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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 unexplicated 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.

REFERENCES

1. Gibbs EL, Gibbs FA. The cross section areas of the vessels that form the torcular and the manner in which flow is distributed to the right and to the left lateral sinus. Anatomical Record 59: 419, 1934
2. Delmas A, Chifflet J. Le pressoir d'Hérophile. Comptes rendus de l'Association des Anatomistes. 1950: 123
3. Saxena RC, Beg MAQ, Das AC. Double straight sinus. Journal of Neurosurgery 39: 540, 1973(fig. 1 UR)
4. Zuckerkandl E. Beiträge zur Anatomie des Schläfenbeins. Monatsschrift für Ohrenheilkunde 7: 101, 1873
5. Kampmeier OF, La Fleur Birch C. The origin and development of the venous valves, with particular reference to the saphenous district. The American Journal of Anatomy

38: 451, 1927

6. Linke GP. Liquorzirkulation und Liquordynamik. p 41 in Der Liquor cerebrospinalis(RM Schmidt, ed) VEB Volk und Gesundheit, Berlin 1968

7. Duke-Elder S, Jay B. Ocular hypotony. p 724 in System of Ophthalmology, vol XI(S Duke-Elder, ed) Kimpton, London 1969

8. Goetzen B. Veines internes du cerveau. Archives d'Anatomie Pathologique 12: 126, 1964

9. Hassler O. Deep cerebral venous system in man. Neurology 16: 505, 1966

10. Schlesinger B. The venous drainage of the brain, with special reference to the Galenic system. Brain 62: 274, 1939

11. Pfeifer RA. Anfänge einer neuen Herdlehre. p 194 in Grundlegende Untersuchungen für die Angioarchitektonik des menschlichen Gehirns. Springer, Berlin 1930

12. Epstein BS. Low back pain associated with varices of the epidural veins simulating herniation of the nucleus pulposus. American Journal of Roentgenology 57: 736, 1947

13. Serfling HJ, Parnitzke KH. Die epidurale Varicosis spinalis als Ursache von Wurzelsyndromen. Zeitschrift für Orthopädie und ihre Grenzgebiete 89: 437, 1958

14. Weisberg LD. Vascular disease of the spinal cord. p 195 in Merritt's Textbook of Neurology, 7th ed(LP Rowland, ed) Lea & Febiger, Philadelphia 1984

15. Schwartz P, Fink L. Morphologie und Entstehung der geburtstraumatischen Blutungen in Gehirn und Schädel des Neugeborenen. Zeitschrift für Kinderheilkunde 40: 427, 1926

16. Alexander L. Clinical and neuropathological aspects of electrical injuries. Journal of Industrial Hygiene and Toxicology 20: 191, 1939

17. Dastur OK, Singhal BS. Eales' disease with neurological involvement. Journal of Neurological Sciences 27: 313, 1976

18. Roveda JM, Roveda CE. Pression veineuse dans les hémorrhagies expulsives et les pertes de vitré. Journal Francais de l'Ophtalmologie 2: 343, 1979

19. Macchi G. The pathology of the blood vessels in multiple sclerosis. Journal of Neuropathology and Experimental Neurology 13: 378, 1954

20. Fog T. The topography of plaques in multiple sclerosis with special reference to cerebral plaques. Acta Neurologica Scandinavica 41: 1, 1965, Suppl. 15(fig 10)

21. Broman T. Blood-brain barrier damage in multiple sclerosis. Acta Neurologica Scandinavica 10: 21, 1964, Suppl. 40

22. Lumsden CE. The neuropathology of multiple sclerosis. p 203 in Handbook of Clinical Neurology, vol 9 (PJ Vinken, GW Bruyn, eds) North Holland, Amsterdam / Elsevier, New York 1970

23. Adams CWM. The general pathology of multiple sclerosis. p 203 in Multiple Sclerosis(JF Hallpike, CWM Adams, WW Tourtellotte, eds) Chapman and Hall, London 1983

24. Bruce A, Dawson JW. Preliminary communication on the pathology of disseminated sclerosis. Journal of Pathology and Bacteriology, 15: 126, 1911

25. Putnam TJ, Adler A. Vascular architecture of the lesions of multiple sclerosis. Archives of Neurology & Psychiatry 38: 1, 1937

26. Adams CWM. Pathology of multiple sclerosis: Progression of the lesion. British Medical Bulletin 33: 15, 1977

27. Borst M. Zur pathologischen Anatomie und Pathogenese der Multiplen Sklerose des Gehirns und Rückenmarks. Beiträge zur pathologischen Anatomie und allgemeinen Pathologie 21: 308, 1897

28. Scheinker M. Histogenesis of the early lesions of multiple sclerosis. I. Significance of vascular changes. Archives of Neurology & Psychiatry 49: 178, 1943

29. Jankovic J, Derman H, Armstrong D. Haemorrhagic complications of multiple sclerosis. Journal of Neurology, Neurosurgery, and Psychiatry 43: 76, 1980

30. Walsh FB, Hoyt FW. Degenerative diseases: Disseminated sclerosis. p 982 in Clinical Neurophthalmology, vol I, 3rd ed. William & Wilkins, Baltimore 1971(fig 7.96)

31. Zimmerman HM, Netsky MG. The pathology of multiple sclerosis. Res Publ Assoc Res Nerv Ment Dis 28: 271, 1950(fig 109)

32. Ule G. Saumartiger Entmarkungsherd der Brücke bei multipler Sklerose. p 9-57 in Organpathologie, vol III (W Doerr, ed) Thieme, Stuttgart 1974(fig 9.74)

33. Bunge RP, Settlage PH. Neurological lesions in cats following cerebrospinal fluid manipulation. Journal of Neuropathology and Experimental Neurology 16: 471, 1957

34. Lhermitte J, Lévy G, Nicolas M. Les sensations de décharge électrique, symptôme précoce de la sclérose en plaques. Presse médicale 39: 610, 1927

35. Oppenheimer DR. The cervical cord in multiple sclerosis. Neuropathology and Applied Neurobiology 4: 151, 1978

36. Kahn EA. The role of the dentate ligaments in the spinal compression and the syndrome of lateral sclerosis. Medical Bulletin, University of Michigan 13: 1, 1947

37. Frommann C. Über die Histologie der grauen Degeneration. p 49 in Untersuchungen über die normale und pathologische Anatomie des Rückenmarks, 2. Teil. Frommann, Jena 1867

38. Brain R, Wilkinson M. The association of cervical spondylosis and disseminated sclerosis. Brain 80: 456, 1957

39. Joffroy A. Note sur un cas de sclérose en plaques disséminées. Gazette Médicale de Paris 41ième année, 3ième série, tome XXViem: 308/324, 1870

40. Liouville H. Nouvelle observation détaillée de sclérose en îlots multiples et disséminés du cerveau, de la moelle et des nerfs rachidiens. Gazette médicale de Paris 41ième année, 3ième série, tome XXViem: 259/269, 1870

41. Schob. Wurzelfibromatose bei multipler Sklerose. Muenchener Medizinische Wochenschrift 59: 1831, 1912

42. Mc Menemey WH. Multiple sclerosis. p 2252 in Systemic Pathology, vol V, 2nd ed(WstC Symmers, ed) Churchill Livingstone, Edinburgh 1979

43. Twomey JA, Espir MLE. Paroxysmal symptoms as the first manifestations of multiple sclerosis. Journal of Neurology, Neurosurgery and Psychiatry 43: 296, 1980

44. Kaiser E. Trauma als ätiologisches Moment der multiplen Sklerose. Thesis, Berlin 1889

45. Currier RD, Martin EA Woosley PC. Prior events in multiple sclerosis. Neurology 24: 748, 1974

46. Bobowick AR, Kurtzke JF, Brody JA, Hrubec Z, Gillespie M. Twin study of multiple sclerosis: An epidemiologic inquiry. Neurology 28: 978, 1978

47. Abb L, Schaltenbrand G. Statistische Untersuchungen zum Problem der multiplen Sklerose, III. Ätiologische Faktoren. Deutsche Zeitschrift für Nervenheilkunde 174: 219, 1956

48. Goldstein JE, Cogan DG. Exercise and the optic neuropathy of multiple sclerosis. Archives of Ophthalmology 72: 168, 1964

49. Prineas JW. The etiology and pathogenesis of multiple sclerosis. p 107 in Handbook of Clinical Neurology, vol 9(PJ Vinken, GW Bruyn, eds) North Holland, Amsterdam / Elsevier, New York 1970

50. Mc Alpine D. Precipitating and aggravating factors. p 99 in Multiple Sclerosis, a reappraisal(D Mc Alpine, CE Lumsden, ED Acheson, eds) Churchill Livingstone, Edinburgh 1972

51. Walter FK. Multiple Sklerose. p 246 in Lehrbuch der Nervenkrankheiten, 2nd ed(H Curschmann, F. Kramer, eds) Springer, Berlin 1925

52. Inzitari D, Capparelli R, Sita D, Barontini F, Marini P, Amaducci L. Isotopic cisternography in multiple sclerosis: possible correlation with disease course. p 250 in Immunological and Clinical Aspects of Multiple Sclerosis. MTP Press, Lancaster 1984

53. Herndon RM, Johnston M. A method for electron microscopic study of cerebrospinal fluid sediment. Journal of Neuropathology and Experimental Neurology 29: 320, 1970

54. Engell T, Andersen PK. The frequency of periphlebitis retinae in multiple sclerosis. Acta Neurologica Scandinavica 65: 601, 1982

55. Rocklin RE, Sheremata WA, Feldman RG, Kies MW, David JR. The Guillain-Barré syndrome and multiple sclerosis. In vitro cellular responses to nervous tissues antigens. The New England Journal of Medecine 284:808, 1971

56. Detlav IE, Drake RB, Skushka MA. Delayed hypersensitivity in patients with craniocerebral injuries(russian). Zh Nevropatol Psikhiatr 80: 326, 1980

57. Kennedy PE, Lisak R. Do patients with demyelinating diseases have antibodies against human glial cells in their sera? Journal of Neurology, Neurosurgery and Psychiatry 44: 164, 1981

58. Roström B, Link H. Oligoclonal immunoglobulins in cerebrospinal fluid in acute cerebrovascular disease. Neurology 31: 590, 1981

59. Miller JR, Burke AM, Bever ChT. Occurrence of oligoclonal bands in multiple sclerosis and other CNS diseases. Annals of Neurology 13: 53, 1983

60. Mizrachi Y, Ohry A. Systemic humoral factors participating in the course of spinal cord injury. Paraplegia 21: 287, 1983