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Incidence of Post-lumbar Puncture Headache is Independent of Daily Fluid Intake

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Summary. The clinical practice of advising patients to increase their daily fluid intake after lumbar puncture in order to increase CSF production by re-hydration and thus try to prevent post-lumbar puncture headache (PLPH) has not yet been shown to be effective. In 100 patients the different effects of re-hydration on the incidence of PLPH (1.5 l compared with 3.0 l oral fluid per day over a period of 5 days) were tested prospectively. The incidence of PLPH was independent of the amount of fluid intake in both groups (18, 36%), as was the duration of PLPH. The physiology of CSF production and resorption suggests that PLPH is not a problem of CSF dynamics but a simple mechanical problem of how to close the dural rent and thereby stop the continuous leakage. It is no longer justifiable to advise patients to drink more than usual since there is no physiological or empirical basis for this and it does not seem to have even a placebo effect.

Key words: Post-lumbar puncture headache – Daily fluid intake

Introduction

Post-lumbar puncture headache (PLPH) is still a major obstacle to performing lumbar puncture (LP) as a routine measure on out-patients. It occurs in 35% to 40% of patients following diagnostic LP (with a 20 SWG needle) and is characterized by an occipital or frontal headache with neck stiffness occasionally accompanied by nausea, numbness and blurred vision. It typically starts within a few hours to 1 day after LP, lasts 3 to 7 days and usually confines the patient to bed for this period.

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Pathogenetically an epidural CSF leakage caused by delayed closure of the dural rent leads to a continuous decrease in CSF pressure with subsequent stretching of pain sensitive structures (Kadrie et al. 1976).

It is clear that physical methods that could effectively prevent the occurrence of PLPH would be beneficial. Two prophylactic procedures after LP are still common worldwide: bed-rest and increased oral fluid intake. Bed-rest for 24 h with various postural manoeuvres following LP did not have any significant advantage in prospective randomized studies (Hilton-Jones et al. 1982; Hilton-Jones 1984; Dieterich and Brandt 1985). The benefit of increased oral fluid intake has never been shown conclusively.

The purpose of this prospective study was to test the different effects of increased oral fluid intake (1.5 l compared with 3.0 l per day over 5 days) on the incidence of PLPH in neurological patients.

Methods

Patients. Diagnostic LP was performed on 100 age-matched, randomly allocated, neurological patients (age range from 17 to 80 years; average age in group-I 50 years, in group-II 45 years; 44% female, 56% male).

Daily fluid intake. After LP 50 patients were asked to drink 1.5 l of fluids per day during the following 5 days in addition to their normal clinic diet (group-I); the other 50 patients were asked to drink 3.0 l of fluids per day (group-II). As a control all patients kept a written record of their daily fluid intake.

Posture. All patients were mobilized immediately after LP.

Excluding criteria. All patients were excluded who (a) had undergone a LP in the previous 4 weeks, (b) had increased intracranial pressure, (c) could not drink 3.0 l a day because of

Table 1. Incidence of post-lumbar puncture headache (PLPH) grade I–III (number of patients; %) in relation to daily oral fluid intake

	1.5 l	3.0 l
no headache	32 (64 %)	32 (64 %)
headache (grade) I	5	5
II	4	7
III	9	6
	18 (36 %)	18 (36 %)
Σ	50	50

cardiac disease, (d) had an organic disease with psychiatric symptoms or disturbed consciousness, (e) did not drink the recommended amount. Patients receiving regular analgesic or psychotropic medication, patients who had headache before LP and patients where the needle's introduction was corrected a few times before the proper position was found, were also excluded.

LP technique. Lumbar puncture was performed by the same physician with the patient in a sitting position using a 20 SWG needle. Access was via the L3/4 or L4/5 interspace and 8 to 10 ml of CSF was removed.

Informing the patients. All the patients were informed in the same way without being told about the symptoms of PLPH.

Evaluation. The intensity of the PLPH was classified over 4 grades (0–III) according to the severity and onset of symptoms after getting up:

0 normal, no symptoms
I mild postural headache, occurring after 30 min of mobilisation with normal daily activity (≥ 30 min)
II postural headache, occurring within the first 30 min after mobilisation and severe enough to make the patient lie down several times a day (< 30 min)
III postural headache, occurring within a few seconds to 10 min after mobilisation and severe enough to make the patient spend the rest of the day lying in bed (≤ 10 min).

Symptoms were recorded only if they could be convincingly reproduced by a change of position and typically improved by bed rest. The nature, localisation and duration of the PLPH were noted.

Results

The proportion of symptom-free individuals was 32 (64%) in both groups of patients. The incidence of PLPH was therefore independent of daily fluid intake: 18 (36%) of the 50 patients in both groups complained of PLPH (Fig. 1), the grades of which are indicated in Table 1. The duration of PLPH was also uneffected by the amount of fluid intake.

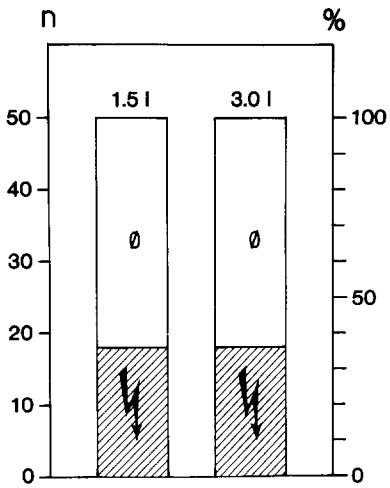

Fig. 1. Incidence of PLPH (number of patients; %) in relation to daily oral fluid intake of either 1.5 l or 3.0 l per day for two groups of 50 patients each. The incidence in each of the groups was 18 patients, i.e. 36%

Discussion

Pathophysiology of PLPH

The main cause of PLPH is continuous epidural CSF leakage through the dural rent due to delayed closure of the dural hole caused by LP (Wolff 1972). This leads to a decrease in CSF pressure resulting in venous dilatation, downward movement of the brain and subsequent stretching of pain-sensitive structures such as dura, nerves and blood vessels. The ninth and tenth cranial nerves and the upper three cervical nerves below the tentorium cerebelli transmit pain from the suboccipital region and the neck; the second and third divisions of the fifth cranial nerve above the tentorium cerebelli transmit frontal pain (Pickering 1948; Wolff 1972).

In the horizontal position the lumbar, cisternal and intracranial CSF pressures are equal (approximately +50 to +150 (+200) mm H_2O). In the upright position they increase to positive values of about +500 mm H_2O at the lumbar level, and decrease to zero at the cisternal level and to negative values of about −300 mm H_2O maximally at the intracranial level. These negative pressures of cisternal and intracranial CSF and positive pressures of lumbar CSF imitate the physical principle of a hanging drop. This also applies to the venous pressure (Kunkle et al. 1943). Headache does not directly correlate with the lumbar or intracranial CSF pressure, but is closely associated with a change in the normal difference of the intra-extravascular pressure of the intracranial veins (Kunkle et al. 1943).

The idea of increased oral fluid intake was to increase CSF pressure, and thereby reduce venous dila-

tation and increase the rate of CSF production in order to compensate for the loss of CSF.

The failure of prone head down tilt posture to facilitate the closure of the dural defect in patients may be due to an erroneous assumption of decreased (hydrostatic) CSF pressure on the dural rent in this position. Because downward tilt of the head leads simultaneously to an increased hydrostatic pressure on intracranial veins which enlarge and occupy space and can thereby maintain pulsating CSF leakage even in the prone head down tilt position (Dieterich and Brandt 1985). There is no longer any justification for requiring patients to stay in bed after LP. But once headache has been established lying down may relieve it, so that bed rest as required is a sensible therapeutic measure.

Why Increasing Hydration Cannot Prevent PLPH?

Increasing the patients' fluid intake as a means of preventing PLPH is based on the assumption that it increases CSF production and thus raises the low intracranial CSF pressure which is the cause of the symptoms. This hypothesis is only correct if increased fluid intake does in fact increase CSF production and if this additional fluid stays in the intradural space and does not leak out via the dural rent. There is reason to believe that neither condition is fulfilled.

From physiological CSF dynamics it is well-known that the rate of CSF resorption is dependent on CSF pressure, while the rate of production is not (Bering and Sato 1963; Cutler et al. 1968; Page et al. 1973; Rubin et al. 1966). Raised CSF pressures increase fluid resorption in the ventriculo-subarachnoid space, whereas low intracranial CSF pressures reduce it (Pappenheimer et al. 1962). This indicates that within physiological limits increased fluid intake has no direct influence on CSF production.

Furthermore, the amount of CSF which leaks through a dural rent depends on the width of the rent as well as on the difference in pressure between the intra- and extradural space, i.e. most CSF is lost in the upright position, a mechanism which cannot be compensated by increased production. An increase in production would only cause increased loss of CSF.

The system is in equilibrium at a pressure of 112 mm H_2O, the theoretical normal CSF pressure, with an equivalent rate of resorption and production of about 0.35 ml/min (Cutler et al. 1968). This means that the loss of 30 ml CSF, for example, due to a persisting dural defect can be replaced in about 85 min after closure of the dural rent.

It therefore seems likely that it is closure of the rent and not CSF loss which is the critical factor in the termination of PLPH. Evidence of this is provided by the striking effect of an epidural autologous blood patch that occludes the defect by a gelatinous tamponade. PLPH is thereby healed in the surprisingly short time of 1 to 2 h in about 90% of the patients (Aboulish et al. 1975).

We conclude that there is neither a physiological nor an empirical argument for a prophylactic increase in fluid intake in patients who have undergone diagnostic LP other than to exploit a possible placebo effect in some individuals.

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