Pathophysiology of Syringomyelia

by

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Submitted as part requirement for the degree of Doctor of Philosophy, August 1996
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ABSTRACT

The normal physiology of cerebrospinal fluid (CSF) in the spinal subarachnoid space and spinal cord and the pathophysiology of non-communicating syringomyelia are poorly understood. The hypothesis examined in this thesis is that CSF is driven from the subarachnoid space into perivascular spaces and the central canal by arterial pulsations and that this is the driving force for the development of non-communicating syringomyelia. Horseradish peroxidase (HRP) was used as a CSF tracer in rats and sheep. In normal rats and in normal sheep CSF flowed rapidly from the subarachnoid space, through perivascular spaces and into the central canal. Flow into the central canal was not via the fourth ventricle or the caudal opening of the central canal. The effect of arterial pulsations on this flow was examined by ligating the brachiocephalic artery in sheep before injecting HRP into the subarachnoid space. There was no flow into the central canal in sheep with damped arterial pulsations. Reducing the spinal subarachnoid pressure did not appear to alter flow into the central canal. CSF flow was also studied in the rat intraparenchymal kaolin model of non-communicating syringomyelia. Rapid flow into the central canal occurred at 1 day, 3 days, 1 week and 6 weeks after kaolin injection. There was rapid flow into isolated, enlarged segments of central canal even when there was evidence that the enlarged segments were causing pressure effects on surrounding tissue. These results support the hypothesis that arterial pulsation-driven CSF flow from perivascular spaces into the central canal is the driving force for the development of non-communicating syringomyelia. An additional finding from this work was that rapid perivascular CSF flow occurs in the cerebellum as it does elsewhere in the nervous system. A technique for studying the three-dimensional morphology of the human central canal was also developed.