Hydrodynamic Properties of Certain Shunt Assemblies for the Treatment of Hydrocephalus*

Part 1: Report of a Case of Communicating Hydrocephalus with Increased Cerebrospinal Fluid Production Treated by Duplication of Shunting Device


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MANAGEMENT of the following unusual case of communicating hydrocephalus has resulted in new methods and considerations regarding the selection, use, and regulation of shunts, reported in this paper.

Case Report

First Admission. A previously healthy 17-month-old girl entered the Bronx Municipal Hospital Center on May 4, 1965, because of irritability and unsteadiness of gait of 1½ weeks’ duration. The head circumference was 53 cm. Macewen’s sign was elicited. Marked ataxia was present in the trunk and limbs. Spinal fluid pressure was 400 mm. Skull radiographs demonstrated suture diastases. Ventriculography and pneumoencephalography revealed pan-ventricular enlargement with a frontal brain mantle thickness of 3 cm (Fig. 1). A diagnosis of idiopathic communicating hydrocephalus was made.

On the 9th hospital day, the patient developed left hemiparesis and right abduces palsy progressing rapidly to coma and decerebrate rigidity with dilatation of the right pupil. Recovery of consciousness with residual hemiparesis and pupillomotor abnormality occurred on external ventricular drain-

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Fig. 1. Brow-down film from pneumoencephalogram of first hospital admission.

age. Consciousness became impaired, and decerebrate manifestations reappeared at ventricular outflow pressures about 175 cm of water. The volume of CSF collected from the ventriculostomy (Table 1) at outflow pressures below that critical magnitude varied between 45 and 108 ml per hour.

On the 14th hospital day, a Pudenz-Heyer ventriculoatrial shunt system was inserted (infant-size cardiac tubing), the flushing de-
vice being placed in the right lower parietal region. The cardiac tip had a hydrostatic closing pressure in air of 70 mm of water. After a satisfactory awakening in the recovery room, the patient lapsed into coma. Ventriculostomy output averaged 81 ml per hour despite evidently unobstructed functioning of the shunt assembly (Table 1). On the 16th hospital day, a standard size Pudenz-Heyer cardiac catheter was substituted with the expectation of increasing the volume flow capacity of the shunt system. However, ventriculostomy outflow showed no appreciable reduction, varying between 67 and 25 ml per hour. Since the ventriculostomy outflow pressures were consistently above the hydrostatic opening pressure of the shunt assembly, we concluded that the properly functioning shunt system was overloaded hydrodynamically by a volume of CSF corresponding to that collected externally.

It seemed mandatory, therefore, to determine the maximal flow capacities of the Pudenz-Heyer shunt assembly at CSF pressures within both the physiological range and the more narrow range tolerated by the patient (150 mm of water maximum). These determinations, which were carried out in vitro, indicated that the flow capacity of a second Pudenz-Heyer shunt system would be required to accommodate the total volume of CSF needing disposal. On the 23rd hospital day, a duplicate ventriculostomy shunt was installed with the flushing device in the right mid-parietal region (Fig. 2) and the cardiac tubing threaded into the right jugular vein alongside the first tubing. The thickness of the parietal brain mantle, estimated by ventricular puncture, was 5 cm. The ipsilateral placement was selected to avoid bilateral homotopic cortical lesions.

Following this procedure, ventriculostomy outflow was negligible. External ventricular drainage was discontinued on the 25th hospital day. The patient was discharged on the 37th hospital day. Her head circumference was 51.5 cm. She showed developmental impairment, residual left hemiparesis, and ataxia which diminished gradually.

**Second Admission.** The patient was re-admitted 18 months later (January 15, 1967) because of recent swelling around the lower parietal flushing device. Head circumference was 51.5 cm. The mid-parietal flushing device could not be compressed, whereas the lower parietal device appeared to be functioning normally although overlaid by a non-tender, fluctuant swelling 1 cm in thickness. Non-function of one shunt had apparently resulted in subgaleal cerebrospinal fluid accumulation and recurrence of symptomatic hydrocephalus.

The patient became lethargic and had seizures accompanied by decerebrate phenomena. Ventriculography again demon-