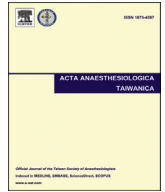




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## Case Report

## Brainstem anesthesia presenting as contralateral third nerve palsy following peribulbar anesthesia for cataract surgery

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## ABSTRACT

Brainstem anesthesia is a serious complication that has been reported to occur more commonly with retrobulbar anesthesia compared to peribulbar anesthesia. We herein report a case of contralateral third nerve palsy following administration of peribulbar anesthesia for cataract surgery. Two hours after the surgery, the patient recovered completely without any residual neurological deficit. The importance of immediate recognition of clinical signs and symptoms of central spread of the local anesthetic and the mechanical factors of the block that could have contributed to this complication are discussed in this report.

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## 1. Introduction

Serious life-threatening complications like brainstem anesthesia have been reported to occur more commonly with retrobulbar anesthesia.<sup>1,2</sup> Although peribulbar block is considered to be safer and easier to teach from a theoretical viewpoint, few cases of brainstem anesthesia following peribulbar anesthesia have also been reported in the literature.<sup>3,4</sup> For a safer orbital regional anesthesia, it has been suggested that a shorter and blunt-tipped needle be used. We herein present a rare case of contralateral third nerve palsy following peribulbar anesthesia with a 25.4-mm (1 in) blunt-tipped steel needle.

## 2. Case report

A 60-year-old nondiabetic, nonhypertensive male was admitted for cataract extraction surgery using the phacoemulsification technique combined with an implantation of intraocular lens in the right eye under peribulbar anesthesia. The axial length of the globe was 25 mm. The patient was monitored with electrocardiogram, peripheral oxygen saturation, and noninvasive blood pressure monitoring, and an intravenous line was secured. The patient was

afebrile and the room temperature was noted to be 19 °C. Approximately 4 mL of 2% lignocaine solution (Xylocaine; AstraZeneca Pharma India Ltd., Bangalore, India) and 4 mL of 0.5% bupivacaine solution (Sensorcaine; AstraZeneca Pharma India Ltd.) along with hyaluronidase 25 IU/mL were used for injection. The first injection was given at the inferotemporal orbital rim, midway between the lateral canthus and the lateral limbus with the eye in primary position. After negative aspiration, 5 mL of the local anesthetic mixture was injected slowly. There was no resistance to the injection, and the feel of the injection appeared as usual. A gentle digital massage was given and the patient was transferred to the operating room.

The planned surgery was completed uneventfully in 15 minutes. The vital signs were stable throughout the procedure. The patient was then shifted to the postoperative observation room. He was conscious, oriented, but unable to open his left eye. On examination there was complete ptosis of the left eye with the eye under the lid being in a depressed and abducted position. Pupillary reaction in the left eye was brisk. Other cranial nerves, motor and sensory functions were found to be normal. A diagnosis of pupil-sparing complete third nerve palsy was made and the vitals were checked every 15 minutes. Two hours after the surgery, the patient recovered completely without any residual defect of nerve palsy. The patient was subsequently discharged. A week later, he underwent cataract surgery, in the other eye, under peribulbar anesthesia uneventfully.

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### 3. Discussion

Brainstem anesthesia occurs when the injected anesthetic gains entry into the subarachnoid space due to direct entry or due to spread of the local anesthetic drug into the central nervous system (CNS). The clinical picture of brainstem anesthesia varies from mild confusion, aphasia, marked shivering, convulsant behavior, bilateral brainstem nerve palsies, amaurosis of the contralateral eye, as well as hemiplegia, paraplegia, or quadriplegia with or without loss of consciousness, changes in blood pressure to apnea, or change in respiratory pattern.<sup>5,6</sup> Although most of these cases have been reported with retrobulbar anesthesia, only a few cases of brainstem anesthesia following peribulbar anesthesia have also been reported.<sup>3,4</sup>

The proposed mechanism for the occurrence of brainstem anesthesia is that the cerebral dura mater provides a tubular sheath for the optic nerve as it passes through the optic foramen. This sheath fuses with the epineurium of the optic nerve and is continuous with the sclera, providing a potential conduit for local anesthetic to pass subdurally into the brain. Central spread occurs if the needle tip has perforated the optic nerve sheath and if injection is made.<sup>7</sup> Another postulated mechanism is the accidental penetration of the optic nerve sheath and the injection of the anesthetic solution into the subdural or subarachnoid space, which travels through the ipsilateral optic nerve, to the optic chiasm, the contralateral optic nerve, and finally to the upper brainstem.<sup>8</sup> It has been reported that intra-arterial injection of the anesthetic agent can also lead to seizures and brainstem anesthesia.<sup>9</sup>

The incidence of the CNS complication with a 38-mm retrobulbar needle is between 0.2% and 0.3%.<sup>7</sup> In one series, there was only one case of CNS spread in 6800 (0.015%) extraconal (true peribulbar) blocks. The length of the needle was 38 mm, only a maximum of 25 mm was advanced within the orbit; leaving at least 13 mm outside.<sup>10</sup> The onset of these symptoms after retrobulbar injection was variable and ranged from 2 minutes to 40 minutes after injection.<sup>11</sup> The effect on the CNS depends on the amount of local anesthetic injected, the depth of needle insertion, force, concentration, and the area to which it spreads into. The possible differential diagnosis that could be considered in such a setting would include any etiology causing isolated third nerve palsy, the most common being microvascular ischemia. However, in cases such as ours, the timing of the onset of palsy, the quick recovery, and lack of any vasculopathic risk factors strongly suggest that it was associated with the peribulbar anesthesia. Furthermore, palsy of the contralateral oculomotor and trochlear nerves with amaurosis is characteristic of CNS spread of the anesthetic agent.<sup>7</sup> Recent

literatures suggest that brain magnetic resonance imaging (MRI) and laboratory workup have a role in the initial evaluation of elderly patients with isolated acute ocular motor nerve palsies regardless of whether vascular risk factors are present, especially in those over 50 years of age.<sup>12,13</sup> Our patient subsequently was examined by neuro-ophthalmologists (R.A.G. and A.G.N.) and an internist and had an MRI of the brain later, which was normal. Therefore, although managing the acute incident is important, it is equally important to rule out other rare causes of isolated cranial mononeuropathy.

From an anesthesiologist's viewpoint, in addition to cardiopulmonary support such as bag and mask ventilation, noninvasive blood pressure monitoring, maintenance of heart rate, and saturation levels, it must be borne in mind that the need to provide cardiopulmonary resuscitation may arise due to systemic complications.<sup>9</sup> The adverse events such as the one discussed here are rare and must be immediately identified so that they can be appropriately managed.

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