PERITONEAL DIALYSIS: ITS USE IN THE CORRECTION OF ALTERED BLOOD CHEMISTRY IN A BRAIN-INJURED PATIENT

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This case report is presented to indicate the effectiveness of peritoneal dialysis in the correction of severe chemical changes in the blood which have occurred in the brain-injured patient. Because of the severe azotemia and hyperkalemia being changed rapidly by this uncomplicated method, we are prompted to render this report.

CASE REPORT

MHMH #158287. L.G., a 25-year-old white male, was admitted in a comatose condition, having sustained a head injury. He had jumped from a fast-moving truck in which the airbrakes had failed.

He was decerebrate and bleeding from his right ear. His blood pressure, pulse rate, temperature, and respiratory rate were normal. His pupils were equal and reacted to light. Roentgenograms of the skull showed a linear fracture of the right parietal bone, and cervical films disclosed an old compression fracture of the body of the 3rd cervical vertebra.

A tracheotomy was done to aspirate a large amount of purulent material in the tracheobronchial pathway. A tentative diagnosis of "brain-stem injury" was made. Peripheral count of white blood cells was ~5,600 and hemoglobin was 15.3 gm. per cent. Blood urea nitrogen was 11 mg. per cent and blood CO2 combining power, and chloride, sodium and potassium of the serum were normal. Count of eosinophils was 5 per c.mm.

He was placed on urinary electrolytic collections which initially were normal. Daily gastric feedings included 2000 cc. of fluid having 40 mEq. of sodium and 65 mEq. of potassium in 24 hours.

Trephines under local anesthesia on his 8th hospital day disclosed a large clotted extradural hemorrhage in the right parieto-occipital region. There had been no change in his vital signs but his coma had persisted. On the day of operation, the blood urea nitrogen was 116 mg. per cent and blood CO2 combining power, and chloride, sodium and potassium of the serum were normal. Count of eosinophils was 5 per c.mm.

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Eight days after removal of the extradural hemorrhage, lumbar puncture was done. The fluid was under a pressure of 90 mm. of water. This same day the blood urea nitrogen was over 400 mg. per cent and the serum potassium was 9.6 mEq. per l. A tentative diagnosis of "brain-stem injury" was made. An electrocardiogram showed evidence of hyperkalemia with "tenting" of the T waves (Fig. 2). The CO2 combining power was 10 mM. per l., the serum sodium was 166 mEq. per l. and the chloride was 123 mEq. per l. His urinary output had dropped to less than 500 cc. in 24 hours. He was placed immediately on sodium bicarbonate, 2 gm. every 2 hours, in the gastric feedings and the standard formulas of feeding were stopped. He was given 20 per cent glucose in water—200 cc. every 2 hours. Peritoneal dialysis was started. Maxwell's instructions were followed—2 liters of dialysing fluid were instilled in the peritoneum, left there for 1 hour and recollected by drainage by gravity. By this means, 14 of 16 l. instilled were recovered. Heparin and Achromycin accompanied the solution which contained the following:

<table>
<thead>
<tr>
<th>Milliequivalents/liter</th>
<th>Millimoles/liter</th>
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<tbody>
<tr>
<td>Sodium 140</td>
<td>140</td>
</tr>
<tr>
<td>Chloride 101</td>
<td>101</td>
</tr>
<tr>
<td>Calcium 4</td>
<td>2</td>
</tr>
<tr>
<td>Magnesium 1.5</td>
<td>1</td>
</tr>
<tr>
<td>Lactate 45</td>
<td>45</td>
</tr>
<tr>
<td>Dextrose — (15 gm./l.)</td>
<td>83</td>
</tr>
<tr>
<td>Total 291.5</td>
<td>372</td>
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No potassium was instilled in this fluid.

His blood pressure remained low. His potassium value reverted to 6.2 mEq. per l, the blood urea nitrogen to 350 mg. per cent, the CO2 combining power to 12 mM. per l., and the urinary output was 1400 cc. in 24 hours. The dialyses successfully cleared 1932 mg. of urea nitrogen, 56 mg. of potassium and also 56 mg. of sodium. The intake was increased to 5000 cc. in 24 hours for 2 days. The patient remained in coma and his arterial pH was 7.32 two days later. The blood urea nitrogen fell to 76 mg. per cent 11 days after the peritoneal dialyses. Within 2 weeks of the dialyses and correction of the very abnormal chemical values of his serum, he awakened and started to walk and talk. He has progressed rapidly in his rehabilitation and 6 months after his injury has a border-line normal intelligence quotient. A drop foot on the left has been relieved by a brace. He has had no convulsions although the electroencephalogram shows bursts of high voltage throughout the record. His dynamometer readings are equal.

DISCUSSION

This case has been most interesting in the major biochemical abnormalities of the serum and their correction by an uncomplicated method which may be particularly useful in the treatment of azotemia in the brain-injured patient regardless of the mechanism. The azotemia may have resulted from dehydration or from specific effects of cerebral injury which may alter patterns of serum and kidney.

Our presumed diagnosis of "brain-stem coma" was proven to be faulty with surgical removal of
the clotted extradural hemorrhage and correction of the severely altered chemistry of the blood. This latter phase required 2 weeks before a noticeable change in the comatose state was elicited despite metabolic changes responding very soon after correction was instituted.

From a review of the literature on disturbances of the electrolytes of serum in head injuries, we are impressed with the paucity of reports and are planning to study the cerebrospinal fluid in these patients. Our early studies indicate that the compartment of cerebrospinal fluid may be changed even more radically than the serum as far as its pH, gaseous contents and concentrations of electrolytes are concerned.

Certain reports have indicated that the values of serum sodium and chloride after head injuries may rise. Allott thought interference with the antidiuretic excretion of hormone caused excessive loss of water and a selective increase in tubular reabsorption of sodium and chloride. Sweet questioned the possibility of an exaggerated alarm reaction of the adrenals despite normal values of urinary cortico-oxysteroids and ketosteroids. Cooper found elevated levels of serum sodium and chloride with decreased values of urinary chloride despite "adequate hydration therapy." He said, "We have had little success thus far in the treatment of this electrolyte abnormality once it is recognized. However, we have had some hint of the direction that such treatment should take." Higgins et al. reported increased values of serum sodium and chloride despite fluid intakes of 2270–2880 cc. in 24 hours. They claimed no serious depletion of water in their cases.

Welt et al. studied the role of the central nervous system in metabolism of electrolytes and water. They observed severe hypertonicity of the blood without diabetes insipidus in medical and surgical neurologic lesions; they believed that not all of these cases represented dehydration and felt that abnormal tonicity of body fluids could be found from certain cerebral lesions. They cited a case of gunshot wound of the brain in which 20 days after the injury a "peripheral vascular collapse" occurred with no flow of urine and a blood nonprotein nitrogen value of 188 mg. per cent. A balance study was started and the urinary excretion value was 17.6 mEq. per 1.

Gordon and Goldner presented two well-documented cases of hypernatremia, azotemia and acidosis after cerebral injury. In both cases there was excretion of small concentrations of sodium but large amounts of potassium; despite this the value of serum potassium rose. After rehydra-

**Fig. 1.** Development of severe azotemia and relief by peritoneal dialysis. Fluid balance studied by intake above line and output of urine below. On days of no apparent electrolytic output, observations were not made. Sodium values are recorded in clear bars for intake and black bars for output. Potassium intake values are stippled while the output values are represented by clear bars.