DAMAGING VENOUS REFLUX INTO THE SKULL OR SPINE: RELEVANCE TO MULTIPLE SCLEROSIS

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ABSTRACT

Unequal propagation of central venous excess pressure into the different cerebral and spinal venous drainage systems is the rule rather than the exception. The intensity of the forces thus to be exerted on vulnerable cerebrospinal structures by the resulting pressure-gradients in the craniovertebral space is unknown. There is a need to consider the various conditions which may cause individual proneness to heavier reflux into particular cerebral as well as epi- and subdural spinal venous compartments. An attempt is made to indicate eventual consequences of excessive retrograde dilatation especially of internal cerebral veins. The importance of elucidating the neuropathological and clinical implications of undue reflux into the skull or spine is deduced from the probability of relations between localized backflow into the craniovertebral space and unexplained cerebrospinal diseases. In this regard the features of multiple sclerosis are discussed.

INTRODUCTION

Detrimental effects of reflux from deep crural veins ("Blow-out", "Battering-ram impulses") and abdominal veins ("Blow-down") in superficial veins of the leg are generally known. This contrasts with the near absence of consideration of venous regurgitation into the skull or spine. Little attention has been paid to the problem of venous variations and anomalies favouring retrograde propagation of central venous excess pressure into particular cerebral or spinal drainage systems. The question of how much vulnerable craniovertebral contents suffer from predisposition to localized dilatation of cerebral or spinal veins is an open one.

REFLUX INTO, AND FLOW FROM, SUB- AND EPIDURAL VEINS

In the closed craniovertebral space, retrograde engorgement...
of a cerebral or spinal venous compartment implies simultaneous displacement within and from the skull and spine. The forces thus exerted on the cerebrospinal structures involved correspond to the momentary pressure-gradients between the cerebral or spinal vessels being dilated, and those being compressed. The violence of the resulting movements further depends on the volume of, as well as the resistance against, the momentary shift into, within, and from the craniovertebral space. As forceful compression of voluminous cervical, thoracic, or abdominal veins gets the more serious, the more the blood momentarily displaced loads isolated cerebral or spinal tributary vessels.

Reflux from an internal jugular vein can be more or less limited to either the superior sagittal sinus, a straight sinus(1,2,3), or even an inferior petrosal sinus(4). The smaller the simultaneous backflow in the other veins, e.g. because of venous valves(5) and kinkings, the greater intracranial engorgement is to be expected. During reflux into epidural or even radicular lumbosacral veins, on the other hand, the upper sub- and epidural vessels will yield more easily, the greater number of valves(5) and kinkings preventing competing backflow in the internal jugular, subclavian, and vertebral veins, as well as in spinal affluents of azygos and hemiazygos veins.

Previous dilatation, especially when coopted with diminution of tension or tensile strength of the vessels to be expanded - whatever the cause -, increases the risk of excessive retrograde engorgement from big central veins. Low CSF-pressure(6) also favours reflux in skull or spine in disposing of great volumes of sub- and epidural blood, and in impairing the tension of the dural sac. Concerning a concomitant rise of pressure behind the optic disc and venous reflux into the eye, the same applies to low intraocular pressure(7).

Considering the various possibilities of localized reflux into sub- and epidural veins, more detailed consideration may be given to the dynamics of reflux in the internal cerebral veins: Excess pressure in these rather isolated veins (8,9) close to larger bulks of movable CSF will easily distend the soft perivenous white matter. Undue periventricular (10) and subcortical(11) venous engorgement may result, if the local overpressure cannot be sufficiently relieved along its path from the internal jugular vein to the internal cerebral veins. An accelerated yielding efflux is thus possible, both from separated cerebral vessels into capacious cervicobrachial veins, and from the spine into less compressed external, for instance, abdominal veins. Ventriculocisternal pressure-gradients effected thereby can especially distend the optic chiasm. CSF pressed into the sheath of the optic nerve will mainly act on the optic disc. Surges of CSF displaced into the spinal canal can damage the cord and
nerve-roots, in particular at narrowed passages. In both tending to pull the spinal cord and to distend the dural sac the surges may indirectly tear the cord along the ligaments anchoring it to its sheaths.

Violent reflux into the brain or spinal canal should cause disseminated cerebrospinal damage of a peculiar pattern and nature. In the areas of overstrain the mechanically most vulnerable unit of myelin-sheaths and oligodendroglia will be injured. The more resistant connective and glial tissue may react with adaptive, isomorphic overgrowth. The neurons, protectively embedded in glia, should be relatively spared.

**CLINICAL AND PATHOLOGICAL CORRELATIONS**

Localized venous hypertension in the spinal canal could be involved in the pathogenesis of epidural varicosis(12,13) or of the Foix-Alajouanine syndrome(14): Nothing excludes the possibility that hemorrhage or destruction related to spinal, cerebral(15,16), or retinal veins(17,18) occasionally results from reflux, overloading the walls or surrounding structures of a particular venous drainage.

One obvious possibility is the correspondence between the probable effects of undue reflux into the internal cerebral veins and multiple sclerosis(MS). Almost half a century ago it was stated: "Artificially produced perivenous extravasations of contrast-medium closely simulate the shape and the distribution of plaques in advanced stages of MS(solution had been forced into the great vein of Galen under heavy pressure)."(10). Thus the uneven venous dilatation(19,20), leakage(21), and degeneration(22), as well as the retrograde(23) perivenous spread of the patches(24,25,20,22,26), perivascular dilatations(27), and hemorrhages in the brain in MS(28,29) may be due to intermittent retrograde venous hypertension.

Venous displacement of craniovertebral contents further can explain other typical localizations of the demyelination and isomorphic gliosis characterizing MS:
- The peculiar involvement of the optic chiasm(30,31)
- The lining of bulbar(32) and spinal surfaces, as correspondingly found after manipulation of CSF(33)
- The dissemination of plaques along the cord, remembering the damage after spinal concussion(34)
- The center of the main patches of the cord towards the denticulate ligament(35) and septum posticum, as caused by displacement of the cord in its sheaths(36)
- The degenerations infiltrating the cord along its external framework(37)
- The focusing of plaques on narrow places in the spine(38)
- The limitation of primary lesions of the peripheral nerve on the nerve-root(39,40,41)
From a clinical point of view the suggestion "Acute and fleeting symptoms, and exacerbation of existing symptoms (in MS), may, however, be due to temporary vascular engorgement in or near the plaque." (42) anticipated the pathomechanism postulated above. Accordingly young patients suffering from MS complained: If I press my vigour momentary vanishes, or I suddenly meet with a painful electric shock, going down my back to the legs. Similarly the initial manifestations of MS (43), its relation to accident and stress (44, 45, 46), and various aggravations of the disease following physical exertion (47, 48, 49, 50) appear primarily compatible with the notion of the effect of excessive outbursts of intermittent venous reflux. The remissions of MS during cessation of activity, on the other hand, are often so remarkable that they stimulated consideration of a causal curative effect of rest (51).

Contemporary concepts concerning the etiopathogenesis of MS do not explain either the cisterno-ventricular reflux of CSF (52), nor the fragments of myelin in the CSF (53), nor the periphlebitis retinae (54), which also constitute features of MS. Thus, it might not be inappropriate to consider, whether the various immunological phenomena observed in MS could not be released by damaging venous distensions and displacements of cerebrospinal structures (55, 56, 57, 58, 58, 60).

CONCLUSION

The pathogenic role of the venous variations and anomalies predisposing to reflux into particular cerebral or spinal drainages needs to be clarified. The damage to be caused by venous distension and displacement of craniovertebral contents offers an explanation for some features of MS which are not understood. Attempts to prevent corresponding injuries could prove to be for the benefit of patients suffering from unexplained cerebrospinal diseases not curable as yet.

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