSA) (Fig. 2, position 2). After 10 minutes, the patient experienced mild facial numbness but less severe symptoms of paresthesia and no ophthalmoplegia. Once again, his symptoms resolved after balloon deflation. No evidence of right hemispheric signs or symptoms occurred during any portion of the BTO.

#### Case 2

This 28-year-old man presented with a severe headache and diplopia.

**Examination.** The patient's physical examination was remarkable for a left sixth cranial nerve paresis. Cerebral angiography performed immediately before BTO demonstrated a giant aneurysm arising from the cavernous portion of the left ICA (Fig. 3).

**Balloon Test Occlusion.** Before BTO, intravenous heparin was administered and nitropaste was applied. A pediatric Swan-Ganz catheter was placed and inflated in the left cervical ICA at the C1-2 vertebral level. Heparinized saline was slowly infused through the distal lumen of the balloon catheter. After 2 or 3 minutes of carotid occlusion, the patient complained of pain and pressure involving his left eye and, subsequently, of left facial numbness. Worsening of the left sixth cranial nerve paresis was evident after 10 minutes of occlusion, and complete ophthalmoplegia occurred after 20 minutes. The balloon was deflated and the patient returned to baseline within 2 minutes. The BTO was repeated in the same region of the ICA and the same symptoms started after 10 minutes of occlusion. There was no left hemispheric dysfunction throughout the procedure. We did not attempt test occlusion of the

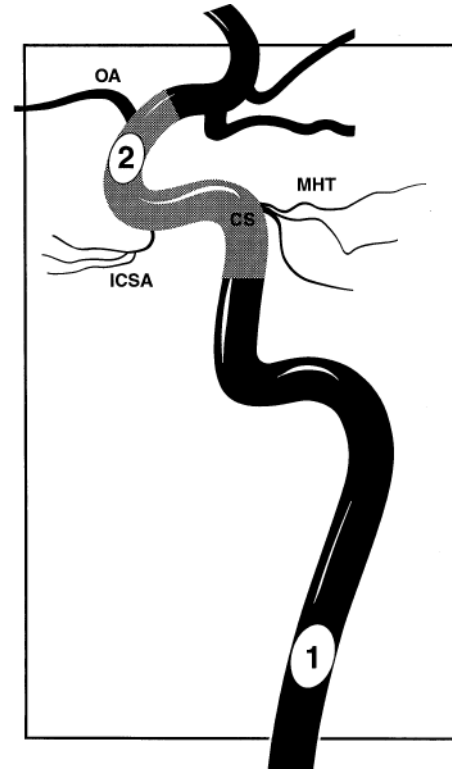


FIG. 2. Case 1. Schematic illustration of the two positions in which the balloon was placed for test occlusion. *Position 1:* Cervical ICA at the C1-2 vertebral level. *Position 2:* Above the origin of the cavernous ICA branches. Abbreviations: CS = carotid siphon; MHT = meningeohypophyseal trunk; OA = ophthalmic artery.

supraclinoid portion of the ICA. To demonstrate collateral circulation, a right carotid angiogram was obtained while the left cervical ICA was still occluded by the balloon. There was patency of the anterior communicating artery with filling of the left cerebral hemisphere. A left vertebral artery angiogram demonstrated filling of the middle cerebral artery bilaterally via the posterior communicating arteries. The giant left carotid cavernous aneurysm was not visualized during either injection, indicating that the reversed flow in the ICA did not reach the cavernous segment where the meningeohypophyseal trunk and ICSA are located. The BTO results suggested poor perfusion of the meningeohypophyseal trunk and ICSA with balloon occlusion below these vessels.

We elected to perform a left extracranial-intracranial arterial bypass prior to ipsilateral ICA balloon occlusion. Angiography performed through the bypass graft during simultaneous high-cervical BTO revealed some retrograde flow to the cavernous segment, but no filling of the aneurysm. No symptoms developed after permanent balloon occlusion. On postoperative evaluation, the patient's left sixth cranial nerve paresis was improving. His neurological status remained otherwise unchanged. Magnetic resonance imaging of the head demonstrated a thrombosed left cavernous ICA aneurysm.

## Cavernous sinus syndrome during balloon test occlusion



FIG. 3. Case 2. Left ICA angiogram, lateral view, showing a giant aneurysm at the cavernous portion of the left ICA.

### Discussion

To our knowledge, cavernous sinus syndrome has never been reported during BTO of the cervical ICA. Debrun, et al.,<sup>1</sup> reported a case of third nerve paresis after multiple balloons were placed in the cavernous sinus to occlude carotid-cavernous fistulas, which may have been related to local mass effect and balloon compression of the cranial nerve. We suggest that the cavernous sinus syndrome in our patients resulted from poor retrograde filling of the ICA cavernous segment. The same mechanism has been reported to cause transient monocular blindness in a patient with an ophthalmic artery originating from the cavernous sinus segment.<sup>3</sup> The monocular ischemic syndrome has been also reported as a result of rapid heparinized saline infusion, which causes a total or partial replacement of blood above the occlusion, not allowing adequate retrograde flow.<sup>6</sup> In each case, we specifically kept the infusion rate very slow and even discontinued the infusion without improvement in the neurological deficit. The deficit occurred only after the cervical (C1–2 vertebral level) ICA balloon was inflated and completely resolved immediately after balloon deflation. This was tested by several inflations with the same outcome. If the symptoms had been related to an embolic event, they would likely not have disappeared spontaneously after balloon deflation.

The most consistent branches of the cavernous portion of the ICA are the meningo-hypophyseal trunk, ICSA, and the capsular arteries of McConnell (Table 1).<sup>2,4,5,8,9</sup> The lack of perfusion of the meningo-hypophyseal trunk and ICSA presumably accounted for the transient dysfunction of the third through sixth cranial nerves. When ICA occlusion was performed above the origin of the ICSA, the symptoms diminished significantly. In both cases, the external carotid artery (ECA) circulation appeared normal on angiography. There are multiple and highly variable connective channels between both ICAs and between the ICA and ECA that usually protect cranial nerve function during BTO. The rich basilar plexus, which is composed of cavernous ICA branches, connects to its contralateral counterpart and meningeal branches of the ECA and to the ophthalmic and vertebral arteries; it has been termed the "rete mirabile."<sup>9</sup> Collateral flow through a patent circle of Willis was sufficient to provide perfusion of the hemisphere during the occlusion test; however, there was probably inadequate retrograde filling of cranial nerve collateral vessels at the level of the meningo-hypophyseal trunk and the ICSA. Whether the patients' neurological symptoms would have persisted with permanent occlusion of the carotid artery is unknown. The potential for ICA collateralization with branches of the ECA and contralateral ICA exists and permanent occlusion could possibly result in enlargement of preexisting connective channels prior to irreversible neurological injury. In Case 2, the patient's tolerance to permanent occlusion after extracranial-intracranial arterial bypass had been performed may have resulted from an increase in ICA retrograde flow, increased perfusion through preexisting connective channels, or both.

### Conclusions

Neurological examination with detailed evaluation of cranial nerve function during BTO is critical for the interpretation of clinical manifestations due to hemodynamic changes. Thorough knowledge of neuroanatomy and blood flow dynamics is essential for accurate interpretation of BTO findings. The current technology for assessment of cerebral blood flow during BTO is inadequate to evaluate blood supply to cranial nerves. We should be aware of the possibility of insufficient connections between the ECA and ICA and between both ICAs prior to high-cervical ICA occlusion.

TABLE 1  
*Anatomy of cavernous ICA branches\**

Cavernous ICA Branches	Secondary Branches	Vascular Territory	Connections
meningo-hypophyseal trunk	inferior hypophyseal artery	posterior lobe of pituitary gland; floor of sella turcica	contralat inferior hypophyseal artery
	tentorial artery	third & fourth cranial nerves; roof of cavernous sinus; tentorium; falx	contralat tentorial artery
	dorsal meningeal artery	sixth cranial nerve	contralat dorsal meningeal artery
ICSA	NA	gasserian ganglion; floor of cavernous sinus	middle meningeal artery
capsular arteries of McConnell	NA	floor of sella turcica	contralat capsular & inferior hypophyseal arteries

\* Data provided in this table obtained from References 2, 4, 5, 8, and 9. Abbreviation: NA = not applicable.

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