THE PREVENTION OF CRANIAL NERVE PALSIES FOLLOWING SPINAL ANESTHESIA

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INTRODUCTION

The etiology of postpuncture cranial nerve palsies is generally regarded as unknown or uncertain, even by recent authors (1–8). The incidence varies from none in 21,000 cases (9) to 5 per cent (10), with intermediate percentages of 0.25 (10), 0.3 (11), 0.5 (12), 0.9 (13) and 3.2 (14). It would seem, therefore, that a cranial nerve palsy following lumbar puncture is an "act of God." Were this true it would be necessary to agree with Kennedy, Effron and Perry "that spinal anesthesia should be rigidly reserved for those patients unable to accept a local or general anesthetic" (7). On the contrary, however, we, like Thorsen (10), have long regarded the primary cause of the condition as sufficiently established for an effective preventive program. On this premise we confidently use spinal anesthesia for the majority of major and minor surgical and obstetric procedures below the diaphragm. This position is more significant in the light of the following reasons for our special interest in postspinal neurologic complications: (1) our practice is composed largely of private patients who remember their anesthesiologist, at least, by name, and (2) we have never had any neurologic complication, other than postpuncture headache, in over 55,000 spinal anesthesias. Even minor instances of cephalalgia have been sought, investigated and vigorously treated (15, 16).

ETIOLOGY AND PATHOGENESIS

The following characteristics are common to the 12 unpublished cases culled by inquiry in our community and to the vast majority of published reports which contain sufficient detail to be of value (214 cases):

1. A postlumbar puncture headache is evident during the latent period of several days to three weeks that elapses before the appearance of the palsy.

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2. The headache has not been treated by any method now recognized as effective in correcting the reduction in volume of cerebrospinal fluid which is the basic cause of postpuncture headache.

3. The palsy occurs only after the patient is no longer recumbent.

4. Little or no attention has been given to the gauge of the spinal needle and other factors that influence the rate of dural leakage and the extent of hydration of the patient. In the relatively few cases in which more details are available, however, and with our present viewpoint, the cause of severe or prolonged reduction in cerebrospinal fluid volume and the absence of adequate hydration treatment of the postpuncture headache may be clearly detected (1, 11, 17–20, cases 2, 4, 5, 6 cited below).

5. The palsy resolves spontaneously.

These regular findings may be correlated by the one proved abnormality of cerebrospinal hydrodynamics that follows lumbar puncture. In other words, the basic cause of a cranial nerve palsy is the same as that which accounts for the antecedent “spinal headache,” namely, decreased volume of cerebrospinal fluid in the intracranial cavity when the head is elevated (10, 21, 22). The cranial nerve palsy may be related to postpuncture loss of subarachnoid fluid by the following sequence of events: first, time elapses for leakage of sufficient spinal fluid through the lumbar dural puncture to decrease the “water cushion” so that, when the patient elevates his head, gravity makes manifest the inadequate volume of subarachnoid fluid and tends to displace the congested brain caudally; second, the typical “postspinal” headache (which has usually appeared during the earlier part of the latent period when the decrease of intracranial fluid began) is not treated with an effective means of restoring the normal volume of subarachnoid fluid; finally, caudal displacement of the brain is sufficiently severe or prolonged to exert traction or pressure, or both, on a cranial nerve that is anatomically or functionally susceptible to such force, usually the abducens which tautly courses with a sharp bend over the upper border of the petrous temporal bone through Dorello’s canal (23, 24).

The benign course and spontaneous resolution of almost all instances of cranial nerve palsies are similar to the behavior of other peripheral nerve injuries associated with mild to moderate traction or pressure elsewhere in the body, for example, brachial plexus palsy in a hyperabducted arm, and peroneal nerve palsy following pressure on the stirrup. The duration of cranial nerve palsies, like that of simple postpuncture headaches, is self-limited because the low volume of cerebrospinal fluid is spontaneously corrected following the eventual healing of the dural puncture and the resumption of normal hydration.

The nature of this complication prevents experimental confirmation of the mechanism so strongly supported by a study of individual case reports and the favorable experience of those who have minimized the
incidence and severity of postpuncture headache by fine needles and hydration. The nearest approach to a controlled experiment, however, was reported by Vandam (25), published in part by Dripps and Vandam (17) and shown in table 1. These findings confirm the generalization that the incidence of postpuncture headache and cranial nerve palsy is greater when the wider needles are employed.

Additional support of the described pathogenesis is provided by the observation that both the headache and cranial nerve paralytic signs were definitely improved in the 2 patients treated early by measures that raised the intracranial volume of subarachnoid fluid, for example, postural drainage for a coincidental bronchietasis (12), and subarachnoid injection of fluid (17).

PREVENTION

Prevention of postpuncture palsies of cranial nerves begins with the prophylaxis of lumbar puncture headache. It has been adequately shown in other reports that this depends on (a) minimizing the size

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\text{Gauge of Needle} & \text{No. of Cases} & \text{Headache, per cent} & \text{Abducens Palsy, per cent} \\
\hline
16 & 459 & 24 & 1 (5 cases) \\
20 & 2,642 & 16 & 0 \\
22 & 1,207 & 12 & 0 \\
24 & 369 & 6 & 0 \\
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\end{array}
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* After Dripps and Vandam (17).

of the dural puncture by the use of fine gauge needles and performing lumbar puncture with the least trauma, and (b) increasing the rate of formation of cerebrospinal fluid by adequate hydration and replacement of significant blood loss (15, 16, 26–28). Prolonged maintenance of the horizontal position is the best safeguard against the effects of sub-normal cerebrospinal fluid volume but it is impracticable, interferes with early ambulation and is unnecessary if the measures mentioned earlier have been used.

Once a patient shows evidence of decreased cerebrospinal fluid volume, that is, pain in the head, neck or back aggravated by the erect position and relieved by recumbency, the possibility of development of a cranial nerve palsy must be removed by effective therapy. The use of analgesics or any other measure that does not restore normal intracranial volume of cerebrospinal fluid in the upright position is only symptomatic; such palliative treatment may relieve headache but does not correct the basic disorder of the headache and paralysis of cranial nerves. The means of removing most promptly the threat of palsy is recumbency; it immediately relieves traction and pressure by
the brain. In the meantime, however, the normal volume of subarachnoid fluid should be restored by oral or parenteral hydration assisted, if necessary, by antidiuretic agents such as posterior pituitary extract (15).

As a corollary of these principles of prophylaxis the treatment of postpuncture palsy of cranial nerves should include, certainly in its early stage, immediate confinement to bed in the head-down position and the administration of large volumes of fluid by mouth and vein until at least the postpuncture headache has disappeared. By reversing the caudad gravitation of the brain and restoring normal intracranial hydrodynamic and vascular equilibrium the severity and duration of a palsy may be decreased. The usual ophthalmological measures should be instituted to re-establish binocular vision.

**ILLUSTRATIVE CASE REPORTS**

The first 2 cases illustrate the value of hydration in preventing the development of abducens palsy in patients who show premonitory signs or formes frustes, noted by Thorsten in 7 per cent of patients who were carefully questioned after spinal anesthesia and frequently observed a few days before the appearance of double vision (19). In case 1, despite the subnormal volume of cerebrospinal fluid, headache was absent, probably because the patient had not yet elevated her head. She undoubtedly had the intracranial congestion and cerebral swelling that follow a notable loss of cerebrospinal fluid even in the horizontal position (21). These changes were probably sufficient to exert pressure on one or more of the motor nerves to the eyes and thus cause initial functional changes, that is, blurring of vision, photophobia and impairment of binocular focus.

**Case 1.** A 30 year old woman underwent cesarean section with continuous spinal anesthesia administered with a 16 gauge needle and a 3½ French ureteral catheter. She was returned to bed with the intraspinal catheter inadvertently remaining in place. The catheter was not discovered until eighteen hours later. A few hours before the catheter was removed the patient complained of blurred vision, difficulty in reading and photophobia. Headache was absent and she was normal in all other aspects. Three liters of 5 per cent glucose was administered that day and two liters more on the next day. The patient was encouraged to drink ten glasses of fluids during each of the first three days after operation. The eye symptoms disappeared in a few hours and headache or cranial nerve complication did not develop. Convalescence was entirely normal.

**Case 2.** A 26 year primipara was given 0.75 cc. of nupercaine, 1:200, and 0.75 cc. of 10 per cent glucose through a 20 gauge spinal needle for vaginal delivery. The blood loss was moderate; prenatally the hemoglobin was 90 per cent and the erythrocyte count 4,500,000, and on the third postpartum day the hemoglobin was 76 per cent and erythrocyte count 3,600,000. The patient underwent anesthesia and delivery without untoward effect. On the first postpartum day she complained of blurring of vision and inability to read. Because
she was a control patient in a special study (15) prophylactic hydration had
not been ordered. She was allowed out of bed on the second postpartum day.
Headache appeared and blurred vision continued; they were so marked that
she was ordered to drink ten glasses of fluids and given two doses of 1 cc. each
of obstetrical pituitrin during that day. When headache and blurred vision
continued to the third day, an infusion of a liter of 5 per cent glucose and two
doses of obstetrical pituitrin were administered. She continued on an ambula-
tory regimen. The headache improved but blurred vision persisted. When
headache returned and blurred vision remained on the fifth and sixth days, she
was again subjected to increased oral fluid intake, 1 cc. of obstetrical pituitrin
subcutaneously every twelve hours for four doses and an infusion of a liter of
glucose in water. She was completely and permanently symptom-free after
the sixth postpartum day.

The special susceptibility of a cranial nerve is well shown by cases
3 and 4.

Case 3. A woman became deaf for three weeks after spinal anesthesia for
an appendectomy; several years later she had recurrence of deafness for three
weeks after spinal anesthesia for a cholecystectomy (29).

In the next instance we deliberately tested the prophylactic value of hydra-
tion in a patient with known susceptibility to postpuncture cranial nerve palsy.

Case 4. A 25 year old woman with acute appendicitis had a history of
bilateral deafness for three months after administration, elsewhere, of con-
tinuous spinal anesthesia with an 18 gauge needle for a cesarean section two
years earlier. The postpuncture headache in the early days of her convalescence
had not been treated by hydration. For the appendectomy we administered
spinal anesthesia with a 22 gauge needle. An infusion of 2 liters of 5 per cent
glucose in distilled water was given; she drank ten glasses of fluids each day
for the next three days and obstetrical pituitrin, 1 cc., was injected hypo-
dermically twice a day for two days to insure hydration. Convalescence was
uncomplicated.

The following case (29) illustrates, among other factors, the func-
tional oculomotor predisposition stressed by Fairclough (12).

Case 5. A 60 year old man was subjected to a first stage prostatectomy
under spinal anesthesia administered with a 19 gauge needle. After two weeks
of a febrile and dehydrated convalescence, diplopia, dizziness and a left external
rectus palsy developed. Marked nystagmus was then noted on the record
for the first time, but the patient claimed to have had it since birth. The
diplopia and nystagmus were present three days later when he had a second
stage prostatectomy under spinal anesthesia administered with a 22 gauge needle.
Vigorous hydration and blood replacement in the first two postoperative days
were instituted. No further note regarding diplopia appeared in the chart.

Case 6. A 66 year old man was subjected to two spinal anesthetics eight
days apart for two prostatic operations. He had suffered "miner's nystagmus"
at age 46 but not since then. For the past seventeen years he had worn glasses
for distant and near vision. The first operation lasted one hour; spinal anes-
thesia was employed, injecting 120 mg. of procaine hydrochloride with a 22
gauge needle. Convalescence was normal. On the eighth postoperative day he
was returned to the operating room for perineal prostatectomy. The anesthetic was pontocaine, 8 mg., and 0.8 cc. of 10 per cent dextrose solution mixed with 1.4 cc. of spinal fluid and injected with a 22 gauge needle. The patient was placed in an exaggerated lithotomy position. The operation was completed in two hours and twenty-five minutes, during which time he received 500 cc. of blood and 750 cc. of physiologic saline solution. On the first postoperative day the patient noted blurred vision, difficulty in reading and photophobia. On the third day he had a headache relieved by emesis. About the tenth day there was double vision owing to paresis of the right external rectus muscle. The diplopia was barely detectable to the patient on the twenty-seventh day and entirely absent by the sixth week.

Case 6 was reported by Parke (19) and has been recapitulated here because it clearly illustrates the need for detailed analysis of the many factors in pathogenesis to account for the occurrence of this complication. (1) A predisposing ocular abnormality was present. (2) The eye symptoms and abducens palsy soon after a second spinal anesthesia and operation probably had their genesis with the first spinal anesthesia eight days earlier. An additional loss of spinal fluid, which precipitated the palsy, was produced by the second lumbar puncture and an exaggerated lithotomy position for more than two hours. This posture, by raising intra-abdominal pressure, increased the leakage of spinal fluid even through a dural puncture made by a 22 gauge needle. (3) Failure to restore cerebrospinal fluid pressure was caused by the dehydrating effect of two long operations and two postoperative periods in spite of a transfusion of 500 cc. and saline infusion of 750 cc. (4) The symptoms of reduced cerebrospinal fluid volume reported by the patient during the first three days after the second operation were not treated with adequate hydration.

Case 7. A 34 year old white woman received spinal anesthesia for repair of a rectovaginal fistula. A mixture of 0.8 cc. of a 1 per cent solution of pontocaine®, 1.4 cc. of 10 per cent glucose and 0.8 cc. of a 5 per cent solution of ephedrine sulfate was injected through a 22 gauge needle. The operation and the first two postoperative days were without incident. On the third day a postpuncture headache appeared for which she was given an infusion of 1 liter of 5 per cent glucose in water and two doses of posterior pituitary extract. On the fourth day she was greatly relieved, but the headache returned on the fifth day and failed to respond to another infusion and subcutaneous administration of posterior pituitary extract. On the sixth day, therefore, an intravenous injection of 100 mg. of nicotinic acid was given (30). The headache was promptly and permanently relieved. Eleven days after the operation the patient complained of double vision and showed paresis of both external recti, more marked on the right. Vision returned to normal six days later.

Case 7 (29) also developed abducens palsy because of inadequate hydration despite the use of a 22 gauge needle. Relief of postpuncture headache with nicotinic acid probably favored the appearance of the palsy by influencing the clinician to discontinue forced hydration prematurely and by allowing the patient to spend more time in an erect
position. Furthermore, intracranial vasodilatation caused by nicotinic acid might have increased the volume and relative weight of the brain and so aggravated the traction or pressure, or both, on the abducens nerves. The fleeting course of the diplopia (only five days, and one of the briefest in the literature) might be attributed to the hydration even though it was incomplete.

SUMMARY AND CONCLUSIONS

Postpuncture palsies of cranial nerves are primarily caused by the effect of gravity on a brain that has lost its intracranial ‘‘water cushion’’ by leakage of cerebrospinal fluid through a lumbar dural puncture in a patient whose state of hydration is inadequate to replace spinal fluid as rapidly as it is lost. In most patients the low intracranial volume of cerebrospinal fluid leads only to a postpuncture headache. In some, however, a cranial nerve palsy follows because of an anatomical or functional susceptibility of one or more cranial nerves, usually the abducens, to traction or pressure, or both, by the brain that is caudally displaced by severe or prolonged reduction of cerebrospinal fluid volume.

Prophylaxis of postpuncture palsies of cranial nerves depends on the prevention of postpuncture headache by the use of fine gauge needles for lumbar puncture and adequate hydration, and on the treatment of established postpuncture headache by effective hydration to restore the normal volume of subarachnoid fluid.

Seven cases are presented to illustrate specific details in our considerations of etiology and prophylaxis.

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REFERENCES

29. Personal communication to the authors.