when its entry, concentration, and source were known and to analyse some of the factors responsible for this spread.

The findings reported here show that contaminated textiles, either in the form of woollen or cotton blankets or of cotton counterpanes, disseminate organisms in detectable numbers to almost all beds in a ward approximately three hours after their introduction (Table II). The extent and intensity of the contamination increases with time irrespective of the nature of the donor textile (Table V). In general, cotton blankets appeared to be more effective in dispersing the marker organism than woollen ones (Table V).

The most interesting finding which emerged in this study was the unexpected effect on ward contamination when contaminated textiles were covered with laundry-clean counterpanes. It was consistently found that covering a contaminated blanket with a counterpane greatly increased the dispersion of the marker organism throughout the ward (Tables V and VI, Figs. 3 and 4). Indeed, one contaminated blanket covered by a clean counterpane was a more productive source of organisms than two contaminated blankets not covered by counterpanes (Table VII). While these observations were made with counterpanes in contact with a contaminated textile surface, it is not unlikely that any clean textile cover will provide the necessary conditions for the release of air-borne organisms from a contaminated textile.

An essential prerequisite for demonstrating the counterpane effect is the movement of the counterpane on the contaminated blanket. When, for example, the movement between the counterpane and the blanket is prevented by pinning the two together the air-borne contamination will be greatly reduced (Fig. 5). Thus the release of organisms from the textile surface is probably due to friction. Their dispersion from the bed will be due to air currents or agitation.

The findings suggest that the use of counterpane covers in wards might be discouraged and that their removal would lower the incidence of air-borne infection. Although we have no clinical evidence to support this view, preliminary experiments have shown that air-borne counts were 20–40% lower in wards in which no counterpanes were used. In these experiments the upper surface of the top blankets was not brought in contact with the other bedclothes. This measure, together with the intermittent use of framycetin sulphate nasal spray (to be reported later) and the frequent laundering of the top blanket, would undoubtedly lower both nasal carrier rate and air-borne dispersion of *Staph. aureus* in a hospital ward.

Summary

The spread of a marker organism, *Staphylococcus citreus*, in a normal surgical ward has been investigated. The organism was introduced in the ward in a known concentration on known textiles and its rate and intensity of dispersion were determined over 48 hours. The results showed that the dispersion of the marker organism in the ward was very similar when cotton and woollen blankets were used as source textiles. Of the two, cotton blankets yielded a higher intensity of air-borne contamination than woollen blankets. The use of counterpane covers greatly increased the dispersion of the organism. This "counterpane effect" seems to be due to the removal of bacteria-carrying particles from a contaminated textile surface by friction. It is suggested that air-borne dispersion of organisms from textiles might be reduced in the absence of counterpanes provided the upper surface of the top blanket is not brought into contact with the other textiles.

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HEAT ILLNESS IN INFANTS AND YOUNG CHILDREN

A STUDY OF 47 CASES

BY

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Stimulated by the waging of two world wars in the tropics, considerable interest has been shown in the reactions of the adult human body to heat stress and in the illnesses induced by heat. Far less has been written on this subject in infants and children, though several authors have made an experimental approach and have obtained valuable information; the work of Cooke, Pratt, and Darrow (1949–50) and of Kuno (1956) is notable.

An opportunity to contribute to knowledge of heat illness in children was provided by a heat wave in Melbourne in the period January 17–20, 1959. Though very variable, summer conditions in Melbourne are normally temperate, with a few days each year over 100° F. (37.8° C.); humidity is usually moderate. This heat wave, the most intense for 50 years, was distinguished by sustained high temperatures—95 to 109° F. (35 to 42.8° C.) by day and 75 to 90° F. (24 to 32.2° C.) at night—and by low humidity. In Fig. 1 it will be noted that two other periods of fairly intense heat occurred in the summer, but that no ill children were seen in these periods.

Case Material and Methods

We saw 47 infants and children who were ill principally as a result of the heat; six of these were dead on arrival at the hospital. Though the heat complicated all cases over this period, only those in whose illness it played the major part are included in this study. A further eight patients died after admission, but in only six of these was death directly attributable to the heat-induced illness. The remaining two (Cases

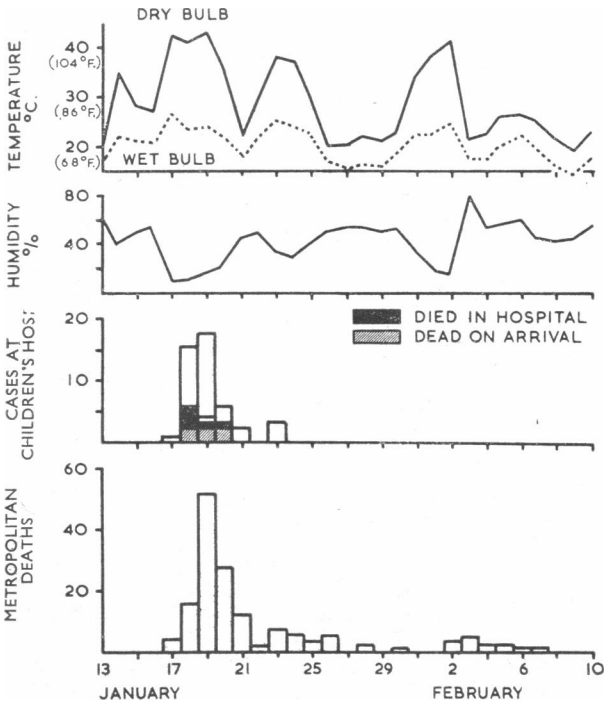

FIG. 1.—Weather conditions in Melbourne from January 13 to February 10, 1959 ; against these are shown the cases of heat illness presenting to the Royal Children's Hospital and the deaths recorded as due to heat illness in the metropolitan area. Temperature readings are daily maxima.

26 and 35) died from underlying chronic diseases some days after recovery from their initial illness, and have, in this discussion, been regarded as survivors.

Admissions occurred mainly on the second and third hot days (Fig. 2), and were most frequent in the afternoon and at night. In some country areas similar conditions had existed for over a week, and four cases came from these parts.

All cases were seen personally, and in all except two, whose parents could not be traced, the history of the illness was carefully retaken by one or other of us. Special inquiries were made about chronic or acute illness, developmental progress, and feeding difficulties. Home visiting by one of us (J. A.) enabled a subjective assessment to be made of the ability of the mother, of the standard of housing, and of the heat of that part of the house in which the patient was kept. Such visits were made to 38 homes—others were too far distant, or mothers refused to be visited or could not be traced. Because of the sudden great burden of work which fell on the reduced staff available during the week-end, few laboratory investigations were made.

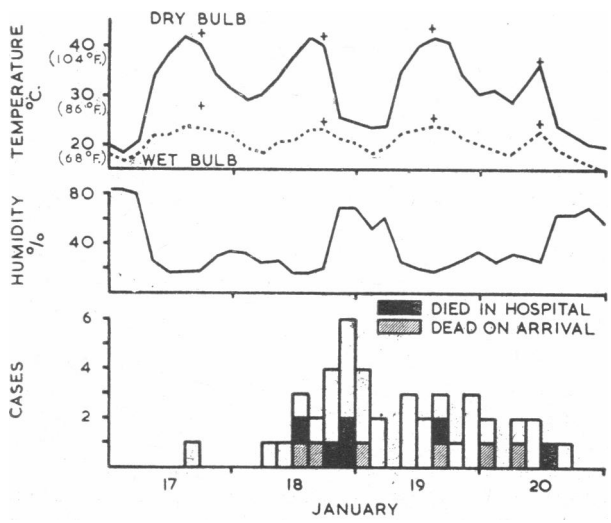

FIG. 2.—Weather conditions during the heat wave (January 17–20) and admissions of cases of heat illness.

Post-mortem examinations were made of all 12 fatal cases. The hospital pathologist examined the children who died after admission, and the pathologist to the Victorian Government examined those dead on arrival. It is hoped to follow the survivors for some years to assess whether any permanent brain damage occurred.

The age range was from 3 weeks to 6 years ; a large group of the babies were between 3 and 9 months (Fig. 3). Many of the older children were mentally deficient or had chronic diseases. Only one infant was breast-fed (Case 1), but at 3 weeks the breast-feeding was not yet satisfactorily established. There was an excess of males over females, which is apparent only in the children over 1 year (Table I), but the severity of cases and the mortality rate were similar in both sexes.

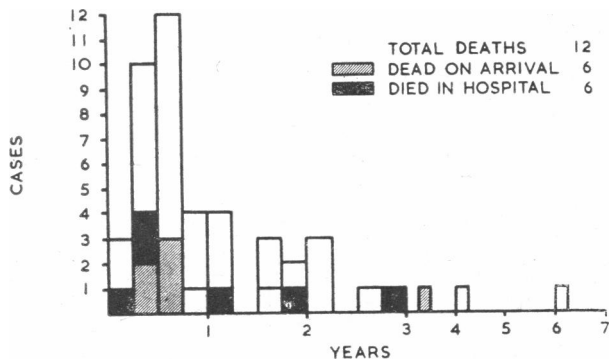

FIG. 3.—Age distribution of 47 children admitted with heat illness, and outcome of their illnesses.

TABLE I.—*Sex Distribution of 47 Cases*

	Total	Under 1 Year	1 Year and Over	Deaths
Males	29	14	15	7
Females	18	15	3	5

Clinical Pattern and Laboratory Findings

On arrival at hospital many of the babies and children were extremely ill and some were moribund. The outstanding features were dehydration, poor peripheral circulation, and impairment of consciousness. Dry mouth and sunken lustreless eyes were noted ; loss of tissue turgor was not marked, but the skin felt "doughy." Peripheral cyanosis with sluggish skin circulation was common, but peripheral arterial pulses were quite strong in all but the most ill. Impairment of consciousness was pronounced in the severe cases, and was present to some degree in nearly all ; lesser degrees of impairment were accompanied by restlessness. Rapid deep respiration, without acidotic breath or ketonuria, was frequent, and irregular respirations occurred in the most ill. Although common, hyperpyrexia was less striking than the preceding features. Table II lists the salient features of all cases. Correlation between the main features was marked, but dehydration was more closely related to severe disturbance of consciousness and to subsequent fits than was hyperpyrexia.

TABLE II.—*Description of Selected Features of All Cases*

Case No.	Sex	Age	Day*	Predisposing Factors	Acute Illness	Extra Fluid Offered	Dehydration	Fever† °F.	Fever† °C.	Conscious State	Serum Na+ (mEq l.)	Serum Cl- (mEq l.)	Fits	Outcome
1	F	3 w.	2	Feeding difficulty	—	Early	Gross	108	42·2	Coma	—	—	Before admission	Death 1 hour after admission
2	M	7 w.	2	—	Severe diarrhoea	Late	Moderate	105·8	41·0	Alert	—	—	—	Recovery
3	M	2 m.	2	—	U.R.T.I.	Never	,,	106·8	41·6	Drowsy	159‡	124‡	6–18§ hours after admission	Probable recovery. Transient hemiparesis
4	M	3 m.	1	Failure to thrive	—	,,	Gross	<105	<40·6	Coma	—	—	—	Recovery
5	M	3 m.	4	—	—	Late	,,	106·8	41·6	,,	150‡	120‡	Before and 8–12 hours after admission	,,
6	M	3 m.	2	No information										Death in 1–2 minutes
7	F	4 m.	2	Failure to thrive	U.R.T.I.	Late	Gross	103·4	39·7	Coma 24 hours	—	—	8–12 hours after admission	Dubious recovery
8	F	4 m.	3	—	—	,,	Moderate	105	40·6	Coma	—	—	—	Recovery
9	F	4 m.	2	—	Severe diarrhoea. Salicylism	Early	Gross	107·6	42·0	Drowsy	—	—	5–12 hours after admission	Death, 16 hours after admission
10	F	5 m.	2	—	Mild diarrhoea	Never	,,	107·2	41·8	,,	—	—	Once, 12 hours after admission	Recovery
11	F	5 m.	2	Slow development	—	,,	Moderate	108	42·2	Coma 12 hours	—	—	Before and ½–12 hours after admission	Alive. Poor development
12	F	5 m.	4	Amyotonia congenita	—	Early	Severe	>105	>40·6	—	—	—	—	Dead on arrival
13	F	6 m.	2	—	No information									,, ,,
14	M	6 m.	3	Feeding difficulty. Hyperkinetic	Severe diarrhoea	Early	Moderate	<105	<40·6	Alert	—	—	—	Recovery
15	M	6 m.	3	Hyperkinetic	Teething	Late	,,	103	39·4	Drowsy	—	—	—	,,
16	F	6 m.	3	Hyperkinetic. Feeding difficulty	Slight diarrhoea	Early	Mild	105·2	40·7	,,	150	124	—	,,
17	F	6 m.	4	—	—	Late	Severe	>105	>40·6	—	—	—	—	Dead on arrival
18	M	6 m.	3	—	—	Never	No information							,, ,,
19	F	6 m.	4	Slow development. Feeding difficulty	Teething U.R.T.I.	Late	Severe	102·5	39·2	Drowsy	146‡	116‡	7–15 hours after admission	Recovery
20	F	6 m.	3	Slow development	,, ,,	Never	Gross	103·4	39·7	Coma	—	—	4–10 hours after admission	Dubious recovery
21	M	7 m.	5	—	Severe diarrhoea	Late	Severe	<105	<40·6	Drowsy	—	—	—	Recovery
22	F	7 m.	2	Slow development. Feeding difficulty	U.R.T.I.	Early	Moderate	106·2	41·2	,,	—	—	—	,,
23	M	7 m.	4	—	—	Late	,,	<105	<40·6	,,	—	—	—	,,
24	M	7 m.	3	—	—	Never	No information						Before admission	Dead on arrival
25	F	8 m.	3	Brain damage from previous hyperpyrexia	—	Early	Mild	<105	<40·6	As usual	—	—	—	Dubious
26	M	10 m.	2	Severe epilepsy. Moderate retardation	Stomatitis	Late	Gross	105	40·6	Coma	—	—	—	Recovered, then inhaled vomitus in fit and died
27	M	10 m.	8	Hyperkinetic	—	Early	,,	Slight		,,	—	—	—	Recovery
28	M	11 m.	3	Slow development	Teething. Slight diarrhoea	,,	Moderate	104·6	40·3	,,	—	—	16–26 hours after admission	Poor recovery
29	F	11 m.	3	Feeding difficulty	—	,,	Mild	Slight		Drowsy	140	103	—	Recovery
30	M	12 m.	3	Spastic diplegia feeding difficulty	—	,,	Moderate	103·4	39·7	,,	—	—	—	,,
31	M	12 m.	3	Slow development	Slight diarrhoea	Late	,,	105	40·6	,,	—	—	—	Dubious recovery
32	M	13 m.	3	Mild retardation	U.R.T.I.	,,	Mild	Slight		,,	—	—	—	Recovery
33	M	14 m.	4	—	Stomatitis	Early	Gross	106	41·1	Coma	160	110	20 hours after admission	Death 24 hours after admission
34	F	16 m.	2	Fallot's tetralogy	U.R.T.I.	,,	Mild	Slight		Alert	—	—	—	Recovery
35	M	18 m.	3	Gross retardation. Urethral valves. Renal failure	Slight diarrhoea	,,	Gross	108	42·2	Coma	—	—	Continual twitching	Recovery, then death post-operatively
36	M	19 m.	2	Slow development	Diabetes Anaesthetic	,,	Severe	Slight		Coma for 2 days	—	—	—	Recovery
37	M	20 m.	8	—	—	,,	,,	Slight		Drowsy	—	—	Before admission	,,
38	M	22 m.	2	Fibrocystic disease	—	,,	Gross	103·8	39·9	Coma	—	—	6–12 hours after admission	Death 15 hours after admission
39	M	22 m.	3	Slow development	—	,,	,,	>107	>41·7	,,	140‡	113‡	Once 18 hours after admission	Dubious recovery
40	M	26 m.	3	Severe retardation	—	Never	Mild	103	39·4	As usual	—	—	—	Recovery
41	F	26 m.	8	Fibrocystic disease	—	Early	,,	Nil		Alert	140	89	—	,,
42	M	26 m.	3	Slow development	—	Late	Gross	>107	>41·7	Coma	150‡	106‡	Before and 17 hours after admission	Dubious recovery

* Day of heat wave on which case presented. † Highest measured level. ‡ Estimation made after considerable intravenous fluid given. § Fits throughout periods indicated. U.R.T.I. = Upper respiratory tract infection.

(*Table II continued overleaf*)

TABLE II—*continued*

Case No.	Sex	Age	Day*	Predisposing Factors	Acute Illness	Extra Fluid Offered	Dehydration	Fever† °F.	Fever† °C.	Conscious State	Serum Na^+ (mEq/l.)	Serum Cl^-	Fits	Outcome
43	F	31 m.	2	Kernicterus. Severe retardation	Teething	Late	Gross	106	41·1	Drowsy	—	—	—	Recovery
44	M	33 m.	3	Severe retardation	—	Early	,,	>107	>41·7	,,	—	—	—	Death 54 hours after admission
45	M	3½ y.	2	Severe retardation and chronic bronchitis	—	Never	—	—	—	—	—	—	—	Dead on arrival
46	M	4½ y.	8	—	—	Early	Moderate	Slight		Coma	—	—	—	Recovery
47	M	6 y.	5	Severe retardation	—	,,	Gross	,,		Drowsy	142‡	112‡	—	,,

* Day of heat wave on which case presented. † Highest measured level. ‡ Estimation made after considerable intravenous fluid given. § Fits throughout periods indicated.

The symptoms preceding this clinical pattern were irritability and refusal to take fluids for a period of from several hours up to a day or two, then listlessness, followed by a rapid or even sudden deterioration with loss of consciousness. Fits occurred before admission in six cases. In some, death resulted within half an hour of this sudden worsening. Oliguria and a late diminution in sweating were noted by some mothers. Considerable vomiting occurred in 20 cases, but all were noticeably unwell before it began.

In the small group of country cases with long exposure to heat the history was of gradual onset of weakness despite good fluid intake; loss of tissue turgor with sunken eyes was apparent, and these cases were neither hyperpyrexic nor unconscious.

As a guide to therapy, serum electrolyte estimations were made in a few typical cases, and, as these showed the expected high levels in the city cases and low levels in country cases and in those with fibrocystic disease, further estimations were made only when unexpected events occurred. No exceptions to these patterns were seen.

Lumbar puncture was performed in eight cases without finding any cellular reaction. Glycosuria was found in nine cases—in all cases a considerable amount of dextrose had been given in the intravenous fluid before the specimen was obtained. The finding was only transient in all but one case. In three cases concentrations as high as 1 to 2% were found, and the blood-sugar levels were 350, 690, and 760 mg./100 ml. respectively. The first baby was in a moribund state despite 18 hours' treatment; the second was an obvious diabetic with classical symptoms for two weeks; and the third showed no ketosis but was regarded as a diabetic, was given insulin, and became irreversibly hypoglycaemic.

Aetiological Factors

Severe chronic disease, of which mental retardation (eight cases) was most frequent, predisposed to illness, and to fatal outcome, by causing difficulty in feeding or excessive loss of fluid. All the retarded children had always been difficult to feed, as had six others. In nine other infants development seemed slower than usual; this fact complicates follow-up studies.

In the recent history the most striking factors (Table III) were the failure of the mother to realize that the child required extra or altered fluid in such heat, the refusal by the child of milk fluids, and the occurrence of vomiting late in the illness, especially in those whose mothers had continued to give milk rather than water. Those children whose refusal of fluid had been prolonged, and those who vomited considerably, showed a higher mortality rate than other cases. Mild concurrent illnesses played a part in some cases, especially by interfering with the child's willingness to drink. However, the presence of diarrhoea or respiratory infection did not influence the overall mortality, though diarrhoea was important in one case (No. 9). Most mothers tried to cool their children and few overclothed them. While the general standards of housing and of maternal ability showed a fairly average distribution for the community which the hospital serves, over half the houses were excessively hot (Table IV), and some parents quoted interior temperatures as high as 115° F. (46.1° C.). Among those dead on arrival poor housing and poor maternal ability were striking.

TABLE III.—*Acute Causal Factors*

	No.	Death in Hospital	Dead on Arrival
Refusal of milk	37	6	4
For less than 1 day	15	2	1
,, 1–2 days	18	4	3
,, more than 2 days	4	—	—
Failure to offer watery fluid from start of heat	21	—	4
Watery fluid never offered	11	—	3
Considerable vomiting	20	4	1
Diarrhoea { Mild	7	—	—
Diarrhoea { Severe	4	1	—
Respiratory infection	12	1	—
Teething	6	1	—
Overclad	5	—	1
No attempt at cooling	11	—	2
Doctor's advice delayed or misleading	13	2	3
Lived more than 10 miles from hospital (far country cases excluded)	16	2	4

Inadequate information available about two patients, both of whom were dead on arrival.

TABLE IV.—*Assessment of Housing and Maternal Ability* (38 *Cases*)

Housing: General standard	Satisfactory 24	Substandard 10	Very poor 4
Room temperature	Cool 9	Average 8	Hot 21
Maternal ability	Good 24	Adequate 6	Poor 8

Thirty-one mothers called their local doctor before coming to hospital, and it is disturbing to find that in 13 cases advice was delayed or misleading: there was a high mortality among these cases.

Distance of the home from the hospital (Tables III, V, and VI) was a factor of definite importance. Because of the rapidity of the final deterioration in the severe cases, a journey to hospital taking half to one and a half hours may well have made the difference between life and death. Four of the children dead on arrival and the two who died soon after admission had such journeys.

Treatment and Progress

The two measures used were replacement of the fluid deficiency and cooling. Antibiotics were given only in the very few cases in which signs of chest infection were found. In 35 cases intravenous fluids were required, and in many the need was so extremely urgent that aseptic precautions were waived. In some, vasospasm in the long saphenous vein was such that fluid could not be forced through a cannula inserted at the ankle, and cannulation of the femoral vein was necessary. The fluid initially given varied, depending on the views of the physicians in charge of the cases, but 5% dextrose in N/4 saline was most often used. After the first few hours and the establishment of adequate urinary output, most cases received a solution containing 5% dextrose and 20 mEq/l. of sodium, chloride, potassium, and lactate ions. Cooling was with ice and/or spirit and a fan, and in most cases hyperpyrexia was rapidly controlled.

In nearly all cases an initial rapid infusion of fluid and cooling produced gratifying improvement in circulation and in the conscious state; the infusion was then slowed to a rate which aimed at replacing the estimated deficit in 12 to 24 hours. However, in 13 cases recovery was interrupted by the occurrence of fits 3 to 18 hours after admission; despite heavy sedation some continued to convulse for up to 12 hours. In one case an ether anaesthetic was used to control the fits, and in two others hypothermia was induced by use of chlorpromazine and ice. Prolonged unconsciousness followed in some cases, and the occurrence of fits was associated with high mortality. This complication was confined to city cases with short exposure to heat—that is, cases with hypertonic dehydration—but analysis of the quantities of fluid and salt and of the rates at which they were given failed to show any influence of these factors on the mortality or on the incidence of these late fits. Volumes given ranged from 30 to 125 ml./kg. body weight over the first four hours, and from 80 to 260 mg./kg. over the first 12 hours. Sodium content of the initial fluid varied between 20 and 80 mEq/l. Similarly, the ease with which satisfactory control of temperature was achieved did not appear to influence the mortality or the occurrence of fitting.

The country cases with hypotonic dehydration recovered with slow infusions of 2.5% dextrose in N/2 saline without any problems arising.

Post-mortem Findings

Gross dehydration of all tissues was noted in all those dying before treatment was begun. Tissue or brain oedema was not found in any case. Intracranial haemorrhage was present only in the child with insulin-induced hypoglycaemia. The significance of the pulmonary infective changes in four of the children who were dead on arrival is difficult to assess (see Tables V and VI).

TABLE V.—*Factors in Patients Dead on Arrival*

Case No.	Age	Underlying Disease	Distance from Hospital	Other Factors	Post-mortem Findings
12	5 months	Amyotonia congenita	4 miles	Overclad ++	Dehydration. Bronchopneumonia
13	6 ,,	—	15 ,,	Not known	Dehydration. Neuronal changes in brain stem
17	6 ,,	—	1 mile	Misleading medical advice Maternal ability and home poor	Dehydration. Pneumonitis
18	6 ,,	—	15 miles	Home very bad. Water shortage	Dehydration. Bronchopneumonia. Purulent otitis media
24	7 ,,	—	20 ,,	—	Dehydration only
45	3½ years	Cerebral palsy. Recurrent bronchitis	10 ,,	Maternal ability and home poor	Brain gliosis. Dehydration. Bronchopneumonia

Discussion

Three distinct clinical patterns were seen. The cases admitted from country areas after prolonged exposure to heat showed, clinically and biochemically, the picture of gradual salt depletion. From the drinking of water but failure to eat solids or take extra salt, the salt deficit due to losses in sweat had gradually mounted. The pathogenesis of the children's illness was clear and the treatment simple. A child with fibrocystic disease of the pancreas showed severe salt loss even after short exposure to heat. In the remaining 42 cases the clinical history, the physical signs, and the few electrolyte estimations made, all suggest acute severe water depletion, described in the Medical Research Council's (1958) classification of heat illness as "water deficiency heat exhaustion"; however, a few cases may have suffered "heat stroke."

In these conditions of low relative humidity heat one would expect body-cooling to be achieved at the expense of the loss of large volumes of sweat and respiratory water vapour until gross dehydration occurred. In their excellent balance studies on infants experimentally exposed to heat, Cooke *et al.* (1949–50) found, at 91° F. (32.8° C.) and 20 to 30% relative humidity, a total evaporative loss of 110 ml. as sweat. Both Kuno (1956) and W. V. Macfarlane (personal communication, 1959) have found losses in children to be double those in

TABLE VI.—*Factors in Patients Dying in Hospital*

Case No.	Age	Chronic Factors	Special Acute Factors	Temperature °F.	Temperature °C.	Factors in Hospital Treatment	Fits	Time of Survival	Distance from Hospital	Post-mortem Findings
1	3 weeks	Feeding difficulties (breast-fed)	Poor home medical advice	108	42·2	—	Before admission. Brief	1 hour	20 miles	Brain petechiae. Pulmonary collapse. Adrenal haemorrhage
6	3 months	—	—			—	—	1–2 mins.	15 ,,	Fatty adrenals
9	4 ,,	—	Severe gastro-enteritis. Aspirin given	107·6	42	Ether anaesthetic to stop fitting	Late. Prolonged	16 hours	5 ,,	Gastroenteritis. Small adrenals
33	14 ,,	—	Stomatitis. Poor home medical advice	106	41·1	Hyperglycaemia. Insulin given. Irreversible hypoglycaemia	Late. Prolonged	24 ,,	7 ,,	Subarachnoid haemorrhage
38	22 ,,	Fibrocystic disease of pancreas	Maternal ability poor	104	40	Inadequate salt given. Water intoxication	Late. Prolonged	15 ,,	15 ,,	Fibrocystic disease of pancreas. Bronchopneumonia
44	33 ,,	Cerebral agenesis. Epilepsy	—	108	42·2	—		54 ,,	1 mile	Early bronchopneumonia. Brain gliosis

adults; and the latter author observed that the fluid requirement of a healthy 7-month-old baby living in central Australia, where intense dry heat is constant, was 190 ml./kg./day. The low electrolyte content of the sweat of infants and children is well established in connexion with work on fibrocystic disease of the pancreas and by Cooke *et al.* (1949–50), so that the stress is one of water depletion. There is much evidence, including that of Cooke *et al.* and of Darrow *et al.* (1954), to show that the water is lost mainly from the intracellular phase and that the clinical picture we encountered develops.

Presumably infants and children in central Australia are able to remain healthy in this intense dry heat because of acclimatization and the large amount of fluid given them by their mothers. Acclimatization in children has not been studied, but in adults it results in the more rapid production of increased volumes of dilute sweat, and so achieves cooling at the expense of water loss. The mother's knowledge of her infant's need for water seems of prime importance and is probably learned from her own mother; however, in Melbourne, where such heat is rare, this particular tradition of infant care was apparently lacking in some otherwise competent mothers (Table VII), and even some doctors and infant welfare sisters were not aware of the problem.

TABLE VII.—*Offering of Extra Fluid Compared with Assessment of Maternal Ability* (38 *Cases*)

Extra Fluid	Total	Mother's Ability		
		Poor	Average	Good
Offered early ..	18	1	3	14
,, late ..	11	3	2	6
,, never ..	9	4	1	4

The study of Cooke *et al.* (1949–50) showed the importance of giving extra water rather than extra milk. When milk feedings of usual strength were continued at 91° F. (32.8° C.) they found marked salt retention; dilution of feedings or provision of *ad lib.* water prevented this. Addition of extra salt caused alarming degrees of retention and shift of water from the cells to the extracellular space—this is important, for many people think about giving extra salt in hot weather.

Breast-feeding may have protected the smallest infants (Fig. 3)—in Melbourne about half of all babies are breast-fed at 3 months—for they could regulate their intake according to thirst, and breast milk leaves a smaller solute load for excretion by the kidney than does cow's milk, so allowing a lower minimum urine output. The breast supply was not satisfactorily established in the only breast-fed infant seen. Also, Kuno (1956) reports work demonstrating the absence of sweating in the newborn and its gradual appearance over days and weeks; the fact that only in the smallest babies was hyperpyrexia more frequent than severe dehydration is in accord with this. The special susceptibility of those aged 3 to 9 months may be related to the tendency of babies of this age to refuse feeding when they are in discomfort. Such refusal is uncommon in smaller infants. The fact that adult men sweat more than adult women is well established (Kuno, 1956), but no good explanation of the marked change in sex incidence at 1 year of age can be offered.

While such conditions as diabetes and fibrocystic disease of the pancreas should be looked out for in the victims of heat waves, it is, as always, important to base such diagnoses on clinical features as well as on biochemical findings. Glycosuria and gross hyperglycaemia would seem to occur when one gives large volumes of dextrose-containing fluid intravenously to a very shocked baby, and the fatal consequence of giving insulin when these findings are the only evidence of diabetes has been mentioned.

The basis of treatment must be fluid replacement and cooling. There seems no doubt that cooling should be achieved as soon as possible, and there may be a place for cooling the more severe cases to a mildly hypothermic (90° F.; 32° C.) level by the additional use of chlorpromazine. This seemed the most effective method of controlling the fits in those cases resistant to heavy sedation. In view of the frequency and severity of fits in the more severe cases, the routine use of such a measure to prevent this dangerous complication may seem reasonable.

In these severe cases one might have expected the intravenous administration of too much salt, or of dilute fluids too rapidly, to cause fits. However, such associations could not be shown, and only in one case of fibrocystic disease was definite "water intoxication" apparent. Finberg (1959) thought that infusion of dilute electrolyte solutions, especially if given rapidly, is harmful in hypernatraemic states. The excessive administration of salt which occurred in the production of this condition in his cases may be a very important difference.

There are no published reports of post-mortem findings in such cases. However, Finberg (1959) has shown the occurrence of subdural haemorrhage in hypernatraemic states of different causation, and several authors, including Haymaker *et al.* (1955), have described, in cases of heat stroke without dehydration, haemorrhagic and degenerative lesions in the central nervous system, especially in the cerebellum, cerebral cortex, and basal ganglia. These authors also mention "necrosis and tubular degeneration of the adrenal cortex" in 20 out of 185 cases. Cerebral congestion was reported in all our patients dead on arrival, and 3 of the 12 fatal cases showed acute focal brain lesions. In two survivors ataxia has occurred, transiently in one.

Recommendations in Low Humidity Heat-wave Conditions

Prevention of Illness.—(1) Dilute all milk feedings and offer extra watery fluids at frequent intervals. Failure to achieve high intake constitutes an indication for special care—for example, tube feeding. (2) Give no extra salt unless the heat wave continues for more than five days. (3) Keep infants or ill children in a cool place, and cool them by sponging or with fans.

Treatment of a Patient Severely Ill After Brief Exposure to the Condition.—(1) Urgent institution of intravenous infusion, giving cool fluids approximating to N/4 saline in dextrose rapidly until satisfactory peripheral circulation is achieved, then at a rate which will replace the estimated deficit in 24 to 48 hours. Allowance must be made for variation in maintenance requirements according to the air temperature. (2) Cool by any convenient method. (3) As recovery begins, administer sedation in anticonvulsant dosage, or produce mild hypothermia by use of surface cooling and chlorpromazine. Maintain such anticonvulsant measures for at least 18 hours.

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.

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"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

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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