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Mechanisms behind post-spinal headache and brain stem compression following lumbar dural puncture – a physiological approach

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Abstract

Background: The cause of post-spinal headache and its specific characteristics are unknown, and whether lumbar dural puncture (LP) may trigger brain stem compression in patients with brain oedema is still controversial.

Methods: Hydrostatic effects of distal opening of the dural sac towards the atmosphere are described and applied to the normal brain and the brain with disrupted BBB. Analogue analyses from an isolated skeletal muscle enclosed in a rigid shell were applied to the brain in an attempt to simulate and verify haemodynamic effects of distal opening of the spinal canal.

Results: The theoretical considerations and the experimental results are compatible with the hypothesis that hydrostatic effects of distal opening of the fluid-filled spinal canal may obliterate the normal subdural venous collapse after change from horizontal to vertical position, which may be compatible with postural post-spinal headache as occurring close to pain-sensitive meningeal regions. The hydrostatic forces may also initiate transcapillary filtration and aggravate oedema when permeability is increased, which may cause a more narrow situation in the brain stem region, perhaps aggravated by venous stasis and a Cushing reflex-induced increase in blood pressure. An MR picture illustrates how this scenario may separate the subdural space into an upper high- and a lower low-pressure cavity, pressing the brain downwards with sagging of the brain. A life-threatening positive feed back situation for brain stem compression may develop.

Conclusion: The present study strongly suggest that post-spinal headache and brain stem compression and other LP-related effects are predictable following LP, without involving CSF leakage, and can be explained by hydrostatic effects triggered by distal opening of the normally closed dural space to the atmosphere.

Key words: brain edema, brain stem herniation, blood-brain barrier, cushing reflex, lumbar dural puncture, intracranial pressure, meningitis, post spinal headache

Introduction

Opening of the spinal canal via lumbar dural puncture (LP) is used clinically for infusion of local anaesthetics or for CSF sampling, or may be a complication of epidural anaesthesia. It is sometimes associated with postural headache, which appears momentarily when rising from supine to upright/sitting position, and disappears when back in supine position (1-3). The headache may be intolerable (1-4) and can continue for days and even for weeks. In most cases it is of temporary nature, but specific treatment such as application of epidural blood patch is sometimes necessary for its disappearance (4,5). The mechanisms behind post-spinal headache are still unknown.

It has been under discussion for decades, originating from anecdotic cases, whether LP is associated with brain stem compression precipitating a life-threatening situation when performed in patients with increased ICP (6-10). An outcome study from our hospital of severe meningitis showed that in most patients developing brain stem symptoms, these appeared shortly after LP (11). It has been argued that the suspected temporal relation between signs of brain stem compression and LP may be a coincidence, as LP often is performed at a critical time point, and often shortly after start of antibiotic treatment with subsequent release of toxic substances (12). An important reason why no consensus has been reached regarding the use of LP in patients with suspected raised ICP is that no reliable scientific studies on potential adverse effects with LP have been made. As a matter of precaution, some authors recommend that LP should be avoided in patients with clear signs of raised ICP, such as progressive loss of consciousness, agitation and motor anxiety, increasing blood pressure, pupil abnormalities and other focal neurological signs (8, 9, 11). LP is, however, frequently used in clinical practice also in risk patients (13).

It is generally believed that post-spinal headache is an effect of sagging of the brain and traction on pain-sensitive structures due to leakage of CSF (1, 3, 4, 14-17). Leakage of CSF, which must be

relatively slow, however, is not consistent with the fact that post-spinal headache appears momentarily when changing from supine to upright position and disappears immediately after returning back to supine position. No data on CSF leakage in vivo are available, but normal production of CSF in all likelihood is large enough to compensate for the CSF volume leaking through the small dural tear. The fact that brain stem symptoms have not been observed following drainage of CSF from the more distal ventricular system (if not collapsed) is another argument supporting the view that loss of CSF volume per se is not a triggering mechanism behind brain stem compression.

There is a great need to find the truth regarding the risk entailed in LP, since LP is used more or less routinely in clinical practice in spite of its potentially serious adverse effects in states of raised ICP. However, it is very difficult or impossible to perform an ethically acceptable study analysing the hypothesis that LP precipitates brain stem compression, especially since LP often has to be performed in a critical situation, or shortly after start of antibiotic treatment.

The complicated anatomy of the brain with impediment for effective recording and evaluation of important cerebral circulatory variables (18), is one explanation of why we still lack a reliable analysis of cerebral effects of distal opening of the closed dural sac. An attempt to overcome this problem has been made in some previous studies from our laboratory by analysing the general haemodynamic effects on an organ of being enclosed in a rigid shell and the consequences of opening of the closed space to the atmospheric pressure (19, 20, 22). These analyses were performed on a skeletal muscle placed in a fluid-filled and closed plethysmograph with increased tissue pressure.

Based on expected hydrostatic pressure effects of distal opening of the closed and fluid-filled dura sac combined with data from the skeletal muscle experiments, this study is an attempt to reveal the mechanisms behind post-spinal headache, and to show whether LP may precipitate brain stem compression. The analysis gave reliable explanations of post-spinal headache, and supported the view that LP may trigger brain stem symptoms if the blood-brain barrier (BBB) is disrupted.

General physiological and physical principles

The normal brain with brain stem and spinal canal enclosed in the fluid-filled intradural space is illustrated in supine and upright position in Fig. 1 a and b, respectively. As the cavity is a closed and fairly rigid system, the intradural hydrostatic pressure of normally 9-12 mm Hg (19) is of about the same magnitude in all parts of the closed intradural cavity, and will be only moderately lowered when changing from horizontal to vertical position (23). Thus, the tissue pressure of the brain is higher than that of other organs of the body, in which it is close to the atmospheric pressure (24), and the epidural hydrostatic pressure may even be slightly negative (25). This means that the intracranial pressure does not only exceed the atmospheric pressure, but also the venous pressure outside the dura of 0-5 mm Hg in both supine and upright position. The pressure fall between the intra- and extra-dural space will cause a passive venous collapse of the subdural draining veins at a limited length just inside the dura as illustrated schematically in Fig. 2. The existence of a subdural venous collapse was demonstrated already in 1928 (26), but its physiological role or its role in various pathophysiological conditions for brain circulation has not until recently received any scientific attention (20,27,28).

The skeletal muscle model enclosed in a plethysmograph is shown schematically in Fig. 3 a-d, simulating head elevation at a closed and distally open spinal canal. The experiments were performed with an elastic or a rigid draining vein to compare the situation in younger individuals

with that in older individuals with a more rigid vascular system. In these experiments we confirmed the existence of the venous collapse when the tissue pressure exceeded the venous pressure, and showed that the venous collapse acts functionally as a variable passive venous resistance, the strength of which is determined by the difference between the tissue pressure and the venous pressure (20, 22). When using a passive draining vein from the plethysmograph, to simulate a more normal physiological condition, this vein showed a passive venous collapse at vertical levels at which the venous pressure was below the atmospheric pressure at elevation of the plethysmograph (Fig. 3a) (19). By using a more rigid draining vein to simulate the less elastic situation in older individuals, there was an increased collapse inside the plethysmograph at elevation as shown in Fig. 3 b.

Since the degree of subdural venous collapse is determined by the difference between intracranial and extradural pressure, it varies not only with ICP, but also with extradural venous pressure variations. An important consequence of this effect is that venous pressure variations will not be transferred retrogradely to the brain (20). Thus, the normal brain is protected not only from arterial pressure variations via the well-established active arterial autoregulatory mechanism (18, 24), but also from venous pressure variations via a passive venous collapse function.

Effects of lumbar dural puncture under normal conditions

If the dural space after LP remains open to the atmospheric pressure, there will be a pressure fall across the dural opening. The fluid resistance in the dural opening is dependent on the size of the dural opening, which means that the reduction in intradural pressure following LP will vary between patients. In supine position, the intradural pressure most likely will stay above the venous pressure, if a normal thin spinal needle is used (Fig. 1c) and, as CSF communicates freely around

the brain, the brain stem and the spinal cord, the pressure inside the dura will be equal in the whole cavity.

Distal opening of the dura means that ICP must be reduced in upright position due to hydrostatic effects (Fig. 1d), as also confirmed experimentally on cat (29). The magnitude of the reduction depends on the vertical distance between the brain and the dural opening. ICP may even be reduced to negative values. The decrease in ICP will increase transcapillary forces across the cerebral capillaries, but this increase will not induce any filtration resulting in oedema, as cerebral capillaries are impermeable for small solutes in the normal brain (intact BBB) (30-32). However, a decrease in ICP will have other haemodynamic effects in terms of disappearance of the subdural venous collapse if ICP falls below the extradural pressure ($p<pv$ in Fig. 1d).

Simulation of this situation with the skeletal muscle model also showed a reduction in tissue pressure during the elevation, and that the venous collapse disappeared above a certain vertical level at which the tissue pressure and the venous pressure were equal (Fig. 3c). At this borderline level, the veins showed an unstable high-frequency opening and closing behaviour. The disappearance of the venous collapse above this level resulted in a prompt and significant venous blood volume increase (19, 20, 22). The model experiments also showed that the draining passive vein outside the plethysmograph remained collapsed (Fig. 3c). If the collapse was prevented by using a non-elastic draining vein, the hydrostatic effect of the distally open “spinal tube” was partly outbalanced, resulting in a preserved venous collapse of the draining vein as shown in Fig. 3 d. It will be discussed below whether such an effect can explain why post-spinal headache is much less common in older individuals, in whom the vessel elasticity is reduced, than in younger individuals with more elastic veins. The dura is insensitive to pain except in the region of the traversing blood vessels,

which means that the subdural volume effects in patients occur close to the pain-sensitive meningeal regions.

Effects of lumbar dural puncture in patients with severe cerebral infection

If there is a significant oedema and raised ICP, and the BBB is disrupted, another scenario than that described above may develop. Before LP, there will be no limitations for CSF to pass the foramen magnum area if no brain stem compression has occurred, and the intradural pressure will be of about the same size in the whole subdural space both in supine and upright position (Fig. 4 a and b). We assumed an ICP of 25 mm Hg in supine position and a slight reduction in upright position (mainly an effect of reduced intracranial blood volume, and the fact that the dura is not a completely rigid shell). If the dural tear remains open after LP, there will be a reduction in interstitial pressure of the brain in turn increasing cerebral transcapillary pressure, and this increase will be larger after head elevation. As BBB is permeable for small solutes, the increased transcapillary pressure will induce filtration and aggravate the oedema. An increase in oedema, combined with a small increase in intracranial blood volume and a small distal movement of the brain, will narrow the width of the split for CSF to pass around the brain stem, and the intradural space may be separated into a high- and a low-pressure cavity (Fig. 4 c-d). The upper cavity comprises a closed high-pressure space, and the lower cavity a distally open low-pressure space. In supine position, the pressure in the low-pressure cavity will be equal in the whole lower space and below the pressure in the brain (Fig. 4 c), and in upright position the pressure in the low-pressure space will be further reduced in its upper parts in relation to the vertical distance to the dural opening, and it may even be below zero just distal to the brain stem (Fig. 4 d). This means that there is a pressure fall across the foramen magnum area creating a force moving the brain distally aggravating compression of the brain stem, a force which must be larger in upright than in supine position. The initial compression of the brain stem may also induce venous stasis, which will

further increase the oedema. Altogether, a positive feed back scenario for brain stem compression may develop.

The skeletal muscle corresponds closely to the injured brain in the sense that its capillaries are permeable for small solutes. The skeletal muscle experiments showed a significant transcapillary filtration following simulation of distal opening of the spinal canal, and the filtration was more prominent with organ elevation (Fig. 3c) (19). These experiments also showed that the filtration rate was dependent on elasticity of the draining veins in the sense that it was larger at low than at high elasticity. It will be discussed, whether vein elasticity is a factor, which may explain the higher frequency of brain stem compression after LP in children and young individuals than in adults.

Fig. 5b shows an MR picture of the brain taken a few hours after LP, on a 14-years-old girl suffering from bacterial meningitis. The girl became unconscious shortly after LP, and ICP measurement about an hour afterwards showed a value above 60 mm Hg. A CT scan a few hours earlier was interpreted as normal. The MR picture illustrates that the scenario described above of a tight subtentorial situation with abolished CSF slit and a swollen brain is a clinical reality. Fig. 5a shows that 5 weeks later when the clinical situation has been normalised, there is free passage of CSF in the foramen magnum region.

Discussion

The present study points at the possibility that post-spinal headache and its postural characteristics may be explained by changes in venous blood volume just subdurally via intradural hydrostatic pressure effects following LP. This conclusion is supported by the fact that the volume alterations mainly occur close to the pain sensitive meningeal areas in the regions of transversing blood vessels, and the fact that MRI has demonstrated what is described as “meningeal enhancement”

after lumbar dural puncture in states of ongoing post-spinal headache (33). A vibrating behaviour of the subdural vein at the borderline level of elevation in the pain sensitive regions may be an additional factor for pain stimulation. This study also pointed to the fact that incidences of brain oedema and sagging of the brain with risk of brain stem compression may be predictable when considering the intradural hydrostatic pressure effects which may occur after LP in a state of disrupted BBB.

There are no clinical data presented of the effect on ICP of changing from supine to head-elevated position in patients developing post-spinal headache. Observations on cat from our laboratory showed that ICP was reduced from 11 to 5-6 mm Hg by puncture of the dural sac in supine position, and that it was reduced further to minus 11 mm Hg following head elevation up to 18 mm Hg (24 cm) above the opening (29). Even though these figures in absolute terms cannot be directly transferred to man, they support the view that ICP exceeds extradural venous pressure in supine position, implying a preserved subdural venous collapse, and that ICP may fall far below extradural venous pressure in upright position, implying disappearance of the venous collapse.

Clinical experience has shown that epidural blood patch by preventing leakage through occluding the perforation is an effective therapy to treat a persistent post-spinal headache, with a success rate of more than 90% (3, 4, 5, 34). However, as mentioned in the Introduction, a small CSF leakage is not compatible with a marked symptomatic influence on pain-sensitive regions of the brain, and especially not with the postural properties of the post-spinal headache, or the fact that the headache often disappears promptly after application of the blood patch. It has also been argued that post-spinal headache disappears after blood patch due to cessation of stretching of the bridge veins and other meningeal-related structures, following closure of the dural opening. Also this explanation is unlikely, as no distal gravitational movement of the brain can occur when changing from supine to

upright position, as the specific weights of the brain and CSF are the same (1.017-1.019 g/mL) (18, 35). Nor can a moderate decrease in CSF volume explain the development of brain stem compression during meningitis, especially since no signs of brain stem compression have been reported after drainage of CSF from the higher ventricular level in a state of non-collapsed ventricles. Instead, the hypothesis that post-spinal headache and brain stem compression after LP are triggered by hydrostatic pressure effects of distal opening of the intradural space to the atmospheric pressure, is compatible with all classical symptoms following LP and with the beneficial effect of a blood patch.

If BBB is intact, an increase in transcapillary pressure following LP will not induce any transcapillary filtration and there will be no increase in brain tissue volume (19, 20, 31). This means that the CSF slit around the brain stem area remains open, and CSF communicates freely in the whole intradural space (Fig. 2 c and d), and no brain stem compression will occur. In a state of disrupted BBB for small solutes, on the other hand, brain oedema may have been developed already before the dural puncture, the greater the higher the blood pressure (22, 36). The oedema combined with an increase in intracranial blood volume and further oedema due to transcapillary filtration when intradural pressure is reduced after LP, and a possible volume-induced distal movement of the brain may together separate the intradural space into two cavities as illustrated in Fig. 4c and d. Fig 5b shows that this scenario is a clinical reality. The hypothesis that opening of the spinal canal may induce filtration found support from the skeletal muscle experiments (19). Even though the filtration data from these experiments cannot be transferred to the brain in absolute terms due to the smaller filtration coefficient in the brain (30), they still can be used to confirm the principle that distal opening of the spinal canal at disrupted BBB may be compatible with brain oedema.

The ensuing pressure difference between the upper high pressure cavity and the lower distally open low-pressure cavity will press the brain stem towards the surrounding walls resulting in a more tight situation and a further increase in ICP. This effect will be stronger at an initial high compared to an initial low ICP and it will be stronger in upright than in supine position, in both cases due to a larger pressure fall between the two cavities. A positive feed back situation may develop when the tight situation is further aggravated, if a subsequent Cushing reflex triggers a simultaneous increase in blood pressure, which increases brain oedema (36). Venous stasis in the narrow subtentorial region may also aggravate the oedema. All these mechanisms taken together may explain the observations that signs of brain stem compression and even death may develop very quickly after LP (11). The significant transtentorial brain swelling showed in Fig 5b also must have developed quickly as the patient was fully communicable just a few hours before the MR investigation.

Results from our skeletal muscle experiments (19) indicated that elasticity of the draining veins could be of importance for cerebral haemodynamic alterations following distal opening of the dura. Thus, when extradural draining vessels are less elastic they may become less collapsed following head elevation, and a preserved fluid column on the venous side from brain to heart will counteract the hydrostatic effect of the distally open spinal column, in turn preserving the subdural venous collapse. Such a communicating vessel mechanism is in line with the fact that post-spinal headache is less frequent in older individuals with their more stiff vessels than in younger individuals (1, 7-10, 14). This mechanism also means a smaller transcapillary filtration rate at a state of disrupted BBB, as also indicated from the skeletal muscle experiments (19), a mechanisms which may contribute to the fact that brain stem compression after LP is less common in adults that in young individuals.

The MR picture in Fig. 5b illustrates that meningitis can be associated with significant brain oedema and a tight situation in the foramen magnum area, and that separation of the intradural space into two cavities is a clinical reality. It also shows that, when comparing with the normal situation (Fig. 5a), the volume of the ventricular system was only moderately reduced in the early phase of the illness despite a marked brain swelling and raised ICP. This finding agrees with the fact that cranial computed tomography (CT) often is judged normal during meningitis, in spite of marked intracranial hypertension (9-11), as also was the case with this patient.

The size of the dural opening and time for tightening vary between patients and must be dependent on the thickness and type of the needle used, which may explain the unpredictable consequences of dural perforation (4). The hypothesis presented, therefore is compatible with the view that a thin and atraumatic spinal needle reduces the risk of post-spinal headache and brain stem compression. It also supports the view that the patient with meningitis or other states with disrupted BBB should be in supine position for the first few hours after LP, and should not raise the head even once for the first period after the puncture to prevent initiation of the positive feed back process for brain stem compression. The hypothesis also means that the risk of developing brain stem compression in patients with cerebral infections is dependent on the degree of BBB disruption. The common view that post-spinal headache can be prevented by having the patient in supine position after LP finds no obvious support from the present study, as upright position does not increase the pressure across the dural opening, and should not reduce the ability to heal the dural tear.

Conclusion

The present study shows that post-spinal headache, brain stem compression and other LP-related effects may be predictable symptoms following LP, when considering expected hydrostatic effects of distal opening to the atmospheric pressure of the closed dural space. The hydrostatic concept is

compatible with venous blood volume variations close to the pain-sensitive meningeal regions, independent of CSF leakage. It is also compatible with brain oedema and brain sagging in a state of disrupted BBB, initiating venous stasis and brain stem compression, and there is no need of CSF leakage to explain these symptoms following LP. Hopefully, this study may contribute to a more respectful attitude to the often uncritically use of diagnostic LP in severely ill brain injured patients, and increased awareness of the fact that refraining from LP in selected cases with clinical signs of raised ICP or brain stem symptoms may be lifesaving.

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Legends

Fig. 1. The brain, the brain stem and the spinal cord enclosed in the fluid-filled dural space in a normal state without oedema before LP in supine (a) and upright (b) position, and after LP in supine (c) and upright (d) position. The assumed lowering of ICP from 10 to 6-8 mmHg when changing from supine (a) to upright position (b) is predicted from a reduced blood volume and the fact that the dura is not a completely rigid shell.

Fig. 2. A schematic illustration of brain circulation surrounded by the dura and the rigid cranium. The tissue pressure (ICP) is higher than the extradural venous pressure (P_V), causing a passive venous collapse just subdurally (R_{OUT}). Q: blood flow, P_A: arterial inflow pressure, P_C: hydrostatic capillary pressure, P_{OSM}: colloid osmotic pressure, P_{OUT}: venous pressure just proximal to R_{OUT}, R_A: arterial resistance, and R_V: venular resistance. P_{OUT} = ICP.

Fig. 3. Schematic illustrations of experiments with a skeletal muscle in a fluid-filled plethysmograph, simulating the haemodynamic situation on the venous side of the brain and effects of the spinal canal before and after LP in upright position. The situation in upright position before “LP” using an elastic draining vein is shown in (a), and a rigid draining vein in (b), and the situation also in upright position after “LP” using an elastic draining vein is shown in (c), and using a rigid draining vein in (d). The arrows point at the status of the venous collapse in upright position before (a, b) and after (c, d) opening of the “spinal tube” , and elastic or rigid draining veins (for details, see text).

Fig. 4. The brain, the brain stem and the spinal cord enclosed in the fluid-filled dural space at a state of disrupted BBB and oedema before LP in supine (a) and upright (b) position, and after LP in supine (c) and upright (d) position. The assumed lowering of ICP from 25 to 22 mmHg when

changing from supine (a) to upright position (b) is predicted from a reduced blood volume and the fact that the dura is not a completely rigid shell.

Fig. 5. An MR picture of the brain of a 14 years old girl suffering meningitis with raised ICP a few hours after LP (b), and 5 weeks later when ICP was normalised (a).

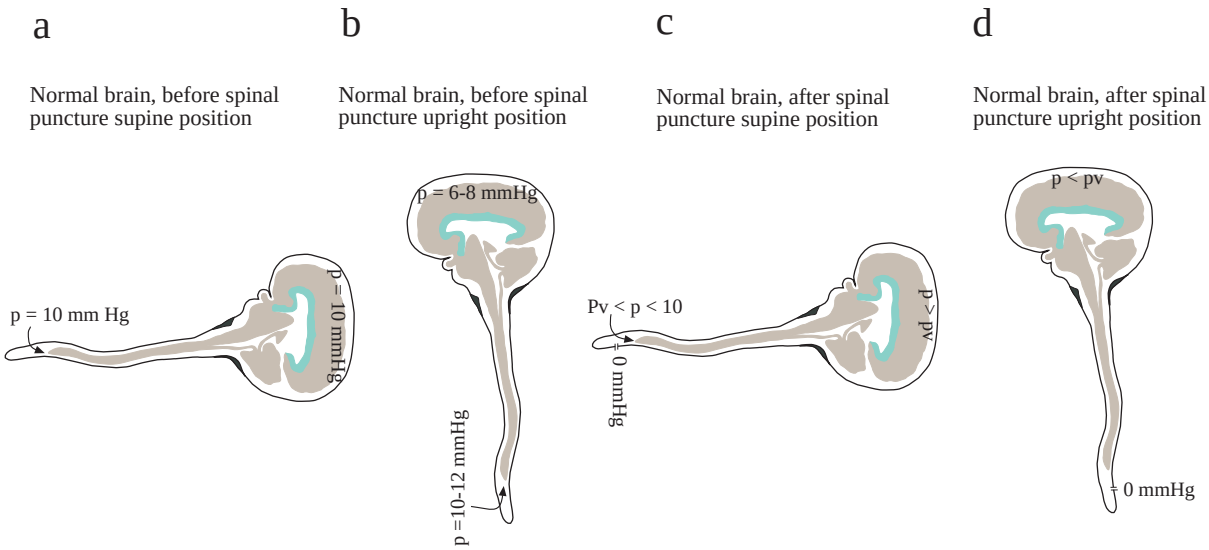

a
Normal brain, before spinal puncture supine position
p = 10 mm Hg
p = 10 mmHg
b
Normal brain, before spinal puncture upright position
p = 6-8 mmHg
p =10-12 mmHg
c
Normal brain, after spinal puncture supine position
Pv < p < 10
p > pv
d
Normal brain, after spinal puncture upright position
p < pv
0 mmHg

Fig.1

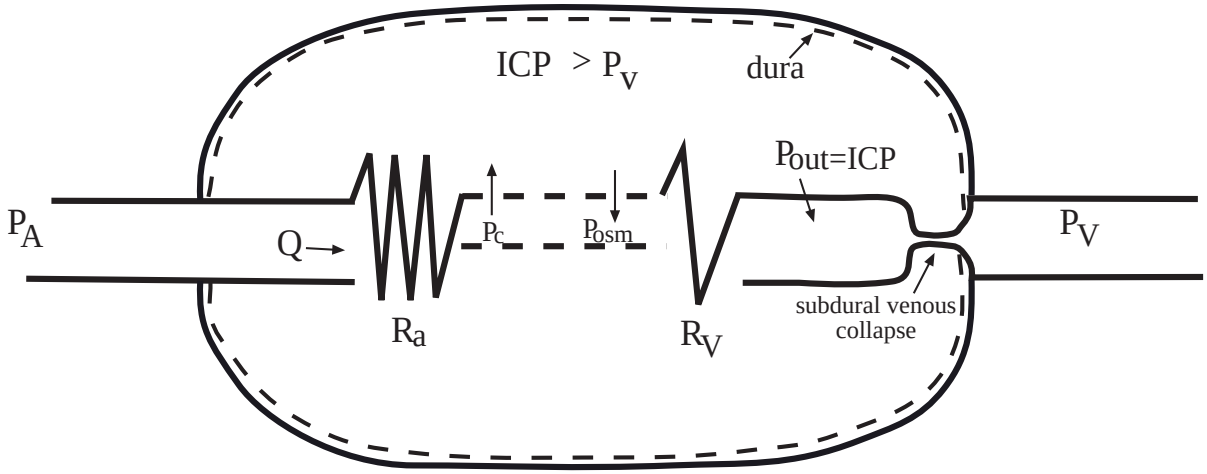

ICP > P_v
dura
P_{out}=ICP
P_A
Q
P_c
P_{osm}
P_V
subdural venous collapse
R_a
R_V

FIG.2

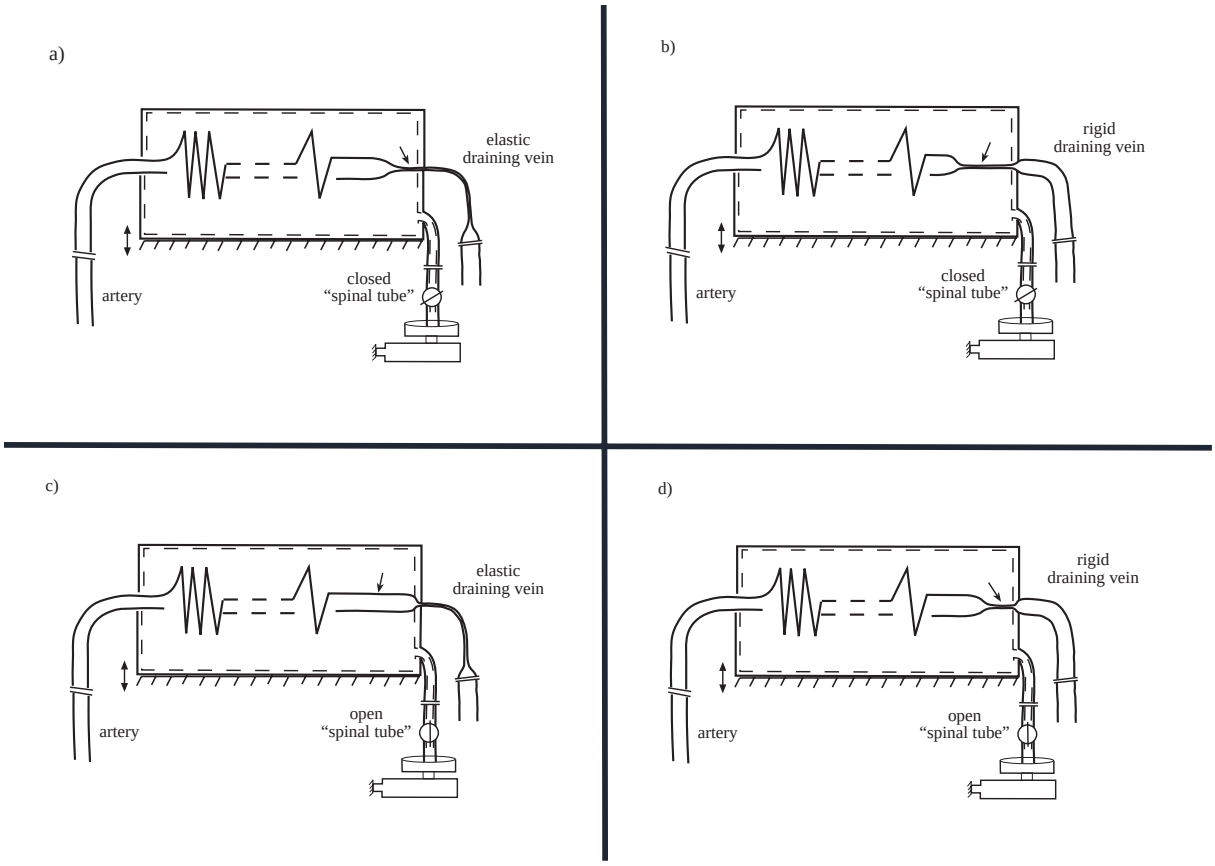

a)
elastic draining vein
artery
closed "spinal tube"
b)
rigid draining vein
artery
closed "spinal tube"
c)
elastic draining vein
artery
open "spinal tube"
d)
rigid draining vein
artery
open "spinal tube"

FIG. 3

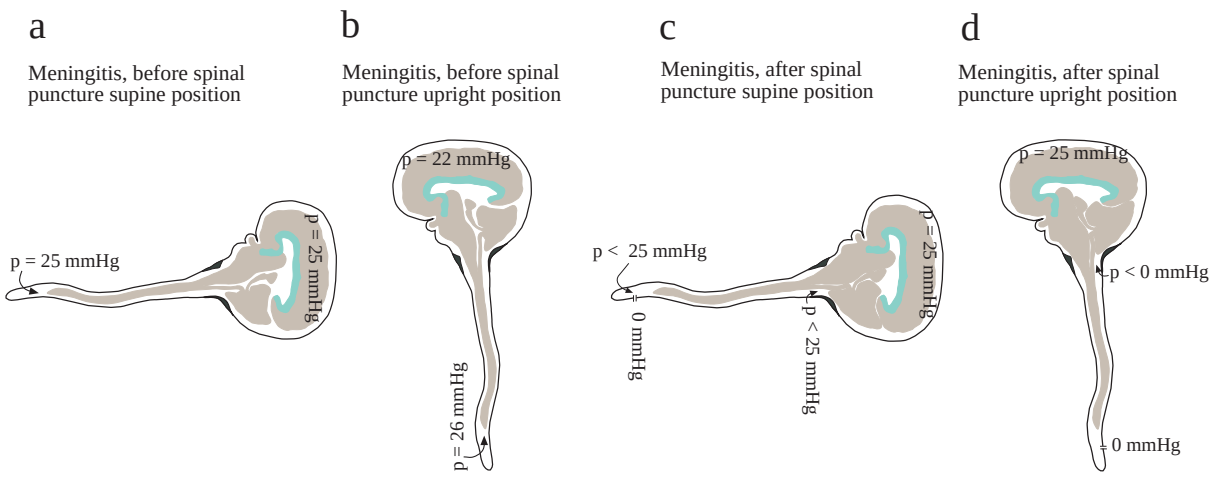
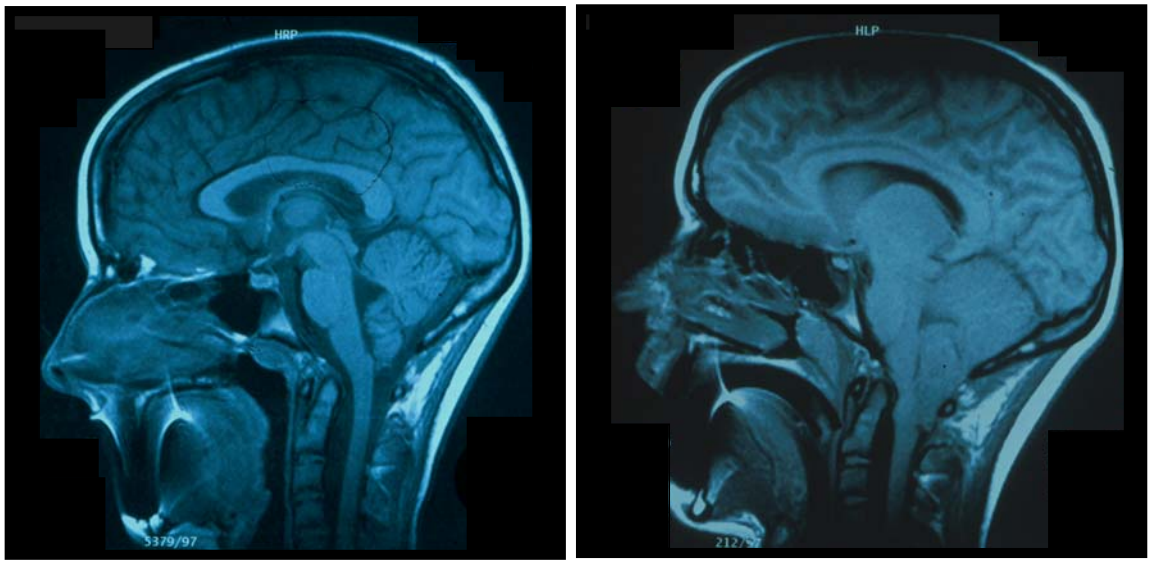

a
Meningitis, before spinal puncture supine position
p = 25 mmHg
p = 25 mmHg
b
Meningitis, before spinal puncture upright position
p = 22 mmHg
p = 26 mmHg
c
Meningitis, after spinal puncture supine position
p < 25 mmHg
= 0 mmHg
p = 25 mmHg
p < 25 mmHg
d
Meningitis, after spinal puncture upright position
p = 25 mmHg
p < 0 mmHg
= 0 mmHg

FIG. 4



Veronica

FI G. 5 a and b