Trigeminocardiac Reflex in Embolization of Intracranial Dural Arteriovenous Fistula

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We report a case of immediate reproducible and reflexive response of asystole upon stimulation of Onyx injection during embolization of a tentorial dural arteriovenous fistula in a 53-year-old man. Upon recognition of the reflexive relationship between Onyx injection and increased vagal tone, the patient was given anticholinergic in an effort to block cholinergic hyperactivity. After atropine was given, no further dysrhythmias occurred.

**Discussion**

The phenomenon of bradycardia during Onyx injection has not previously been described. During Onyx injection, bradycardia was reported by the anesthesiologist and resolved upon cessation of injection as the Onyx refluxed to the foramen spinosum. The patient’s vital signs returned to baseline. Three subsequent individual and temporally discrete injections reproduced similar responses of bradycardia. After intravenous administration of atropine, the response was no longer reproducible, and the procedure could be completed. The reflex bradycardia seen during Onyx injection is not likely attributable to other factors. The entire procedure was performed under a standard anesthetic protocol. We did not stimulate the brain to produce the reflex. Research of anesthesia monitoring in patients treated with Onyx embolization for intracranial aneurysms showed no changes in heart rate or blood pressure following DMSO and Onyx injections, nor were any arrhythmias observed. We consider that this response was a TCR, which has been previously described in the literature as a reflexive response of bradycardia, hypotension, and gastric hypermotility seen upon mechanical stimulation in the distribution of the trigeminal nerve. Clinically, TCR has been reported to occur during craniofacial surgery, tumor resection in the cerebellopontine angle and falx cerebri, and trans-
sphenoidal surgery for pituitary adenomas. The literature failed to identify a previously reported case of TCR during embolization of a DAVF. TCR as a physiologic reflex has been clarified. The reflex was reproducible during light manipulation of the pedicle of the middle meningeal artery, so the reflex probably began from the foramen spinosum. We feel that the anatomic studies of Penfield and McNaughton reasonably display the nervous innervation of the dura and a rational pathway from the foramen to the dural middle fossa. They described a mandibular branch of the trigeminal nerve, the nervus spinosus, that follows the middle meningeal artery outside the cranium and through the foramen spinosum to innervate the middle fossa dura. We think that direct compression of the nervus spinosus by the dilated middle meningeal artery due to formation of Onyx plug produces and sends neuronal signals via the Gassarian ganglion to the sensory nucleus of the trigeminal nerve, forming the afferent pathway of the reflex arc. This afferent pathway continues along the short internuncial nerve fibers in the reticular formation to connect with the efferent pathway in the motor nucleus of the vagus nerve and causes bradycardia. When stimulation of the foramen spinosum causes TCR, treatment of hemodynamic instability consists of ceasing procedure and administering anticholinergic drugs. Atropine was shown to effectively extinguish the TCR in our patient. Anticholinergic drugs are not given prophylactically because they can cause refractory arrhythmias.

Conclusion
The present case gives evidence for the first time that TCR may occur during transarterial embolization of DAVF with Onyx, leading to a significant decrease in heart rate under a standard anesthetic protocol. This case confirmed that the reflex was blunted by the anticholinergic effects of atropine. Recognition of TCR allows for early detection and appropriate treatment.

References