

## Successful treatment of perinatal intraventricular haemorrhage

By

E. PARAICZ

Institute of Neurosurgery, Budapest, Hungary

Received 7th February, 1979

In 18 cases of perinatal intraventricular haemorrhage, continuous ventricular drainage was applied. As a result, ICP hypertension was inhibited and thus brain damage prevented. The early intervention prevented the formation of hydrocephalus (evidence of shunt dependence), and aspiration of the bleeding prevented DIC and maintained coagulation factors.

Massive haemorrhage into the cerebral ventricles is one of the tragic occurrences during the perinatal period. Almost all patients are lost; and even to date 25% of the perinatal mortality is due to ventricular haemorrhage [1, 2, 4]. The condition occurs mainly in premature infants

with a birth-weight below 2000 g, and the coincidence with hyaline membrane disease is characteristic; according to Tsiantos et al. [3] it occurs in 95%.

Successful treatment is principally based on the improvement of medical care in two fields. First, the improv-

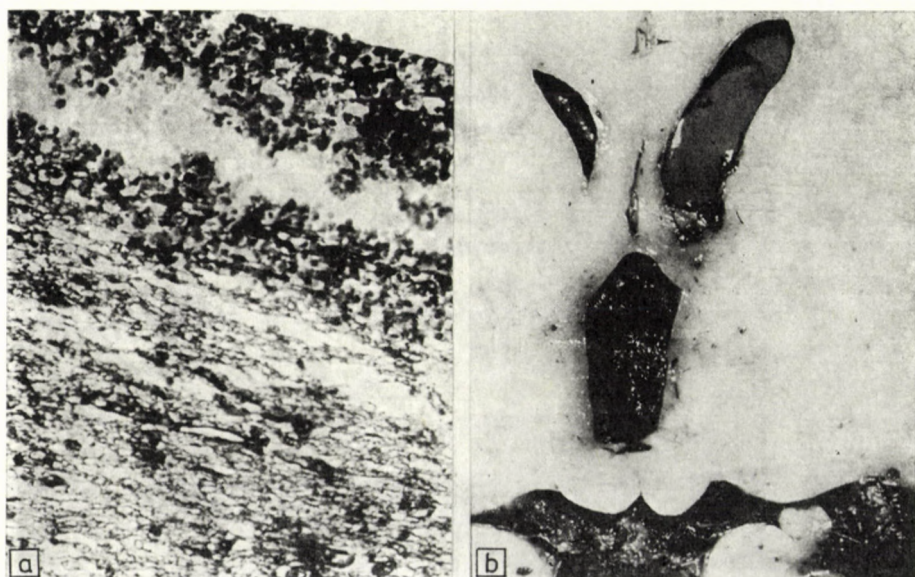


FIG. 1. a) Subependymal haemorrhage near the foramen of Monro. b) Bleeding in intraventricular cavity



ment of perinatal intensive care (PIC) and of respiratory therapy. Second, improvement of the neurosurgical results by the widespread use of continuous drainage of the CSF spaces.

In the last 3 years, we have applied a method of treatment similar as that of Wise et al [5]. When elaborating the method, the pathological and pathophysiological considerations were as follows.

In the premature newborn the fragile veins of the germinal matrix rupture in the subependymal region of the ventricular wall, mostly at the head of the caudate nucleus near the foramen of Monro. This leads to subependymal haemorrhage (Fig. 1), and then breaks into the ventricular cavity. In hyaline membrane disease venous congestion, acidosis, and hypervolaemia in connection with sodium bicarbonate therapy are predis-

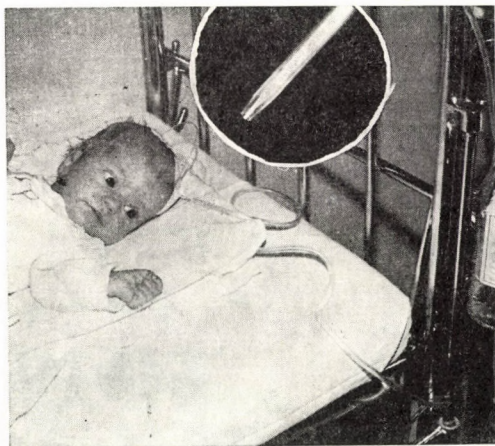


FIG. 2. Simple method of continuous external CSF-drainage

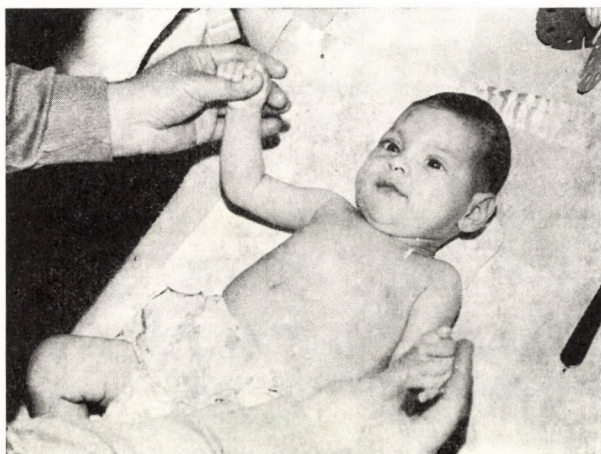


FIG. 3. M. Anette, 4 months after therapy

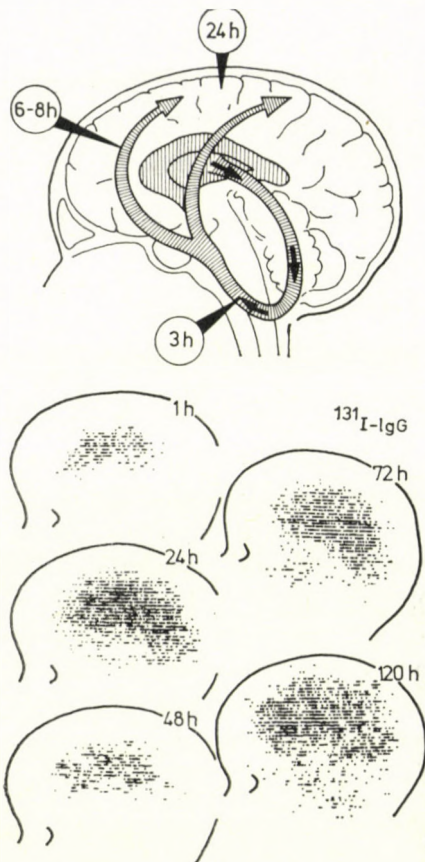


FIG. 4. The persistence of marked proteins for some days in the ventricular cavity indicates a slow CSF-absorption

posing factors. Congenital thrombopenia and DIC also occur.

Haemorrhage in the CNS causes a blockade of absorption and/or of passage. This gives rise to ICP hypertension with increasing respiratory disturbance, acidosis, and finally a reduction of cerebral blood flow.

A silicone catheter was introduced into the frontal horn of the lateral ventricle (Fig. 2). The fluid was received in a sterile reservoir through a sterile tube. The pressure level in the system was about 100 mm H<sub>2</sub>O.

Drainage was maintained for several days or 1–2 weeks until the continuously observed new bleeding in the ventricle had stopped and the CSF became clear.

In this way we could avoid the high ICP, prevent the occurrence of brain ischaemia, and the serious impairment of cerebral function. Bradycardia diminished, consciousness returned, vomiting ceased, and tube or oral feeding became possible. Subsequently, the process may take two different courses.



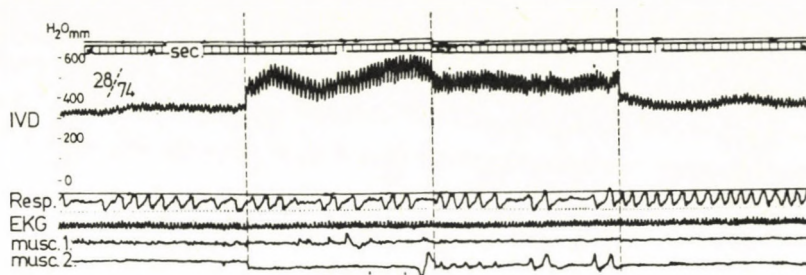


Fig. 5. ICP monitoring shows pressure-waves and an elevated mean pressure level

*Course 1.* Following the immediate management of the respiratory distress and early introduction of continuous ventricular drainage, the disturbed quotient CSF pressure/flow is normalized, resulting in a steady state without shunt implantation (3 cases in our material Fig. 3).

*Course 2.* If the prolonged bleeding is left untreated or if only ventricular punctures are done during the first period and continuous drainage is delayed, the disturbed CSF resorption

causes ventricular reflux, as shown by isotope cisternography (Fig. 4). The continuous recording of ICP revealed infantile A waves and an elevated pressure (Fig. 5). So hypertensive hydrocephalus develop which then necessitates atrial shunt nplantation (See Table I).

Five patients died immediately after the treatment; the majority are alive. The late functional results are fairly good, the IQ is not worse in other patients with hydrocephalus.

TABLE I. RESULTS

Without shunt	3 cases	18
normally developed	6	
Shunted		
retarded	4	
Died	5	

#### REFERENCES

1. HARCKE, H. T., NAEYE, R. L., STORCH, A., BLANCK, W. A.: Perinatal cerebral intraventricular hemorrhage. *J. Pediat.* **80**, 37 (1972).
2. TOWBIN, A.: Cerebral intraventricular hemorrhage and subependymal matrix infarction in the fetus and premature newborn. *Amer. J. Path.* **52**, 121 (1968).
3. TSANTOS, A., VICTORIN, L., RELIER, J. P., DYER, N., SUNDELL, H., BRILL, A. B., STAHLMAN, M.: Intracranial hemorrhage in the prematurely born infant. *J. Pediat.* **85**, 854 (1974).
4. VALDES-DAPENA, M. A., AREY, J. B.: The causes of neonatal mortality: an analysis of 501 autopsies of newborn infants. *J. Pediat.* **77**, 336 (1970).
5. WISE, B. E., BALLARD, R.: Hydrocephalus secondary to intracranial hemorrhage in premature infants. *Child Brain* **2**, 234 (1976).

E. PARAICZ, M. D.,

Amerikai út 57.

H-1145 Budapest, Hungary