several days, and it was interesting to note that venous thromboses did not occur at the sites of infusion.

Pain along the course of a vein into which potassium is being infused has been reported (Semler, 1925; Pudenz *et al.*, 1938; Nadler *et al.*, 1950). The majority of instances occurred after the use of concentrated solutions of potas-

sodium citrate, which removes ionized calcium and increases the ratio of potassium to calcium ions acting on the nerve fibre (Katz, 1936; Brink and Bronk, 1937; Lehmann, 1937). Presumably the non-medullated nerve fibrils which subserve pain respond in a similar manner.

In our case the blood being transfused contained 2 g. (1%) of disodium citrate as an anticoagulant, as well as 1.5 g. (0.278%) of added potassium citrate. It is probable, therefore, that the lowering of the pain threshold was due to potentiation of the potassium effect by the citrate.

Summary

Acute renal failure, with complete recovery, is reported in a haemophiliac after massive haemorrhage into the abdominal wall.

Treatment included transfusion with large volumes of fresh plasma and blood.

Potassium citrate was given intravenously for a time, and was found to produce local pain when a threshold concentration was exceeded.

We wish to thank Dr. C. B. V. Walker, of the Regional Blood Transfusion Service, Cambridge, for his unfailing co-operation.

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SALT RETENTION AND URAEMIA IN BRAIN INJURY

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Cerebral disorders of various kinds may be followed by biochemical disturbance in which abnormally high levels of sodium and chloride in the blood are associated with extremely low levels of these ions in the urine. The term "salt-hoarding syndrome" has been suggested for this condition (de Wardener, 1956), and the term seems to be a satisfactory one so long as it is appreciated that uraemia appears to be an invariable concomitant of the hypernatraemia and hyperchlor-
aemia.

This syndrome was first reported by Allott (1939), who observed it in association with cerebral haemorrhage, adenoma of the choroid plexus, and tuberos sclerositis. It has since been observed following a large variety of cerebral lesions. These include brain damage following head injuries (Higgins *et al.*, 1954), bulbar poliomyelitis (Allott, 1957), bilateral ligation of the anterior cerebral arteries (MacCarty and Cooper, 1951), operations on the pituitary (Cooper and MacCarty, 1951), leucotomy (McLardy, 1950), subarachnoid

Chart showing serum electrolyte changes, haemoglobin, blood urea, and volume of urine during patient's stay in hospital.

sium chloride (2–10%), given within a matter of minutes. A few were observed after a 1.14% solution of potassium chloride (155 mEq of potassium per litre) had been given at the rate of 3–5 ml. a minute. However, during the treatment of diabetic ketosis, once diuresis has started, solutions containing up to 30 mEq of potassium per litre are frequently given at a rate of 3 ml. a minute without causing pain. In our case pain was caused by an infusion containing practically the same concentration of potassium (25 mEq/l. as potassium citrate plus 5–7 mEq/l. in the plasma of the blood), given at the same rate, suggesting that a low threshold of excitability was present in the pain receptors in the vein wall.

It is known that spontaneous activity may be induced experimentally in nerve fibres by exposing them to potassium, which lowers the threshold of excitability (Brown and MacIntosh 1939). The same effect may be produced by

haemorrhage, subdural haematoma, brain tumour, meningitis, Wernicke's encephalopathy, "encephalomalacia" (Schoolman *et al.*, 1955), right frontal lobectomy, removal of a glioma invading the third ventricle (Sweet *et al.*, 1948), and anoxia in premature infants (Natelson and Alexander, 1955). The picture of hypernatraemia, hyperchloraemia, and uraemia has also been seen in sulphonamide toxicity in which the drug caused both kidney and brain damage (Luetscher and Blackman, 1943; Goodale and Kinney, 1949).

The phenomenon of salt-hoarding associated with lesions of the brain is not a rare one: 63 such cases were seen in one hospital over a period of four years (Schoolman *et al.*, 1955), and others claim to have seen the condition frequently (Natelson and Alexander, 1955). Higgins *et al.* (1954) recently reviewed the findings in 76 consecutive patients with closed head injuries who had remained unconscious for more than twelve hours, and found that 12% developed hyperelectrolytaemia.

We report here a further case of this syndrome in a patient who had sustained a head injury. The case appears to have been a typical example of the salt-hoarding syndrome. The patient's blood volume was measured when there was marked hypernatraemia and hyperchloraemia. It is the first case of this kind, so far as we are aware, in which this investigation was performed. The result has a bearing on the problem of whether dehydration or "desiccation" plays an essential part in the production of the hyperelectrolytaemia.

Case Report

An African man aged about 30 was transferred to Coronation Hospital on March 9, 1957, from a country hospital where he had been in a coma for three weeks. He had been found lying in the street, apparently having been assaulted. When seen by us he was semi-comatose, responding to painful stimuli. He was emaciated, weight 7 st. 3 lb. (45.8 kg.), height 5 ft. 6 in. (167.5 cm.). There were numerous large blebs filled with clear fluid distributed on the trunk and extremities. The tongue was somewhat dry, and clinically there appeared to be slight dehydration. The temperature was 97° F. (36.1° C.) the pulse rate was 120 a minute, and the blood pressure 90/60. The tendon reflexes appeared to be slightly brisker on the left, but otherwise there were no localizing neurological signs and all limbs were seen to be capable of movement.

The initial investigations showed the following results. Urine: specific gravity 1018, sugar and protein absent, microscopical examination showed no abnormality. Blood: leucocytes 13,000/c.mm., haemoglobin 13 g./100 ml. Electrocardiogram normal. Radiological examination of the chest showed no abnormality; radiological examination of the skull showed a linear fracture of the left parietal bone involving the base of the sphenoid. An air encephalogram showed nothing abnormal. Serological tests for syphilis were negative.

To combat the dehydration he was given 3 litres of fluid (1 litre of 5% dextrose in 0.9% sodium chloride, 1 litre of 0.9% sodium chloride, and 1 litre of 5% dextrose in water) intravenously in the first 15 hours. On the morning after admission blood analyses gave the following results: urea, 380 mg./100 ml.; chloride, 126 mEq/l.; sodium, 156 mEq/l.; potassium, 6.1 mEq/l.; CO₂, 13.5 mEq/l. The findings in the blood and urine relevant to the state of the patient's electrolytes are shown in Tables I and II. The blood urea was persistently elevated, rising still higher shortly before the patient's death on the tenth hospital day. The serum sodium and chloride, however, began to drop on the sixth day. There was no corresponding increase in the urinary levels of these electrolytes, which remained at

TABLE I.—Blood Urea (mg./100 ml.), Serum Electrolytes (mEq/l.), and Haematocrit

Date	Urea	Sodium	Chloride	Potassium	Bicarbonate	Haematocrit
March 10	380	156	126	6.1	13.5	
" 11	372	165	133	4.7	18	43 %
" 12	360	165	141	4.4	16	43 %
" 13	328	162	134	4.2	21	
" 14	332	156	131	4.2	15	
" 15	345	151	113	4.6	15	41 %
" 16	400	141	120	4.0	16	42 %
" 17	444	139	108	5.4	16.8	
" 18	536	148	107	3.6	18	
" 19	492	142	103	4.1	18	

TABLE II.—Urinary Findings: 24-hour Output (ml.); Specific Gravity; Urea (mg./100 ml.); Sodium, Chloride, Potassium (g./l.)

Date	Output	S.G.	Urea	Sodium	Chloride	Potassium	Remarks
March 10	1,600	1015					
" 11	1,200	1015					
" 12	1,700	1014					
" 13	2,000	1014	2,080	0.68	1.62		
" 14	1,300	1015	1,680	0.74	1.63		
" 15	1,000	1012	1,920	0.63	0.382		
" 16	450	1012	1,220	0.88	0.32	0.15	Urine contained pus
" 17	400	1012	1,160	0.96	0.275	0.15	
" 18	500	1010	1,180	1.0	0.278	0.15	
" 19	250	1012	980	1.4	0.32	0.14	

extremely low levels throughout. The serum potassium, apart from a high initial level, remained within normal limits until two days before the patient's death, when the level fell below normal. There was no change in the urinary excretion of potassium, which remained scanty. It may be seen from the tables that in the first six days the picture was different from that in the last four days. In the first period there was hyperelectrolytaemia and good urinary output, whereas in the last few days there were normal levels of serum sodium and chloride, accompanied by oliguria.

On the sixth day the blood volume was measured, using ⁵¹Cr labelling of the patient's red cells. The total blood volume was 3,200 ml.—that is, 70 ml./kg. body weight—and the plasma volume calculated from the venous haematocrit was 2,100 ml. On the same day the plasma creatinine was 4.1 mg./100 ml. and endogenous creatinine clearance showed a value of only 11.4 ml. a minute. On the day before the patient's death the serum calcium was found to be 3.3 mEq/l., and the output of calcium in the urine was 35 mg.—that is, 14 mg./100 ml.

Throughout his stay the patient received fluids by vein or intragastric drip. Apart from the sodium chloride solution given on admission, and 2 litres of 6-molar lactate given on the sixth day to combat acidosis, these fluids were salt-free. The amount of fluid given daily was about 500 ml. in excess of the daily urinary output.

The patient's clinical condition deteriorated slowly. The systolic blood pressure remained at or just above 100 mm. of mercury. On the fourth hospital day a trace of protein appeared in the urine, and microscopical examination showed the presence of a few leucocytes and red cells, but no casts. It was thought that the indwelling catheter had caused a cystitis. Next day there was frank pyuria and haematuria and the patient became febrile. The urinary infection failed to respond to antibiotics. The patient died on the tenth hospital day.

Necropsy Findings

Necropsy revealed slight organizing bruising in the left temporal muscle, a fine crack fracture in the left temporal region extending into the petrous temporal bone, slight haemorrhage into the sphenoidal sinus, marked generalized flattening of the cerebral convolutions, slight prominence of both uncus, more marked on the right side. In the right temporal lobe on its lateral aspect were several areas of softening of the cortex over an area approximately 1½ by 1 in. (3.8 by 2.5 cm.). The surrounding brain substance

was very swollen and oedematous. There were occasional small areas of subarachnoid haemorrhage scattered over the brain surface. The pituitary fossa and pituitary gland showed no macroscopic evidence of damage. The lungs were congested and slightly oedematous and showed early signs of bronchopneumonia in both basal lobes.

Kidneys.—These appeared somewhat pale. The cut surface showed both the cortex and the medulla to be paler than normal. Microscopy showed some scattered areas of interstitial oedema. Dilatation of several tubules was seen, affecting mainly the distal tubules. There were scattered areas of interstitial infiltration with chronic inflammatory cells, and one or two areas of peri-glomerular fibrosis. An occasional vessel showed mild arteriosclerotic changes. There were a few pale hyaline casts. All the histological changes were of a minor degree.

Other Findings.—The bladder mucosa was very haemorrhagic. The lungs showed early changes of bronchopneumonia in both bases. The suprarenals were macroscopically normal.

Discussion

It is generally agreed that in this type of case the brain damage is causally related to the metabolic changes. There has been no general agreement, however, on the possible mechanism of the biochemical derangement.

Kidney

The role of the kidney itself has received scant attention. In none of the published cases was renal function thoroughly studied, yet in all those cases which we have traced in the literature the blood urea, where reported, was raised and usually markedly so. Schoolman *et al.* (1955) acknowledged the possibility of primary renal disturbance, but suggested that this disturbance could be the diuretic phase of "lower nephron nephrosis." They stated that in two of their cases of hypernatraemia "lower nephron nephrosis" existed. However, Allott (1957) has pointed out that the urinary findings in "lower nephron nephrosis"—namely, a low specific gravity, usually a high concentration of electrolytes, and a low concentration of urea—are in complete contrast with the findings in the salt-hoarding syndrome. Higgins *et al.* (1954) considered that the uraemia which may follow brain injury is of renal origin despite the absence of morphological changes in the kidney at necropsy. They have pointed out that the proteinuria and renal glycosuria which may follow cerebral injury afford evidence of renal dysfunction secondary to brain damage. Allott (1957) has noted an inverse relationship between creatinine clearance and hyperelectrolytaemia, and in our case the creatinine clearance was only 11.4 ml. a minute.

In most of the reported cases which have come to necropsy the kidneys were normal on histological examination, although some showed slight changes similar to those in the present case. It is not possible to state the significance of the changes seen in the kidneys of our patient. Even in acute renal disease with severe functional derangement, histological changes may appear to be almost negligible (Brun and Munck, 1957). That the kidney may in fact be damaged secondarily to brain dysfunction is suggested by the work of Cort (1947), who elicited renal cortical ischaemia and complete anuria following stimulation of the posterior orbital surface (area 13) in the cat. Higgins *et al.* (1954) reported that a constant finding in the brains of their cases with hypernatraemia and hyperchloraemia was damage to the undersurface of the frontal lobes, and McLardy (1950) placed the lesion necessary for the development of uraemia following leucotomy in area 47 in the orbital cortex. He advanced reasons for suggesting that in man this area may correspond to area 13 in the cat.

A critical evaluation of renal function in the salt-hoarding syndrome is obviously called for and might possibly provide a clue to the intermediate steps in the mechanism by which brain dysfunction could provoke renal dysfunction.

Diabetes Insipidus

Following the work of Engstrom and Liebman (1953) several authors have suggested that the hyperelectrolytaemia following cerebral disease may be due to the development of acute diabetes insipidus in these cases. Engstrom and Liebman studied the effects of "pitressin" (vasopressin) and cortisone in a patient with anterior and posterior pituitary insufficiency. When pitressin was withheld the patient developed hyperchloraemia with low levels of sodium and chloride in the urine. The effect was exaggerated when pitressin was withheld and cortisone administered. Two patients in whom salt-retention had appeared following the removal of a craniopharyngioma had earlier been described by Peters (1948) and Welt *et al.* (1952) respectively. The mechanism was thought to be acute diabetes insipidus leading to extreme dehydration in patients who were too mentally confused to take fluids. Allott (1957) has suggested the following course of events in the salt-hoarding syndrome: an appropriately located injury to the brain leads to acute diabetes insipidus resulting in the flushing out of excessive amounts of electrolytes; this in turn stimulates the secretion of aldosterone, which is responsible for the salt-retention. In Allott's case the excretion of aldosterone on the day it was measured was 4.5 $\mu\text{g.}$, the upper limit of normal in the particular laboratory being 2 $\mu\text{g.}$ daily.

Others have suggested that the course of events is similar to that which existed in the case of Peters (1948)—that is, diabetes insipidus leading to severe dehydration in unconscious patients. There are, however, certain objections to these hypotheses. In the patient studied by Engstrom and Liebman (1953) the specific gravity of the urine did not rise above 1007, and in the patient of Welt *et al.* (1952) with the craniopharyngioma the specific gravity of the urine was 1005. In our case the specific gravity of the urine was 1018 on admission. Anthonisen *et al.* (1954) reported a typical case of this condition in which the urinary specific gravity was 1022, and Sweet *et al.* (1948) found the specific gravity to be 1030 in a case of hyperelectrolytaemia. In a case of Welt *et al.* (1952) the specific gravity of the urine was 1022 at the time when the serum chloride was markedly elevated. They considered that this makes diabetes insipidus quite unlikely. The other objection, which is related to the question of dehydration in these patients, is discussed below.

It is of interest that Engstrom and Liebman (1953) and Welt *et al.* (1952), whose work forms part of the basis for the hypothesis of diabetes insipidus in these cases, considered that the mechanisms of hyperelectrolytaemia following on intracranial lesions may differ with different intracranial lesions. The former two authors, while stating that there is no direct evidence to support the suggestion, thought that direct central-nervous control of renal function is possible.

We also feel that, although salt-hoarding may undoubtedly occur in diabetes insipidus, this mechanism does not operate in the type of case under discussion.

Dehydration

It is most unlikely that simple dehydration could account for the phenomenon of intense salt conservation, especially in the presence of a normal or high urinary output. Some authors have postulated a more complex type of dehydration. Anthonisen *et al.* (1954) suggested that derangement of the "osmoregulatory centre" causes dehydration and that this in turn stimulates the "volume receptor" to initiate mechanisms responsible for salt retention. A similar disturbance of cerebral mechanisms controlling electrolyte and water balance was postulated by Cooper and MacCarty (1951), although their patient was not clinically dehydrated.

There have been a number of conflicting views on whether or not dehydration, from whatever cause, is an essential part of the mechanisms responsible for salt hoarding and uraemia. It has been suggested that in these cases as much as 40–50% of body water may be lost (Schoolman *et al.*, 1955), while others have stressed that their patients were not clinically dehydrated. In our patient the daily excretion of between

1 and 2 litres of urine with a specific gravity of 1012–1018 during the period of hyperelectrolytaemia, did not suggest dehydration. Study of the blood volume showed 70 ml. of blood per kg. of body weight. This figure would appear to exclude severe dehydration.

Other Features

Two other features deserve mention. In the present case, as in other cases, the electrolytes in the blood returned to normal levels, although the urinary content of sodium chloride remained very low. This observation suggests that in addition to salt retention there is an internal shift of electrolytes in this condition. Allott (1957) also inferred that a redistribution within the body is the likely explanation.

The other point worthy of note in our case was the presence of numerous bullae on the body surface. McLardy (1950) considered these bullae to be evidence of a trophic change consequent on cerebral damage. Our patient thus exhibited two distinct manifestations of his brain injury—the trophic lesions and the biochemical changes. In none of McLardy's cases did the lesions appear within two months of the brain injury. In only one case did both uraemia and the trophic changes coexist, and this was in a patient with bilateral involvement of the orbital cortex.

Conclusion

In attempting to discover a mechanism responsible for hyperelectrolytaemia provoked by cerebral dysfunction, it would seem advisable to bear in mind Selkurt's (1954) warning that "in view of the multiplicity of factors concerned with water and electrolyte balance that may be disturbed under the above circumstances, attempts to draw discrete conclusions from clinical observations are extremely hazardous." However, this type of case is not rare, and a planned approach, directed particularly at elucidating renal function, should result in progress in the understanding of this most interesting problem.

Summary

A case of hyperelectrolytaemia following a brain injury is described. Uraemia appears to be an integral part of the biochemical upset.

Evidence is produced that severe dehydration is not responsible for this disturbance, nor is diabetes insipidus thought to be a likely causative factor.

It is pointed out that renal function has not been sufficiently studied to enable an assessment to be made of the role of the kidneys in the syndrome.

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APPENDICITIS AND ACUTE TERMINAL ILEITIS DURING AN OUTBREAK OF GASTRO-ENTERITIS AMONG BRITISH TROOPS IN PORT SAID

BY

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During the period of occupation of Port Said from November 5 to December 18, 1956, of the 919 British troops who were admitted to medical units 365 had acute gastro-enteritis. Of the 29 patients admitted with suspected acute appendicitis 14 were subjected to laparotomy: 10 of these had acute suppurative appendicitis and three had acute terminal ileitis. The operations were in every case performed by me. Clinical details of the cases of terminal ileitis are presented.

All patients were young men living under similar conditions, the majority in vacated buildings in Port Said. In view of the damage to water mains and sewage, tap-water was unsafe for drinking, and all water supplied to troops was sterilized, either in bulk or by individual sterilizing tablets (halazone). During the early weeks of the occupation tinned "compo" rations were consumed.

The incidence of gastro-enteritis was higher in units where living conditions and hygiene were below the general standard, flies being particularly prevalent in such areas. In ships anchored in the harbour, where there were few flies, gastro-enteritis was uncommon.

Clinical Description

There appeared to be two different groups of patients with gastro-enteritis. *Group 1.*—A small group of patients presented with the typical picture of bacillary dysentery and had profuse diarrhoea with blood and mucus in the stools; most of these cases occurred in one unit in a forward position. *Group 2.*—The majority of cases occurred among troops stationed in Port Said itself. These presented with high sustained fever, frontal headache, and occasionally neck rigidity. This was followed by severe abdominal colic and profuse diarrhoea 12 to 24 hours later. Few of these cases had blood or mucus in their stools. Recovery usually occurred after four or five days' illness, and relapses were rare. Skin rashes were not observed, nor did rheumatic symptoms occur. Dengue and sandfly fever were excluded on clinical grounds.

Treatment.—All cases received sulphonamides in some form or other, so that it is impossible to predict whether they would have recovered equally quickly without treatment. It was the general impression, however, that sulphonamides had little effect on the course of the disease of the patients in group 2.

Pathology.—Unfortunately only very limited pathological facilities were available. No cultures or agglutination reactions could be performed. Bacillary cellular exudate was found in the stools of patients in group 1 only. Moderate leucocytosis was the rule, with a preponderance of neutrophil granulocytes. Blood slides taken to exclude malaria were consistently negative.

Incidence of Appendicitis

About one-third of the patients in group 2 had right-sided abdominal pain and tenderness, simulating appendicular pain, at some time during the course of their illness. Apart from the frequency of this appendicular form of gastro-enteritis occurring in group 2, the incidence of true appendicitis was high during this epidemic.