Citation

Abstract
Because abducens palsy is a very common problem in our daily practice we decide to review the anatomical variations of abducens nerves that could explain clinical variations under the similar etiologies. Therefore, we report our findings on fixed brain specimens looking for anatomical variations of abducens nerves at the Department of Anatomy from Walter Sisulu University in Mthatha, South Africa.

INTRODUCTION
The abducens nerve contains only somatic efferent fibers that supply the lateral rectus muscle. Several sources have attributed the vulnerability of the abducens nerve to its long intracranial course. Because of the bilateral projections of the nucleus abducens, nuclear palsy of cranial nerve VI usually implies some difficulty in adducting the contralateral eye. Pure cranial nerve VI palsy, therefore, does not suggest nuclear palsy. Neither does it suggest fascicular lesions because the latter rarely involve cranial nerve VI alone but also cranial nerve VII (ipsilateral facial palsy) and/or the mesencephalic root of cranial nerve V (ipsilateral facial hypoesthesia). These associated disorders are known as Foville's and Millard-Gubler syndromes. They are mostly due to demyelinating disease in young persons, arterial disease in elderly peoples. However, other anatomic factors likely contribute to the apparent vulnerability of the abducens nerve to mass lesions and trauma.

From its brainstem exit to its entry into the cavernous sinus, the sixth cranial nerve has a long course in the prepontine cistern where it can be damaged by expansions of nasopharyngeal carcinoma, meningitis, and tumors of the clivus (chordomas or meningiomas). The nerve crosses over the tip of the petrous bone where lesions from infection of the middle ear may cause cranial nerve VI and VII palsies with facial pain as a result of involvement of the trigeminal ganglion (Gradenigo's syndrome).

Several diseases present with isolated sixth nerve palsy in adults but in the cavernous sinus, cranial VI may be involved with cranial nerve III and IV and the ophthalmic branch of cranial nerve V by aneurysm of the carotid siphon, tumors such as meningioma, pituitary apoplexy, or infections, but for some unknown reasons, this nerve is especially vulnerable reason why we will try to identify abnormalities that could explain this situation. An isolated cranial nerve VI palsy may result from cavernous sinus lesions. Cranial nerve VI paralysis is a rare complication of spinal tap and diabetes mellitus and inflammatory systemic disease such as giant-cell arteritis are possible causes.

The causes of actual sixth nerve palsy include aneurysms of the vertebral artery (VA), tumor, head trauma when the examination must look for deafness, otorrhagia of blood or cerebrospinal fluid, and mastoid ecchymosis. Other cause include: diabetes mellitus, arteriosclerosis, multiple sclerosis, meningitis, increased intracranial pressure (one of the most common false localizing signs of increased intracranial pressure, and lumbar puncture.[]), isolated abducens nerve palsy related to dolichoectatic vertebral artery (DVA) compression has been reported[]. There is a report about a patient with lifelong, bilateral horizontal gaze palsies and the anatomical findings of bilateral absence of the abducens nerve as it affected the brainstem, the course of the cranial nerves to the extraocular muscles, and muscle innervations are described and the possible relationship of these findings to Duane's syndrome and Moebius syndrome is discussed.[4]

MATERIAL AND METHOD
One of us (GMR) made all dissections on selected fixed brain specimens at the laboratory of anatomy from Walter Sisulu University. Selection criteria were based on the good preservation of the brain and good quality material for dissection. The main aid was to identify abnormalities of
The Abducens Nerve in Neurology

The abducens nerve in its gross anatomy of the abducens nerve or on its pathways.

**RESULTS AND COMMENTS**

The abducens nerve was consistently approximately one-third the length of the trochlear nerve at all ages that we studied. The endoscopic views revealed the structural and vascular relationships of the abducens nerve in situ. The fibers arise from a nucleus located in the pons close to the midline and beneath the facial colliculus. Axons from the facial nerve loop around the abducens nucleus, (Figure 1) creating a slight bulge (the facial colliculus) that is visible on the dorsal surface of the brain stem, on the floor of the fourth ventricle. Motor axons leaving the abducens nucleus run downward and forward through the pons, lateral to the corticospinal tract and emerge in the sulcus between the lower border of the pons and the upper part of the pyramid of the medulla oblongata, into the subarachnoid space.

Figure 2

This nerve had the longest extradural course in our disserted material. Because of this, palsy of this nerve may develop in association with meningitis and subarachnoid hemorrhage.

It runs upward between the pons and the clivus, (cisternal segment of the abducens nerve) (Figure 3) and then pierces the dura matter to run between the dura and the skull into Dorello's canal (the bow-shaped canal through which courses the abducens nerve before reaching the cavernous sinus. (petroclival part of the abducens nerve). The nerve next traverses the cavernous sinus, on the lateral side of the internal carotid artery. Figure 3 and 4. In the cavernous sinus, the oculomotor, trochlear, ophthalmic branch of the trigeminal nerves is place in the lateral wall.

As these nerves passes forward to the superior orbital fissure their position change.

In the superior orbital fissure the nerve enter the orbit through the common tendinous ring together with the oculomotor nerve.

In the orbit abducens nerve is situated medial to the lateral rectus muscle. Figure 5

Nerve injury:

Injury to the VI cranial nerve results in paralysis of the ipsilateral lateral rectus muscle and diplopia on attempted horizontal gaze toward the side of the paralyzed muscle. The affected eye will tend to deviated inward because of the unopposed action of the medial rectus muscle.

The central anatomy of the sixth nerve through the pons predicts that infarcts affecting the dorsal pons at the level of the abducens nucleus can also affect the facial nerve, producing an ipsilateral facial palsy together with lateral rectus palsy. Infarcts involving the ventral pons can affect the sixth nerve and the corticospinal tract simultaneously, producing a lateral rectus palsy associated with a contralateral hemiparesis.

In the peripheral course, the nerve could be affected by:

1. Sudden caudal displacement of the brainstem that causes avulsion of the nerve.
2. Sphenoid fracture or fistula in the path through the cavernous sinus.
3. Tumors, aneurysms, or fractures – anything that directly compresses or stretches the nerve.
4. Demyelization, infections (e.g. meningitis) and various neuropathies. One of the most common overall causes of sixth nerve impairment is diabetic neuropathy.

The authors conclude from these findings and the review of medical literature [5,6,7,8,9] that abducens nerve vulnerability results from factors other than its intracranial length.
Figure 1: Anterior view of the brainstem. Adducent nerve nucleus located in the pons, midlevel in the brainstem, close to the midline. The fibers from the motor nucleus of the facial nerve made a genu around the abducent nucleus.

Figure 2: Base of the brain. Exit of the abducent nerve between the pons and medulla.
Figure 3
Figure 3: Superior view of the base of the skull. Abducent nerve located between pons and the clivus in the way to the cavernous sinus.

Figure 4
Figure 4: Lateral view of the base of the skull from the inside showing the path of the abducent nerve in the cavernous sinus and its relation with the internal carotid artery.

Figure 5
Figure 5: Base of the skull from above, showing the abducent nerve from the exit in the pontomedullary junction, subarachnoid space, and cavernous sinus and in the orbit.

References
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