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## Paresis of the Fifth Cranial Nerve Following Spinal Anesthesia

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Cranial-nerve paralysis is a recognized but uncommon complication following spinal anesthesia. Since cases involving the abducens and oculomotor nerves were first described by Venus,<sup>1</sup> in 1907, involvement of every cranial nerve except the olfactory, glossopharyngeal and vagus has been reported.<sup>2,3</sup> Paralysis of the abducens (VI) nerve has been reported most frequently. Oculomotor (III), trochlear (IV), facial (VII), and auditory (VIII) nerves have also been involved but much less frequently. Involvement of the trigeminal nerve (V) is extremely rare. We have been able to find only two reported cases.<sup>4,5</sup> We present a further case, in which paresis of the fifth cranial nerve followed spinal anesthesia.

### REPORT OF A CASE

A 28-year-old black woman was admitted to the delivery unit at 3 AM. Pregnancy had been uneventful apart from a slightly elevated blood pressure (150-160/90 torr). There was no history of prior disposition to headaches, neural paresis, or recent dental work. The patient received hydroxyzine, 50 mg, im, on admission and meperidine, 25 mg, iv, at 5 AM. Three hours after admission, the cervix was fully dilated. The patient was taken to the delivery room, where a subarachnoid block was performed without difficulty with the patient in the sitting position, and she was given 60 mg 5 per cent lidocaine through a 25-gauge spinal needle. Both anesthesia and low forceps delivery proceeded uneventfully. Except for an occasional elevation of blood pressure, requiring no medical treatment, the next 48 hours were uneventful.

On the third postpartum day the patient complained of headache brought on by raising her head and relieved by lying flat. Treatment consisted of rest in bed, increased oral intake of fluids, and acetaminophen (Tylenol). On the fourth postpartum day the patient complained of dizziness and numbness in the left side of her face and left anterior two thirds of the tongue, and pain in the left neck and shoulder. These symptoms were exacerbated by standing up and relieved by lying flat. The next day numbness persisted even when the patient was lying flat. At this time she denied any headache, visual disturbance, or dizziness. Neurologic consultation revealed no abnormal finding apart from paresthesia and diminished sensation over the affected area. The neck was not stiff, the patient was afebrile, and blood pressure varied between 140/80 and 165/90 torr. The patient was given 1 l 5 per cent dextrose in 0.2 per cent saline solution, iv, over a period of two hours. A tight abdominal binder was applied and bed rest was ordered. By the afternoon of the same day, the symptoms disap-

peared, and the patient was discharged the following day. A month after her discharge from the hospital, she denied any further symptoms.

### DISCUSSION

Cranial-nerve paralysis has been attributed to low cerebrospinal fluid pressure following subarachnoid block.<sup>6,7</sup> Thorsen,<sup>2</sup> reviewed cases of cranial-nerve paralysis following spinal anesthesia and found an incidence of 173 in a series of 68,179 spinal anesthetics (approximately 1:400). In a review of 97 cases of cranial-nerve paralysis following dural puncture, Blatt<sup>8</sup> found 87 cases of abducens, six cases of oculomotor, and four cases of trochlear nerve involvement. The incidence of paralysis of the abducens nerve is variously quoted as 1 in 250<sup>3</sup> to 1 in 1,300.<sup>9</sup> Eighth-nerve dysfunction was reported in one study as occurring in 34 cases following 9,277 spinal anesthetics (1:265).<sup>10</sup> Cranial-nerve paresis is often preceded by postdural-puncture headache, nausea, and stiff neck. The majority of cases occur from the third to the fifteenth postoperative days, and 90 per cent of the patients recover within three months.

The brain is suspended in cerebrospinal fluid (CSF) with additional support from structures such as the cerebral and cerebellar vessels and their tributaries to venous sinuses. From below there is support by the tentorium cerebelli and by the circle of Willis and other large vessels at the base of the brain. Without a full fluid cushion to rest on, the brain tends to sag when the patient is upright, and the resultant traction on pain-sensitive blood vessels and anchoring dura produces headache. Pain arising from the structures above the tentorium cerebelli has been shown to be transmitted mainly by the second and third divisions of the fifth cranial nerve, and the pain is referred to the anterior half of the head. When pain arises from below the surface of the tentorium cerebelli, it is transmitted chiefly by the ninth and tenth cranial nerves and the upper three cervical nerves and is referred to the suboccipital region and the back of the neck. In this particular case, the pain in the neck and shoulder can be explained on this basis.

The abducens nerve is relatively fixed in the cranium and the trochlear nerve is described as being the most slender, with the longest intracranial course.<sup>11</sup> There is a direct anatomic connection between the

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cochlea and the subarachnoid space,<sup>12</sup> which may account for auditory disturbances due to decreased CSF pressure after dural puncture. This understanding of the anatomic pathways in the skull is essential to an appreciation of the frequency with which the sixth, fourth, and eighth cranial nerves are affected by lowered CSF pressure.

The bed of the semilunar ganglion of the fifth nerve is formed partly by the internal carotid artery and the bony structure of the temporal bone. The arachnoid mater is attached to the ganglion and the root bundles are bathed in CSF. The fibers from the mandibular division occupy a posterolateral position while passing from the ganglia through the dural foramen (Meckel's cave) to the pons.<sup>13</sup> Compression of the posterior root as it passes through the dural foramen<sup>14</sup> and its angulation over a tilted petrous ridge have been cited as possible causes of trigeminal neuralgia.<sup>15</sup> The sensory root can be stretched, distorted, or compressed by a variety of vascular malformations and tumors compressing the root between the petrous bone and the pons.<sup>11,16</sup>

After passing through the foramen ovale, the mandibular division of the trigeminal nerve curves sharply, and this anatomic relation may contribute to the frequent involvement of this division by stretching of intracranial contents.<sup>13</sup>

Benign trigeminal sensory neuropathy, due to arachnoiditis or viral infection,<sup>17</sup> with full recovery within three weeks to several months, has been reported. The third division of the fifth cranial nerve is most often involved. Minor brain-stem thrombosis<sup>18</sup> has also been suggested as a cause of neuritis.

Toxic cranial neuropathy, especially paralysis of the trigeminal nerve by trichloroethylene, has been clearly recognized.<sup>19</sup> A transitory lesion of a cranial nerve, especially the trigeminal, has been reported to occur after chronic or acute industrial exposure. In the practice of obstetric anesthesia, trichloroethylene is sometimes used as an inhalational analgesic during labor, followed by spinal anesthesia for the second and third stages of labor. This could have clouded the etiology, but was not used on this occasion.

It is difficult to rule out completely the possibility of minor brain-stem thrombosis, especially in view of the hypertension. Coincident idiopathic benign trigeminal neuropathy would be expected to have had a much longer course.

The timing of its onset, preceded by postural headache, and its coincidence with pain in the left side of the neck and left shoulder, its distinct relation to posture, and its rapid course with complete recovery, suggest that the transient paresis of the third division of the trigeminal nerve was the result of low cerebrospinal fluid pressure following dural puncture.

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