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# Case Reports

## PARTIAL SIXTH NERVE PALSY RESULTING FROM SPINAL ANAESTHESIA — A CASE STUDY

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**Abstract**

*Sixth Nerve palsies have been reported following spinal anaesthesia these are rare and often incomplete. A 36 year old woman presented with a severe headache and diplopia after delivery of her second child for which she underwent two attempted spinal anaesthesia. A sixth nerve palsy had developed ten days after the delivery and resolved by the fourteenth week.*
*Mechanical theories present the most plausible explanations for the cause of the sixth nerve palsy. However, none of the theories adequately explain why the palsy is more likely to occur unilaterally than bilaterally.*
**Key words:** *Abducens nerve, epidural anaesthesia, lumbar puncture, esotropia.*

### INTRODUCTION

This paper describes a case history of the rare association between spinal anaesthetics and sixth nerve palsy. Sixth nerve palsies have been reported[1-5] following spinal anaesthesia these are rare and often incomplete.[5] Patients usually recover within a matter of weeks.[1-5]

Before discussing this case history, it is useful to review the course of the sixth nerve in order to understand the theories that may explain the cause of this palsy and its association with spinal anaesthesia.

The abducens (sixth) nerves rise from the lower border of the pons and the lateral part of the pyramids. They are about ten millimetres apart with the basilar artery lying between them. The two nerves are then crossed by the anterior inferior cerebellar arteries (Figure 1). The nerves travel superiorly, ventrally and laterally in the posterior cranial fossa for approximately fifteen millimetres before piercing the dura. From here they run superiorly along the back of the petrous temporal bone over its apex (Figure 2) and forward through the cavernous sinuses into each of the orbits via the superior orbital fissure to the lateral rectus.[6]

### CASE HISTORY

A 36 year old woman during delivery of her second child underwent two failed attempts of dural taps to administer epidural anaesthetic. This procedure was abandoned when the anaesthetist observed leakage of cerebral spinal fluid. Lack of progress in the labour lead to a general anaesthetic being given for a caesarian section.

After delivery, the patient developed severe generalised headaches, neck pain and photophobia. Ten days post partum she developed horizontal diplopia, initially intermittent, and then constant for distant fixation and on left gaze.

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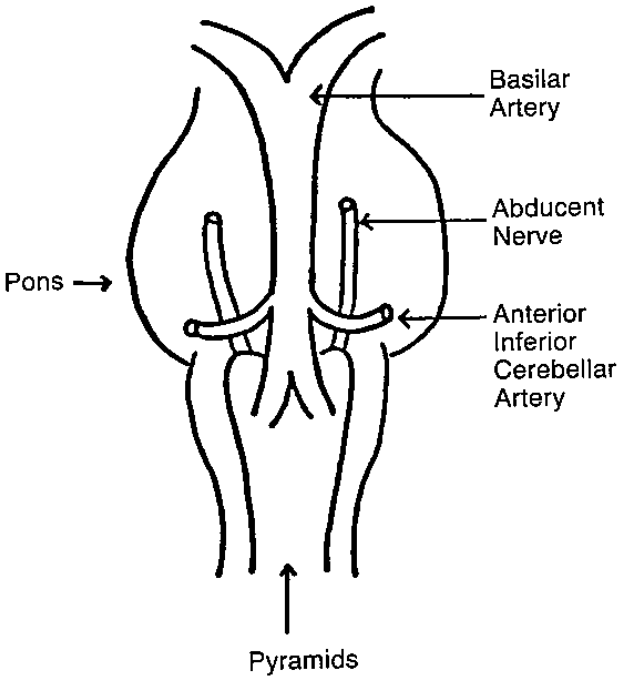


*Figure 1:* Schematic diagram illustrating the relationship between the abducens nerve, basilar artery and the anterior inferior cerebellar artery — ventral view.

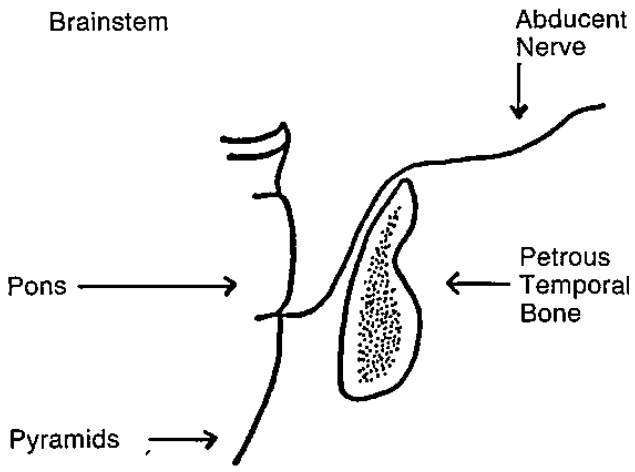


*Figure 2:* Schematic diagram illustrating the relationship between the petrous temporal bone and the abducens nerve — lateral view.

## METHODS

The following are details of ocular examinations conducted over fourteen weeks and demonstrate the rate of recovery (Figure 3).

### *Two Weeks Post Partum*

The patient presented with a severe constant headache which was relieved by lying down.

General health was good, and in particular there was no diabetes or other conditions to account for the palsy.

Visual acuity without glasses was 6/12 right eye and 6/9 left eye. Visual acuity with pin hole was 6/9 right and left eyes. Pupils were equal and reactive. Cover test distance demonstrated a large left alternating esotropia 30$^{\Delta}$ base out. Cover test near indicated a small esophoria 4$^{\Delta}$ base out. Extraocular movements indicated under action of the left lateral rectus. Examination showed that the Fundus was normal.

### *Three Weeks Post Partum*

The headache had improved although there was no change in the patient's strabismus. At this stage the patient was referred to a neurologist.

Full neurological examination, including a Computed Tomography Scan (C.T. scan), revealed no abnormal neurological signs apart from a sixth nerve palsy. There was no neurological evidence to explain the presence of a sixth nerve palsy (e.g. tumour, lesion, etc.).

The neurologist's report also noted that the patient had experienced some initial neck pain and photophobia.

### *Five Weeks Post Partum*

The patient's headache, neck pain and photophobia resolved. The esotropia showed improvement.

Prism cover test distance demonstrated a large left alternating esotropia 20$^{\Delta}$ base out. The prism cover test near remained unchanged with a small esophoria 4$^{\Delta}$ base out.

### *Eight Weeks Post Partum*

Diplopia was not evident in the primary position. However, there was still some diplopia in left gaze.

Prism cover test distance disclosed a small esophoria 6$^{\Delta}$ base out. Prism cover test near was stable. Extra-ocular movements revealed slight under action of the left lateral rectus.

### *Fourteen Weeks Post Partum*

The patient was now fully recovered.

Uncorrected visual acuity was 6/6 right and left eyes. Cover test distance was noted to

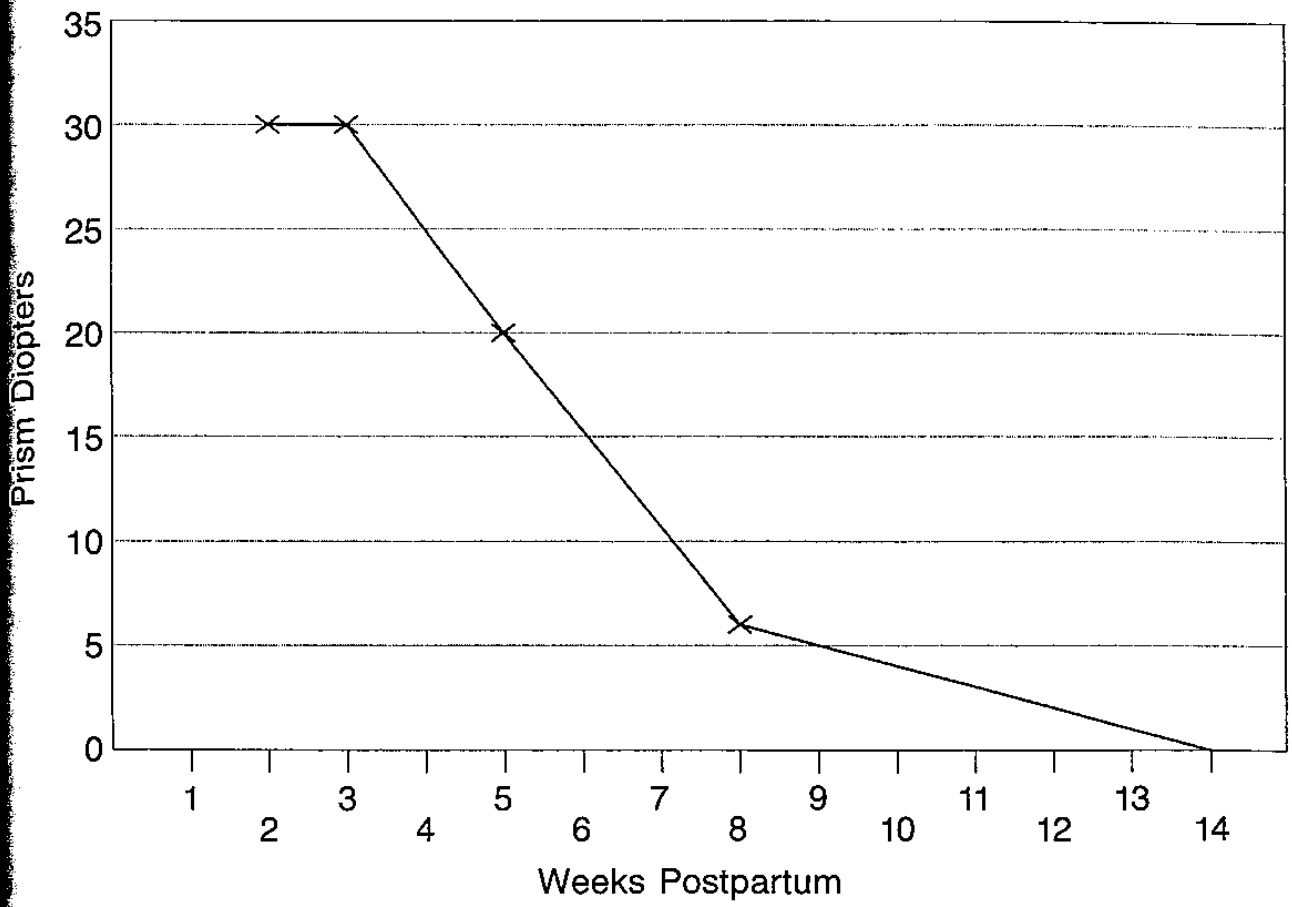


*Figure 3:* Recovery rate of esotropia.

be orthophoric. Cover test near remained unchanged. Extraocular movements were normal (there was no diplopia evident on left gaze).

## MANAGEMENT

Management of this condition is conservative. On the second visit, however, a 25$^{\triangle}$ base out fresnel prism was fitted to a pair of sunglasses to relieve the diplopia at distant fixation. The prism proved of little assistance to the patient due to induced blurred vision by the prism, diplopia on left gaze, and incomitance of the deviation.

Lee and Atkinson[5] have suggested occluding the lateral third of the patient's glasses to avoid diplopia in lateral gaze as well as the wearing of sunglasses to relieve the photophobia.

## DISCUSSION

Spinal anaesthetics have given rise to palsies of all the cranial nerves except the olfactory (first), glossopharyngeal (ninth) and vagus (tenth) cranial nerves.[1,2,4,5] It appears that the sixth nerve is the most commonly affected, either unilaterally or bilaterally. Although it is rare, occurring approximately one in every two hundred to three hundred cases, unilaterally more than bilaterally.[1,4,5]

Symptoms which appear to precede this type of sixth nerve palsy are headaches, neck pains, dizziness, nausea and photophobia.[1] Diplopia occurs from the second day to the third week after the anaesthetic, most commonly occurring on or about the seventh day. Full recovery occurs anywhere from the fourth week to the fourth month,[1,2,4,5] but may be delayed for up to twelve months.[1]

Various theories have been postulated as to the cause of the sixth nerve palsy. These are: toxicity theory;[1,4,5] inflammatory theory;[1,4,5] and mechanical theory.[2,4,5]

*Toxicity Theory*

The toxicity theory suggests that a specific action of the drugs used in anaesthesia may lead to a degeneration of the cells[1] along the exposed course of the sixth nerve which is why it is most commonly affected.[1,4]

The alternative view is that a general cerebral toxaemia may lead to a breakdown in binocular coordination thus causing diplopia.[4,5]

*Inflammatory/Infective Theory*

The inflammatory theory suggests that the procedure of spinal anaesthesia leads to low grade meningitis[1,5] or aseptic meningitis.[1]

The toxicity and inflammatory theories may be connected because the anaesthetic solution may alter the chemical balance in the cerebral spinal fluid (CSF) and lead to irritation of the meninges.[4]

*Mechanical Theory*

There appear to be two views regarding the possible mechanical causes that lead to a sixth nerve palsy.

(a) Spinal anaesthesia alters the hydrodynamics of the CSF pressure leading to a displacement of the cerebellum. This causes stretching of the anterior inferior cerebellar artery which is attached to the basilar artery — thus possibly compressing the nerve[5] (Figure 1).

(b) The spinal anaesthesia causes a downward displacement of the brain due to change in the CSF pressure. This displacement may lead to a stretching of the nerve over the petrous temporal bone[2,4] (Figure 2).

The mechanical theories present the most plausible explanations for the cause of the sixth nerve palsy. This is because decompression of the subarachnoid space is likely to be common to all such spinal procedures,[2] as may be a change in the hydrodynamics of the CSF.[5]

## CONCLUSION

There was no anatomical evidence in the C.T. scan of this patient to offer any additional evidence to explain why the palsy was unilateral.

In addition, none of the theories adequately explain why the palsy is more likely to occur unilaterally than bilaterally.

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