Intraventricular Pressure After Intravenous Injection of Urea

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There is a divergence of opinion concerning the so-called secondary rise of intracranial pressure or "rebound phenomenon" following the intravenous administration of urea.4 Significant alterations in intracerebral, cisternal and spinal-fluid pressures following the administration of urea have been observed in experimental animals under controlled laboratory conditions.1,5 Others have reported the measurement of cerebral intraventricular pressure in humans, but it would appear that such observations were made in instances in which there was acute progressive intracranial pathologia.2,3

It appeared, therefore, worth while to make such observations under quiescent controlled conditions in which there was no discernible abnormal intracranial process. We wish to report our observation on the effect of intravenous administration of urea upon the cerebral intraventricular pressure in human subjects under stable physiological conditions. Additional data on blood volume, venous (peripheral) pressure and concentrations of serum electrolytes were also obtained.

Materials

The patients who cooperated in these studies had had previous bilateral posterior parietal perforations 21 to 36 days preceding our observation. The clinical indications for the original placement of the openings were varied. In Case 1, a chronic subdural hematoma had been evacuated through the right posterior parietal perforation 3 weeks before. He had a subsiding bilateral papilledema but there were no other residual abnormal neurological findings.

In Case 2 the patient had a ventriculogram following head injury to exclude the possibility of an intracranial hematoma. The ventriculogram was normal. At the time of our study, the patient was alert and without complaint. Objectively, the only neurological abnormality was a residual incomplete palsy of the oculomotor nerve on the left.

In Case 3 the patient was hospitalized for 6 weeks prior to our studies, for treatment of a severe head injury. He had remained in coma. Previous arteriograms and ventriculograms had excluded the presence of an intracranial space-occupying lesion. Other than coma and a general hyperreflexia, the neurological findings were normal.

In Case 4 the patient was suspected of having a brain tumor. Normal ventriculograms were obtained 1 month prior to the studies on pressure reported here.

Methods

Values of blood-urea nitrogen and the serum electrolytes were determined prior to the administration of urea. In 3 patients, determinations of blood volume were made using the technique of tagging red blood cells with Cr51.6 The actual procedure employed in these determinations of pressure was the same for each patient studied. The patient was fixed in a position of left lateral decubitus. The head was maintained in position of normal anatomical relationship to the vertebral column. A #18 gauge Intracath tube was inserted in the antecubital vein of the right arm in preparation for measurement of venous pressure. The patient's scalp was shaved and the skin was washed with Septisol followed by a water rinse and alcohol bath. A needle for injecting air was introduced into the right lateral (uppermost)
Pressure After Injection of Urea

cerebral ventricle. Both the intravenous cannula and the cerebral intraventricular needle were connected to separate transducers of a Model 60–1300 B Sanborn twinviso continuous recording mechanism. The recording system was then calibrated. The accuracy of the measurements of the control pressure was established by observing and recording the pressures for a period of 1–2½ hours preceding the administration of urea. These recordings were continued for a period of 6 hours following administration of urea.

A 250 cc. volume of a 10 per cent Travert solution containing 90 gm. of urea (Urevert) was administered intravenously in a period of exactly 30 min.

Samples of blood were obtained at hourly intervals following the injection of urea. Determinations of blood volume, serum electrolytes and blood-urea nitrogen were carried out on these samples. The period of observation was 6 hours.

Results

Fig. 1 is a record of the measurements of cerebral intraventricular pressure following the intravenous administration of urea in these patients. In each instance, there was a progressive fall of the cerebral intraventricular pressure. The decrease in the ventricular pressure varied from 40 to 100 mm. of water from the control levels. This represented a reduction of 28–55 per cent of the level before injection of urea. The drop was maximal, between 50 to 70 min., following administration of urea. In each instance there was a subsequent increase of the intraventricular pressures, reaching the “control level” within 3 to 5 hours following administration of the urea, in each patient.

In 2 of the 4 patients, a secondary rise or “rebound pressure phenomenon” was recorded. The maximal levels of rebound observed were, in Case 1 a 20 per cent increase and in Case 4, a 40 per cent increase above the control pressures. The “secondary rise” in Case 1 was first recorded at 90 min., and in Case 4 at 135 min. after completion of the administration of urea. In both instances, this elevation persisted for the 6-hour period of observation (Fig. 1).

A significant increase in the peripheral venous pressure was obtained in 1 patient. This increased venous pressure persisted for 25 min. following completion of the administration of the urea. Fig. 2 demonstrates the values of the venous pressure associated with the actual administration of urea.

Determination of blood volume (Cr\textsuperscript{31}) showed a significant variation in 1 patient. This increase persisted throughout the 6-hour period of observation. However, the maximal increase in blood volume existed for approximately 150 min. following the com-