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REVIEW

Brainstem anaesthesia revisited: Mechanism, presentation and management

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SUMMARY

Brainstem anaesthesia is the temporary loss of functions of the brainstem. Although rare, it is a life threatening complication with varied presentations following traditional retrobulbar block. This complication had been reported and continues to be reported following other relatively safer orbital blocks. A systematic search of Medline, EMBASE, and Cochrane databases with the subject headings "brainstem", "anaesthesia", "regional anaesthesia", "orbital block", "eye blocks" and "local anaesthesia" was performed. There were several case reports of brainstem anaesthesia and none of the orbital regional blocks were considered absolutely safe. Most published cases reported in the previous decades were usually associated with retrobulbar block and these continued to be reported despite the recommendations. This review focuses on brainstem anaesthesia incidence, mechanism, diagnosis, immediate management and preventative measures.

1. Introduction

The use of local anaesthesia is common worldwide for routine ophthalmic surgical procedures including cataract and glaucoma. The technique of local anaesthesia used varies around the world and ranges from topical anaesthesia to akinetic blocks such as retrobulbar, peribulbar and sub-tenon's blocks.[1–5] Retrobulbar block was first described by Knapp in 1884, and later described as a classical or traditional retrobulbar block advocated by Atkinson.[6] Atkinson used a 38 mm needle and introduced it at the junction of medial 2/3rd and lateral 1/3rd of the inferior orbital margin whilst the eye was rotated upward and inward. The needle was aimed towards the apex of the eye. A small volume of a local anaesthetic agent was injected posteriorly into the apex. This block was often combined with VII cranial nerve block (facial nerve block). The block provided excellent akinesia and anaesthesia by blocking the transmission in cranial nerves III, IV, VI and the ciliary nerves. Retrobulbar block has been associated with several cases of brainstem anaesthesia (temporary loss of functions of the brainstem following orbital block).[7–21] Traditional or classical retrobulbar block has undergone several modifications based on research and peribulbar block was introduced[22–24] as a relatively safer alternative where the needle is advanced along the floor of the orbit, keeping it tangential to the globe and not directed upward and inward at the equator. Peribulbar blocks utilise higher volumes of a local anaesthetic agent. Descriptions of both retrobulbar and peribulbar blocks in published literature vary and Thind and Rubin[25] have highlighted in an editorial that a wide range of orbital local injections are in use, some of which may be described as retrobulbar by one clinician and peribulbar by another. There have been several cases of brainstem anaesthesia following peribulbar block.[26–28] Peribulbar block has undergone several modifications as well. The modern needle-based blocks utilise shorter needles and a higher volume of a local anaesthetic agent in the more anterior part of the apex whilst the eye is kept in the primary gaze position.[29,30] Sub-tenon's block was introduced as a relatively safer technique to needle blocks in the early 1990s.[32,33] After obtaining surface anaesthesia, the conjunctiva and tenon's capsule are gripped with a non-toothed forceps 5–10 mm away from the limbus, usually in the inferonasal quadrant while the patient is asked to look upward and outward. A small incision is made through these layers with Westcott scissors to expose the white sclera. A subtenon cannula (2.54 cm blunt metal cannula or similar) is gently inserted along the curvature of the globe.[34] Although sub-tenon's block is considered much safer than retrobulbar and peribulbar blocks, cases of brainstem anaesthesia[35] including death[36] have been reported.

A systematic search in the Medline, EMBASE, and Cochrane databases with the subject headings "brainstem", "anaesthesia", "regional anaesthesia", "orbital block", "eye blocks" and "local anaesthesia" was performed. Based on the research, this article

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focuses on recent understanding in the aetiology, mechanism of action, clinical presentation, diagnosis and management of brainstem anaesthesia following orbital blocks.

1.1. Anatomy and physiology of brainstem

The brainstem is the dorsal part of the brain that performs multiple important functions. Major subdivisions from rostral to caudal are the midbrain, pons and medulla.[37,38] Pons houses the reticular activating system and the medulla houses the cardiac and respiratory centres.

The brainstem is a conduit for multiple ascending and descending pathways and contains the most cranial nerve nuclei. The central core of the brainstem, formed by the reticular formation participates in a plethora of functions such as control of movement, modulation of pain, autonomic reflexes, arousal, and consciousness. Other key integrative functions include cardiovascular control, respiratory control, and pain sensitivity control. The brainstem also produces monoamine (norepinephrine, dopamine, serotonin) neurotransmitters that affect behaviour, cognition and mood. Vascular supply is from the posterior cerebral artery and vertebral basilar system. Table 1 includes the localising features of brainstem lesion by combining cranial nerve related symptoms (which provide rostral–caudal localisation) and long tract-related symptoms.[38]

1.2. Incidence

Brainstem anaesthesia following retrobulbar block was approximated to occur in 1 in 350–500 patients[28] with central nervous system (CNS) involvement in 0.27%[12] and apnoea in 0.79% in one study.[39] The true incidence of intrasheath injection during retrobulbar block was estimated to be as high as 3 in 150 patients (2%) who underwent a pressure orbitograph and had contrast material in the subdural space, with some reported temporary amaurosis.[40] The estimated incidence of brainstem anaesthesia following peribulbar block is 0.020%[3] and 0.023% after sub-tenon's block.[35]

1.3. Mechanism of brainstem anaesthesia

The exact mechanism of brainstem anaesthesia is unclear. Most of the proposed mechanisms are speculative and mostly reported following traditional or classic retrobulbar block especially using the 38 mm needle.

It has been proposed that the dural sheath of the optic nerve could be punctured and the local anaesthetic injected into the subdural space during retrobulbar injection.[21] The eye is embryologically a part of the brain with an extension of the meninges forming a part of the optic nerve.[41] As the optic sheath is continuous with the subdural and subarachnoid space surrounding the brainstem and cranial nerves, this provides a conduit for accumulation around the brainstem. A study by Brod[42] demonstrated that after retrobulbar block, there was an air bubble in the ipsilateral optic nerve sheath under a computed tomography scan, supporting the intra-nerve sheath injection mechanism.

Another possible mechanism is the local anaesthetic agent spreading directly from the apex of the orbit via submeningeal pathways to the CNS.[16] The spread is often in a cephalad direction and affects specific areas of the CNS.[12] Also, local anaesthetic injection into the cerebrospinal fluid (CSF) space can lead to toxicity at the control centres in the brainstem.[21] Kobet et al.[43] found high levels of bupivacaine and lidocaine from the CSF after retrobulbar block.

The injected local anaesthetic agent could also be systemically absorbed. However, in such cases, the doses of lignocaine and bupivacaine used have to be high enough to cause toxicity i.e. 6 mg/kg and 1.5 mg/kg respectively.[21]

It is possible that a direct intra-arterial injection (ophthalmic artery) of local anaesthetic can reach the internal carotid artery[7] and then into the cerebral circulation.[21] In cases where there is a presence of high brain concentration of local anaesthetic, CNS signs of toxicity would present instantaneously and signs of haemorrhage and aspiration of blood into syringe may or may not be visible.

Studies have been performed on cadavers to ascertain the spread of local anaesthetic agents into the CNS. Drysdale et al.[44] detected the presence of dye in the midbrain surrounding the respiratory centre when radio-opaque dye was injected into the intraorbital subdural space. Another study by Wang et al.[15] found that intrasheath injection of methylene blue dye tracked along the subarachnoid space of the optic nerve sheath to the chiasmatic cistern in the middle cranial fossa. In another unrelated study orbitography was performed in patients who had loss of vision and

Table 1
Signs and symptoms for brainstem lesion localisation (adapted from Gates[38])

Medial injury		Lateral injury	
Ipsilesional symptoms	Contralesional symptoms	Ipsilesional symptoms	Contralesional symptoms
Midbrain			
Cranial nerve III (oculomotor): eye depressed and abducted; ptosis; dilated unreactive pupil	Corticospinal tract: weakness (body)	Oculosympathetic tract: Horner's syndrome	Spinothalamic tracts: loss of pain and temperature (body)
Cranial nerve IV (trochlear): eye unable to depress when turned inward (may cause diplopia)	Medial lemniscus: loss of vibration and proprioception (body)		
Pons			
Cranial nerve VI (abducens): adducted eye	Corticospinal tract: weakness (body)	Oculosympathetic tract: Horner's syndrome	Spinothalamic tracts: loss of pain and temperature (body)
Medial longitudinal fasciculus: internuclear ophthalmoplegia	Medial lemniscus: loss of vibration and proprioception (body)	Spinocerebellar tracts (inferior cerebellar peduncle): ataxia (body)	
		Cranial nerve V (trigeminal-sensory): loss of pain and temperature (face)	
Medulla			
Cranial nerve XII (hypoglossal): flaccid tongue weakness	Corticospinal tract: weakness (body)	Oculosympathetic tract: Horner's syndrome	Spinothalamic tracts: loss of pain and temperature (body)
Medial longitudinal fasciculus: internuclear ophthalmoplegia	Medial lemniscus: loss of vibration and proprioception (body)	Spinocerebellar tracts (inferior cerebellar peduncle: ataxia (body)	Spinothalamic tracts: loss of pain and temperature (body)