ICP mm saline 0 500 1000
CSF formation ml/min 0.104 0.136 0.284
RISA clearance ml/min 0.062 0.787 2.85
Inflow Outflow ml/min -0.047 0.650 2.70

intracranial pressure. This is probably due to the alteration of the permeability by the disturbance of cerebral circulation. RISA clearance was very great at 1000 mm saline of intracranial pressure. In such high intracranial pressure, cerebrospinal fluid escapes out of the dural sack at the site of needle puncture. Clearance of RISA comes to zero at -42.8 mm saline of CSF pressure. Outflow rate becomes equal to inflow rate at 38.5 mm saline. The resistance to the CSF absorption was calculated as follows:

\[ R = \frac{\text{Pressure}}{\text{Flow}} = \frac{P_0}{Kf} = \frac{P_1 - P_0}{Kf - F_1} \]

\[ P_0 = \text{CSF pressure}, \quad Kf = \text{rate of CSF formation}, \quad F_1 = \text{infusion rate}, \quad P_1 = \text{CSF pressure during infusion}. \]

The resistance to CSF absorption was constant in the range 150–1000 mm saline of CSF pressure. The value was 459 mm saline/ml/min.

C-10. Clinical Aspects of Acute Brain Swelling in the Severe Head Injury

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The purpose of this investigation is to ascertain the incidence, the extent and the pathogenesis of acute brain swelling in the clinical materials of head injury.

The simultaneous and continuous measurement of systemic arterial pressure (SAP) and intraventricular pressure (ICP) were performed on the unconscious patients suffering from acute, severe head injury. Respiratory gases and acid-base status of arterial blood and intraventricular cerebrospinal fluid (CSF) were also measured. Furthermore the roles on acute brain swelling of removal of CSF, intravenous administration of hypertonic solution, sodium bicarbonate or THAM solution and artificial hyperventilation were investigated.

In all the patients who had rapid deterioration of clinical symptoms and fatal outcome, ICP showed progressive rise and finally reached to the mean SAP level mostly within 24–48 hours after injury. At the final stage, ICP that equaled SAP varied directly with it and the increase in pulse pressure of ICP was another finding. In other word, acute brain swelling should be defined by such relation between ICP and SAP in the clinical subjects.

In the aggravation of acute brain swelling, the removal of ventricular CSF or intravenous administration of hypertonic solution such as 20% mannitol gave minor or entirely no effect on ICP. Mechanical hyperventilation by respirator was effective
in some extent, but was not effective on far advanced brain swelling. And when it
decreased ICP, there was large rebound increase of ICP on weaning the respirator off. As for the intravenous administration of sodium bicarbonate or THAM solu-
tion, the definite tendency was not disclosed in the present study. Recently we found
a distinctive effect of THAM solution in only a patient, who had shown marked aci-
dosis of CSF and elevated ICP that had gone near by diastolic SAP. Following the
injection of 0.3M THAM 200 ml on him, rapid and persistent decrease of ICP was
observed as 48 hours long as the monitoring was continued. It might be fortunately
suggested that the chemical response of cerebral vessels to CO₂ or PH would possibly
exist even after the disappearance of Bayliss effect.

Decreased bicarbonate ion concentration of CSF was of a common finding in
the patients with severe head injury. But it was proved in the same patinets that CSF
PH was maintained within normal range by lowered CSF PCO₂. On the contrary,
CSF PH of the patients suffering from acute brain swelling showed extreme fall
unexceptionally. It would be well recognized that low PH and raised PCO₂ of CSF
was the expression of not only cerebral hypoxia but also the cesession of cerebral
circulation. As mentioned in our previous report, total cessesion of cerebral circula-
tion went together with acute brain swelling. Further investigation is necessary to
conclude that the cerebral acidosis is a main cause of acute brain swelling, and not
theresult of it.