Summary

The clinical histories, physical findings, and management of 47 infants and children ill as the result of low-humidity heat-wave conditions are presented. Results of an inquiry into the factors of disease and home management which may have caused the special susceptibility of these cases are discussed.

It is suggested that failure of mothers to offer extra fluid was the most important factor, but that pre-existing feeding difficulties were important, and in most of the older children were due to severe mental defect or chronic disease. Loss of water in excess of replacement resulted in acute cellular water-depletion with unconsciousness, and hyperpyrexia occurred in many.

Cooling and urgent intravenous infusion constituted the treatment, and during recovery fits were common, difficult to control, and dangerous; they were not related to the type of fluid infused. In all, 12 patients died of heat illness, six of whom were dead on arrival. Post-mortem findings in these cases are briefly recorded.

Four children from country areas who had been exposed to heat for a longer period showed salt depletion, and responded to intravenous administration of saline.

It is hoped later to report a follow-up of the survivors.

At the time of this investigation we were all members of the Clinical Research Unit, and we are grateful to the Director (Dr. H. E. Williams) for allowing us the opportunity to carry it out and for helpful criticisms of the manuscript. We thank Dr. Charlotte Anderson, who was acting director of the unit during the period of the heat wave, for her useful advice. The medical director of the hospital then was Dr. V. L. Collins and his encouragement was appreciated, as was the co-operation of the members of the staff in allowing access to patients under their care. The Department of Public Health and the pathologist to the Victorian Government gave access to valuable information, as did the Commonwealth Statistician.

REFERENCES

- Cooke, R. E., Pratt, E. L., and Darrow, D. C. (1949-50). *Yale J. Biol. Med.*, **22**, 227.
 Darrow, D. C., Cooke, R. E., and Segar, W. E. (1954). *Pediatrics*, **14**, 602.
 Finberg, L. (1959). *Ibid.*, **23**, 40.
 Haymaker, W., Teabeaut, J. R., Krainer, L., and Schickele, Elizabeth (1955). *Acta neuroveg. (Wien)*, **11**, 142.
 Kuno, Yas (1956). *Human Perspiration*, No. 285 in American Lecture Series. Blackwell, Oxford. Thomas, Illinois.
 Medical Research Council (1958). *Brit. med. J.*, **1**, 1533.

"Now that we are happily installed in our new hospital we have no further use for our property in the Old City of Jerusalem. Watson and Strathearn Houses served us well after we were compelled to leave the hospital buildings in which we worked from 1883 to 1948, now in Israel. It is true that these two traditional Arab houses were extremely unsatisfactory and difficult to run as a hospital; nevertheless they provided a means whereby the humanitarian activities of the Order [of the Hospital of St. John of Jerusalem] in Jordan could be maintained without intermission. The success with which it did so can be realized when it is remembered that 1,700,000 patients passed through their doors in the few years of their existence. We are glad that their usefulness is not to cease, for they have been taken over by the Lutherans for the extension of the school which they run in an adjoining building; in this way we have been of service to the Johanniterorden in Germany with which we are historically and sentimentally related." (*Ophthalmic Hospital, Jerusalem. Report of the Committee for 1961.*)

DIETARY MANAGEMENT OF ACUTE OLIGURIC RENAL FAILURE

BY

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Modern treatment of acute oliguric renal failure is designed to maintain the volume and composition of the body fluids as near normal as possible, and to restrain tissue breakdown to a minimum. This entails careful control of fluid, electrolyte, and calorie intakes, with restriction of protein. The use of extracorporeal haemodialysis has improved the results obtained by this regime, but deaths still occur, particularly in the group with a surgical cause. We have been concerned with the difficulty of administering sufficient calories to these acutely ill patients, and by the extreme degree of wasting which may be encountered during the long periods of survival now possible with repeated haemodialysis. The results obtained in some of our earlier cases, when patients were given carbohydrate as their only source of calories, were disappointing, and studies were therefore undertaken to improve the composition and nature of the diet, with considerably increased calorie intakes and some protein supplements.

Twelve patients, admitted to the artificial kidney unit at the General Hospital, Birmingham, with acute oliguric renal failure from a variety of causes, have been treated with oral feeding of specially prepared diets. These were designed to supply 1,250-1,400 calories a day, and contained from 30 to 40 g. of first-class protein, in addition to carbohydrate and fat (see Appendix). Foods with relatively low sodium and potassium content were chosen to prevent undesirably high serum concentrations of these ions; maximum daily intake of sodium was 0.6 g. (26 mEq) and potassium 1.2 g. (30 mEq).

The diet was started in each patient after an initial period of three to eight days after admission, during which time electrolyte or fluid deficiencies were corrected and control data on conventional dietary treatment could be collected. Daily estimations of serum sodium, potassium, chloride, and urea were carried out, and the daily rise in serum-urea level on the prepared diets was used as an index for comparison with the similar rise in the pre-dietary period. All the patients received anabolic steroids (norethandrolone) 50 mg. daily by intramuscular injection.

Haemodialysis with a Travenol "twin coil" kidney was carried out in most of these patients when the serum-urea level reached approximately 400 mg./100 ml., and this was often necessary during the first

few days after admission or transfer from another hospital.

Results

There were eight survivors from the 12 cases studied. In all 12 cases there was a reduction in the mean daily rise of urea, which varied from 10% to over 50% of the control period. The serum-potassium level did not rise above 7 mEq/l. in any patient, nor was it found necessary to dialyse these patients more frequently for any other reason. The results obtained are summarized in the Table. The diets prepared were palatable, and even these acutely ill patients were able to take them satisfactorily, with considerable improvement in morale. A typical case from the series is described.

Summary of Results

Case No.	Sex	Weight (kg.)	Cause	Mean Daily Rise of Urea				Result
				Before Diet		During Diet		
				mg./100 ml./day	mg./100 ml./kg./day	mg./100 ml./day	mg./100 ml./kg./day	
1	M	70.5	Crush injury	81	1.15	63.7	0.9	Lived
2	M	53	Bile peritonitis	84	1.6	35	0.66	Died
3	M	66	Renal amyloid	20	0.31	11.9	0.18	„
4	F	55	Ruptured uterus	88	1.6	52	0.95	Lived
5	M	98	Stone in ureter	35	0.35	28	0.28	„
6	F	65	Abortion	51	0.83	24	0.4	„
7	F	52	Accidental haemorrhage	30	0.6	27	0.54	„
8	M	74	Mismatched transfusion	31	0.42	20	0.27	„
9	M	86	Trauma	68	0.79	59	0.68	„
10	M	66	Bile leak	30	0.45	20	0.30	Died
11	M	64	Incarcerated hernia	58.4	0.9	32.4	0.5	Lived
12	M	71	Acute nephritis	56.8	0.75	25.5	0.35	Died

Case 2.—A 50-year-old man underwent cholecystectomy for cholelithiasis. On the third post-operative day he developed profound hypotension because of leakage from the stump of the cystic duct. Acute oliguric renal failure ensued and he was transferred to this unit. Urgent haemodialysis was required on admission for hyperkalaemia (serum K, 7.8 mEq/l.), when the serum urea was 450 mg./100 ml. (see Chart). From the onset of renal failure he was treated by oral and intravenous carbohydrate only, and for the five days after dialysis the same regime was employed. By this time a second dialysis was necessary as the serum urea was again above 400 mg./100 ml. His general condition was then poor, with a severe biliary peritonitis and gross wasting. He became increasingly reluctant to take any carbohydrate (lactose 20%) by mouth. Change was then made to the high-calorie protein-containing diet. There was an improvement in morale, related to the return to a more normal diet, and the wasting process was checked. The rate of formation of urea diminished (mean daily rise fell from 1.6 to 0.66 mg./kg./day), a reduction in the rate of protein breakdown of over 50%. This change persisted for the remainder of the course of the illness, until his death on the 21st day from a massive secondary haemorrhage from the splenic artery.

Discussion

Bull, Joekes, and Lowe (1949) and Bull (1955) emphasize the importance to the oliguric patient of a high-calorie intake. The advantages attributed to the high-fat, high-carbohydrate diet would appear to be related to the non-specific effect of the calories supplied. In practice, however, the provision of adequate calories presents a problem, since strong solutions of sugar given orally often induce thirst or nausea, and intravenous

solutions are limited by considerations of maximum volumes, so that only some 800 calories can be supplied in a 24-hour period. The original peanut oil emulsion necessitated intragastric feeding, which for several weeks is undesirable. The problem becomes more acute in those cases of oliguric renal failure resulting from trauma or surgical misadventure. These patients are severely ill, with the serum urea rising rapidly from factors such as haematomata, tissue necrosis, and sepsis. Experience has now shown that they may be kept alive and in a relatively normal "biochemical" state for long periods by the use of the artificial kidney. In those surviving for two weeks or more the degree of wasting may become extreme, as during this period the effects of protein deprivation may become obvious and the plasma protein levels fall. Patients succumbing after prolonged illness not infrequently show a terminal rise in the rate of formation of urea.

It thus appears logical to provide both adequate calories and a small quantity of dietary protein in addition, in an attempt to minimize endogenous catabolism of protein. By provision of essential amino-acids vital resynthesis may continue without recourse to excessive breakdown of muscle tissue. Provided sufficient calories are available from sources of carbohydrate or fat at the same time, it appears likely that the provision of such protein will be utilized for resynthesis, and not merely used for production of energy, with liberation of increased urea (Edgren *et al.*, 1960). The inclusion of a small quantity of first-class protein into the diet allows the preparation of a more interesting daily menu, which an ill patient is better able to tolerate. Because of this, greater daily calorie intakes were consistently achieved in our series. By comparison, in a group of nine patients treated earlier by us on exclusively carbohydrate diets (20% lactose), the highest

Biochemical data for Case 2, showing decreased rate of rise of serum urea per day and steady weight while on higher protein and calorie diets.

intake in 24 hours was 900 calories, and the majority of intakes were much lower than this. The mean daily rise of serum urea in this group remained unchanged throughout the duration of their illness, contrasting with the reductions obtained in the 12 patients studied while receiving the prepared diets.

It is possible that the improvement could be due merely to the provision of a higher intake of calories, but, nevertheless, it is apparent that the inclusion of some protein into the diet had no adverse effect, nor did it increase overall nitrogen catabolism. We believe that provision of exogenous protein may well be a vital factor in the survival of those cases of acute renal failure of surgical aetiology, and of those cases in which repeated haemodialysis may be required before recovery of renal function occurs.

Appendix

	Protein g.	Sodium mg.	Potassium mg.	Calories
½ pt. (300 ml.) milk daily ..	9.0	144.0	460.0	190
Breakfast:				
½ oz. (7.5 g.) oatmeal ..	0.85	2.4	26.0	28
1,, (30,,) lactose ..				112
1,, (30,,) cream ..	0.4	7.6	22.0	130
100 ml. water ..				
Dinner:				
1 oz. (30 g.) white fish ..	5.1	34.0	79.0	26
½,, (15,,) butter ..		30.0	2.0	112
½,, (15,,) potatoes ..	0.25	6.0	47.0	10
1,, (30,,) vegetables ..		6.4	31.0	6
Tea:				
1 oz. (30 g.) bread ..	2.2	147.0	30.0	69
½,, (7.5,,) butter ..		15.0	1.0	56
½,, (15,,) tomato ..	0.1	0.4	41.0	2
Supper:				
½ oz. (15 g.) chicken ..	3.7	13.9	54.0	26
3,, (90 ml.) milk ..	2.7	42.6	138.0	57
50 ml. water ..				
½ oz. (7.5 g.) cornflour ..		3.6	4.2	25
½,, (7.5,,) butter ..		15.0	1.0	56
½,, (7.5,,) cream ..	0.1	1.9	5.5	32
½,, (7.5,,) "casilan" ..	6.3			24
3 oz. (90 ml.) milk ..	2.7	42.6	138.0	57
1 egg ..	6.8	74.8	78.0	92
1 oz. (30 g.) cream ..	0.4	7.6	22.0	130
1,, (30,,) lactose ..				112
(Total liquids, 630 ml.) ..	40.6	594.8	1,179.7	1,352

Summary

Twelve patients with acute oliguric renal failure have been treated with specially prepared diets providing essential calorie requirements and varying amounts of first-class protein.

The advantages of this regime are seen in the reduction of the daily rise in serum urea in renal failure, with decreased body protein catabolism resulting from the provision of essential amino-acids for protein synthesis. The survival of patients with prolonged oliguria and frequent haemodialysis may depend upon adequate protein-calorie replacement, especially in those with burns, trauma, or surgical complications.

We wish to acknowledge the generous assistance of Mr. G. M. Widdowson for the repeated biochemical estimations and the continuous help of the sisters and nursing staff of the General Hospital in the care of these patients. We also acknowledge a full-time grant to L.J.L. from the Endowment Fund, United Birmingham Hospitals.

REFERENCES

- Bull, G. M. (1955). *Lancet*, **1**, 777.
 — Jockes, A. M., and Lowe, K. G. (1949). *Ibid.*, **2**, 229.
 Edgren, B., Schuberth, O., and Wretling, A. (1960). *Wien. klin. Wschr.*, **72**, 365.

TOTAL EXCHANGEABLE SODIUM IN DEPRESSIVE ILLNESS

BY

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Recent reports have suggested that there are changes in sodium metabolism during affective illnesses. Coppen (1960) reported a decrease in the rate of entry of ^{24}Na from blood into C.S.F. in a group of 31 depressed patients; this returned to normal with clinical recovery. Gibbons (1960) estimated the total exchangeable sodium with ^{24}Na on two occasions in 24 patients suffering from a depressive illness and found a mean decrease of exchangeable sodium of about 10% after recovery. Russell (1960) studied overall sodium metabolism in depressive illness by means of extremely careful sodium-balance measurements and failed to find any evidence of sodium retention during the depressive phase. Less carefully controlled studies, not employing true balance techniques, have given variable findings during affective illnesses. Schottstaedt, Grace, and Wolff (1956) reported sodium and water retention in normal subjects during periods of transient depression, and the converse was recorded by Ström-Olsen and Weil-Malherbe (1958) in mania. Crammer (1957) recorded large gains in weight associated with water and sodium retention in cyclical psychosis. Trolle (1945), however, could find no consistent change in the pattern of sodium and water excretion in patients recovering from a depressive illness.

These contradictory reports have left the position of sodium metabolism in affective disorders uncertain, and it was decided to initiate further investigations into this problem, starting with a study of total exchangeable sodium.

The measurement of total exchangeable sodium with the long-life isotope ^{22}Na was used to pursue this problem further. This method combined the advantages of a sodium-balance technique in enabling day-to-day variations of sodium balance to be estimated together with the ease of administration and accuracy of an isotope-dilution method. In addition, even though depressed patients are frequently disturbed or agitated, the technique employed allowed daily estimations of total exchangeable sodium to be made while requiring the minimum co-operation from the patient.

Material and Methods

Serial estimations of total exchangeable sodium were carried out on 14 recently admitted male and female patients. The diagnosis of depression was made on a history of severe and unremitting depression associated with a varying proportion of accompanying symptoms such as guilt, delusions of unworthiness, retardation, suicidal ideas, sleep disturbance, poor appetite, etc. Patients suffering from concomitant physical disease, malnutrition, or dehydration were excluded, and no patient who had taken tranquillizing drugs or who had