Transient Asystole During Surgery for Posterior Fossa Meningioma Caused by Activation of the Trigeminocardiac Reflex—Three Case Reports—

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Abstract

Three patients undergoing surgery for cerebello-pontine angle meningioma suffered transient episodes of asystole. All patients exhibited return to the previous heart rate with cessation of surgical manipulations and administration of anticholinergic agents. These reactions were apparently elicited by activation of the trigeminocardiac reflex (TCR) by direct stimulation of the trigeminal nerve or branches in the dura mater or cerebellar tentorium. Remifentanil was used in all three cases as an anesthetic agent, so may be a cause of the TCR. The possibility of activation of the TCR should be considered during surgical manipulation around the trigeminal nerve or the distribution of the trigeminal nerve branches. Transient bradycardia, hypotension, or asystole can occur regardless of whether there is pressure on the brainstem during posterior fossa meningioma surgery.

Key words: trigeminocardiac reflex, posterior fossa meningioma, asystole, remifentanil, bradycardia

Introduction

Cauterization of the cerebellar tentorium or dura mater of the skull base and detachment of tumor from the cranial nerves are routine manipulations in surgery for posterior fossa meningioma. Transient bradycardia, hypotension, and asystole are rare complications of these manipulations, probably arising from excitation of the trigeminocardiac reflex (TCR) by direct stimulation of the trigeminal nerve or branches in the dura mater or cerebellar tentorium.

The TCR results from stimulation of the trigeminal nerve or sensory branches distributed in the cerebellar tentorium, falc, or skull base dura mater which is conducted to the vagal motor nucleus, which delivers inhibitory stimulation to the heart and systemic vascular system via the sensory nucleus of the trigeminal nerve in the brainstem via various neural circuits. However, the detailed pathways of conduction between the trigeminal and vagal nervous systems are unclear. Numerous cases of TCR have been caused by intracranial manipulation during cerebello-pontine or falk meningioma surgery. In addition, bradycardia, hypotension, and asystole have been caused by TCR during craniofacial surgery, endovascular embolization of dural arteriovenous fistulas in the cerebellar tentorium, and transsphenoidal surgery for pituitary lesions. In almost all cases, cessation of surgical manipulations or administration of anticholinergic agents normalized the heart rate, and prevented recurrence of TCR without postoperative complications.

We describe three cases of transient asystole which occurred during posterior fossa tumor resection.

Case Reports

Case 1: A 36-year-old woman presented with sudden onset of headache at 26 weeks and 2 days of pregnancy. Computed tomography (CT) and magnetic resonance (MR) imaging revealed a 5-cm diameter mass in the left cerebello-pontine angle with intratumoral hemorrhage (Fig. 1A, B). After admission, she gradually developed disturbance of consciousness, and CT demonstrated slight hydrocephalus. Her baby was delivered by cesarean section, with priority given to the baby’s health rather than the mother.

Ten days later, suboccipital decompressive craniectomy was performed to avoid cerebral herniation. After the surgery, her hydrocephalus and level of consciousness improved. One month later, during recovery from the birth, endovascular embolization of the tumor feeding artery was performed prior to tumor resection. No major problems occurred during or after embolization. Tumor resection was performed under general anesthesia maintained with propofol, vecuronium, and remifentanil, at 4 days after embolization. Surgery was begun in the right
park bench position. Temporal craniotomy was added to the previous suboccipital craniectomy, a portion of the petrous bone was excised, the dura mater and cerebellar tentorium were cut open, and then tumor resection was begun through a presigmoid approach.

A short time after initiation of tumor resection, transient periods of asystole lasting 3 to 5 seconds occurred without warning signs during cauterization of the dura mater around the cerebellar tentorium and petrous bone, to which the tumor was attached. After cessation of surgical manipulation, her heart rate returned to that present previously, and then surgical manipulation was resumed. After most of the tumor had been resected, during cauterization of the cerebellar tentorium, a further period of asystole lasting approximately 15 seconds abruptly occurred (Fig. 1C, D). All surgical manipulations were immediately discontinued, bolus infusion of atropine sulfate 0.5 mg was administered, and her heart rate then returned to that previously present. Although surgical manipulations were resumed, no subsequent asystoles occurred.

The histological diagnosis of the resected tumor was benign meningioma. No perioperative complications occurred due to intraoperative asystole. No arrhythmia was noted except during surgery.

**Case 2**: A 52-year-old woman presented with headache, dysarthria, and wobbling. MR imaging revealed a 3-cm diameter mass in the left cerebello-pontine angle (Fig. 2A, B). Surgery was performed in the right park bench position under general anesthesia maintained with propofol, vecuronium, and remifentanil. Tumor resection was begun through a retrosigmoid approach. The tumor was detached from its origin in the cerebellar tentorium, and the tentorium was cauterized. During this manipulation, heart rate transiently decreased from 60 beats to 40 beats per minute, but then returned to that previously present.
After most of the tumor had been resected, during cauterization of the cerebellar tentorium around the petrosal vein, an approximately 5-second period of asystole abruptly occurred (Fig. 2C, D). All surgical manipulations were immediately discontinued, and heart rate then returned to that present previously without administration of atropine sulfate. Although surgical manipulations were resumed, no asystole subsequently occurred.

The histological diagnosis of the resected tumor was benign meningioma. No perioperative complications were caused by the intraoperative asystole. No arrhythmia was noted except during surgery.

Case 3: A 48-year-old man presented with tinnitus. MR imaging revealed a 2.7-cm diameter mass reaching Meckel’s cave and extending to the right cerebellopontine angle (Fig. 3A, B). Surgery was performed in the left park bench position under general anesthesia maintained with propofol, vecuronium, and remifentanil. Tumor resection was begun through a suboccipital retrosigmoid approach. The tumor was adhered tightly to the trigeminal nerve. During traction on the tumor for dissection from the trigeminal nerve, a 7-second period of asystole abruptly occurred (Fig. 3C). All surgical manipulations were immediately discontinued, and the heart rate then returned to that present previously. Atropine sulfate 0.5 mg was infused, and surgical manipulations were continued. After most of the tumor had been resected, transient bradycardia occurred during manipulation around the trigeminal nerve, but with cessation of surgical manipulations the heart rate returned to that previously present.

The histological diagnosis of the resected tumor was benign meningioma. No perioperative complications were caused by the intraoperative asystole. No arrhythmia was noted except during surgery.

Discussion

Our three patients presented with large meningiomas compressing the brainstem, but asystole occurred during cauterization of the cerebellar tentorium in Cases 1 and 2 and during traction of the trigeminal nerve in Case 3. These reactions appeared to involve the activation of the TCR via manipulation of a dural branch of the trigeminal nerve in Cases 1 and 2, and to direct manipulation of the trigeminal nerve trunk in Case 3. In Case 1, the asystole in the early phase of surgery was almost certainly caused by compression of the brainstem, but little compression of the pons or medulla oblongata was observed. However, asystole and bradycardia also occurred after removal of most of the tumor, which was unlikely to be caused by compression of the brainstem. Moreover, these reactions all occurred during cerebellar tentorium cauterization and manipulation around the trigeminal nerve, so appeared to involve the TCR.

We reviewed 14 patients who underwent cerebello-pontine angle meningioma surgery between September 2001 and December 2007, and found that intraoperative TCR was observed in 1 of the 14 patients. Transient bradycardia occurred during manipulation around the internal auditory meatus, although the relationship between the manipulation and TCR was unclear. However, the previous and the present three cases all involved the use of remifentanil as an anesthetic agent. In the cases without TCR, fentanyl or dinitrogen monoxide was used as an anesthetic agent.

Use of remifentanil became widespread in Japan from April 2007. The half-life of remifentanil in the blood is shorter than that of fentanyl, enabling early recovery from anesthesia. Remifentanil does not usually induce postoperative respiratory depression after lengthy surgery since the cumulative effect is lower, and thus can be used in high doses to obtain stronger anesthetic effects. Remifentanil caused severe bradycardia or asystole during the induction of anesthesia,2,4) particularly when patients had taken both beta blocker and calcium antagonist agents together,2,9) though such drugs were not used in our 3 patients. TCR has also been reported in cases that involved use of fentanyl or dinitrogen monoxide. Therefore, we suggest that remifentanil may more easily induce hypotension or bradycardia than fentanyl or dinitrogen monoxide.

Hypotension, bradycardia, arrhythmia, or asystole associated with manipulation around the brainstem may be caused by compression of the brainstem or stimulation of the vagal nerve nucleus, so the surgeon may consider continued tissue manipulation risky and thus discontinue the surgery. However, it is important to consider whether the reaction originates from the TCR or brainstem compression. Any reactions caused by TCR can be expected to
recover with cessation of all surgical manipulations or additional administration of anticholinergic agent, and so surgical manipulations can be resumed. Bradycardia, hypotension, and asystole induced via the vagus nerve involve adrenergic transmission, so administration of anticholinergic agents cannot prevent TCR completely, but cardiovascular complications tend not to linger. However, hearing preservation in patients undergoing vestibular schwannoma surgery was worse in the group with intraoperative TCR compared to the group without TCR. All neurosurgeons and anesthesiologists should consider the possibility of induction of the TCR during manipulation of the main trunk of the trigeminal nerve, cerebellar tentorium, falx, or intracranial or extracranial courses of the trigeminal nerve.

References

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