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THE INTRAVENTRICULAR AND VASCULAR PRESSURES AND THEIR RELATIONSHIP IN THE ELASMOBRANCH

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A study has been made of the cerebral ventricular and vascular pressures and their relationships in the elasmobranch <u>S. acanthias</u> as part of a larger study of the comparative physiology of the CSF. Pressure measurements were made in the conus arteriosus, the dorsal aorta, the orbital sinus, anterior cardinal vein, the lateral and cerebellar cerebral ventricles, and the space between the brain and the skull.

The passage of the arterial blood through the gills before going to the brain caused a reduction in blood pressure from about 50/35 mm Hg to about 30/25 mm Hg or 40% reduction in systolic pressure and 29% reduction in diastolic pressure. The pulse pressure dropped from 14 mm of mercury to 5 or about 64%. In nine experiments, the mean arterial pressure distal to the gills was 25/20 mm Hg. In 11 experiments, the mean intraventricular pressure was 48/45 mm H₂O with a pulse which ranged from 1.1 to 5 mm H₂O with a mean of about 3 mm of water. The pressure seemed to be about the same in the two lateral ventricles and in the cerebellar ventricles, but communication sometimes did not seem to be very good. The initial measurements always showed equal. The orbital venous sinus was usually lower than the intraventricular pressure with a mean of 19 mm H₂O and never showed any pulsation. The anterior cardinal vein pressure seems unrelated to the CSF intraventricular pressures, sometimes being somewhat higher and sometimes lower than the intraventricular pressure. The mean was 55/51. The anterior cardinal vein always showed a pulse of about 3 to 5 mm of water but this pulse was related to the auricular contraction of the heart. The cardinal vein pulse was completely out of phase with the CSF pulse which was exactly in phase with the arterial pulse. The extradural fluid pressure was usually about 7 mm H₂O below that of the CSF but never showed any pulsation.

The intraventricular pulsations found in the elasmobranch were considerably smaller than those found in the mammalian species with choroid plexuses of similar size. The reason for this was the damping of the arterial pulse pressure by its passage through the gills before re-entering the dorsal aorta.

These observations are consistent with the hypothesis that the pulsatile pressure caused by the choroid plexus as each heart beat fills it with blood is a major factor in the enlargement of the cerebral ventricles.