of their mother. The wording suggested was "To live in the hearts of those we love is not to die." This had tremendous emotional significance for the patient, whose feelings of guilt became overwhelming. Next day the symptoms began.

Psychosomatic Assessment

The major personality features in cases of ulcerative colitis described by other authors are easily recognizable in this patient. She was emotionally immature, particularly in her relationship with a dominant and possessive mother, and the keynote of that relationship was the hostile-dependence which evoked a considerable degree of guilt. Her intelligence was well above average.

Environmental stress situations immediately preceded the onset of symptoms on each occasion. The situations were associated with marriage, proposed marriage, or with a threat to the security of her emotional dependence on her mother, such as the visit of her sister in 1942. It was felt that her desire for marriage and emancipation from her mother's domination was in conflict with her basic personality pattern.

The resentment, and emotional tension resulting from her conflict, apparently manifested in disturbances in the function and ultimately the structure of the lower bowel, as has been described by other authors.

This case report seems to strengthen the plea for psychiatric investigation in patients with ulcerative colitis.

Summary

The literature on the psychosomatic aspects of ulcerative colitis has been reviewed.

The report of a case of ulcerative colitis is presented in which the patient displayed disturbances in interpersonal relationships, characterized by emotional immaturity, guilt, dependence, and resentment towards a dominating mother. The stressful situations evoking the disturbances were associated with marriage, proposed marriage, and situations threatening the security of the patient's dependence.

The major personality features and the precipitating environmental factors in the report conform to those stressed by other authors.

It is felt that this case report strengthens the plea for psychiatric investigation in patients with ulcerative colitis.

I wish to record my thanks to Dr. M. M. Suzman, specialist physician on the staff of the Johannesburg General Hospital, for his constant encouragement and guidance, both in the handling of this case and in the preparation of this article, and to Dr. Stanley Cobb, psychiatrist-in-chief, Massachusetts General Hospital, for his most helpful suggestions regarding the review of the literature.

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ELECTROLYTE IMBALANCE IN TUBERCULOUS MENINGITIS

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Mestrezat (1912) first focused attention on the diagnostic value of the chloride content of the cerebrospinal fluid (C.S.F.) in tuberculous meningitis, and he and other workers (Mestrezat, 1924; Greenfield and Carmichael, 1925; Foord and Forsyth, 1933) have all laid great stress on the value of a low C.S.F. chloride in the diagnosis of this disease. Merritt and Fremont-Smith (1937) observed a correlation between the chloride contents of the C.S.F. and plasma, and found a lowered concentration of chloride in the C.S.F. in any condition in which the plasma chloride concentration was low. As a low C.S.F. chloride associated with a low plasma chloride is often found in purulent meningitis, in intracranial disease, and in any condition causing vomiting, the occurrence of a low C.S.F. chloride in tuberculous meningitis cannot be regarded as being specific.

The use of streptomycin in the treatment of tuberculous meningitis has made possible the long-term study of the hypochloraemia and other electrolyte disturbances, of which a lowered C.S.F. chloride concentration is a manifestation. Rapoport et al. (1951) studied three children aged between 2 and 3 years by load-clearance techniques one week, two weeks, and three months respectively before death, when the patients whose food intake was poor were more or less comatose and having mild convulsions, but were not vomiting. The effects of loads of hypertonic saline, ammonium chloride, potassium chloride, and sodium thiosulphate were studied in these three children and one control. These workers concluded that, because the concentration of sodium and chloride in the urine did not fall to minimal amounts when the levels in the plasma were low, there was some "disturbance in renal electrolyte control," probably located in the proximal tubule of the kidney. At post-mortem examination the kidneys of these children were normal macroscopically and microscopically.

Further studies on the electrolyte disturbance in tuberculous meningitis were made by Harrison, Finberg, and Fleishman (1952). They investigated 16 children, finding that plasma concentrations of sodium and chloride were reduced, but that no consistent change in the potassium occurred. They found the sodium concentration in red blood cells and skeletal muscle to be increased, and the potassium to be normal in the former and reduced in the latter. Although they concluded that a disturbance governing ion equilibrium existed in these cases, no definite hypotheses on the causation were advanced.

It is conceivable that successful therapy against these electrolyte disturbances may complement specific therapy in tuberculous meningitis and assist recovery.

In the present investigation the electrolyte patterns of children with tuberculous meningitis have been related to their clinical conditions. Cases showing abnormal patterns have been followed for long periods, and the effects of intravenous injections of hypertonic saline and potassium salts orally have been studied.

Material

Nineteen patients with tuberculous meningitis and two with miliary tuberculosis only have been studied in this investigation. Their ages ranged from 1 to 13 years. Thirteen of these were receiving streptomycin by intrathecal and intramuscular routes, and one of these, who had gross internal hydrocephalus, was also receiving "purified protein derivative" (P.P.D.) intrathecally. Five patients who were improving had finished their course of intrathecal treatment but were still receiving streptomycin intramuscularly, and the two with miliary tuberculosis only were receiving the same. One patient was on the point of discharge and was receiving no treatment. Four, all in a poor general condition with an electrolyte disturbance, were given loading doses of saline intravenously. They were all vomiting at the time and were receiving streptomycin intramuscularly and intrathecally. Two of these who were still in the same condition were given potassium salts orally. All these patients were under treatment in the Streptomycin Unit of this department. Hypertonic saline was given to two cases in the general wards as controls for the saline-loading investigations.

Procedure

Blood was collected from the 21 patients at various stages of the disease, and the plasma CO₂ content, plasma or serum chloride, and serum sodium concentrations were determined. Urine was collected at the same time and the urine chloride content estimated. The estimations of various electrolytes in plasma or serum were repeated in seven children with hypochloreaemia at intervals varying from 6 to 164 days after the initial investigations.

Infusions of hypertonic saline were given to four patients with hypochloreaemia who were in the active stages of the disease. The loading dose of saline was given intravenously as a 15% (w/v) solution in amounts calculated to bring the plasma chloride back within the normal range, assuming that the Cl ion is mainly extracellular and that the extracellular compartment is approximately 25% of the body weight. Two controls received an arbitrary dose of sodium

chloride. Three of the patients and the two controls were given 30–40 ml. of water every half-hour for three to four hours before the load and one and a half to two hours after it. One patient was given N/5 saline by mouth in doses of 30 ml. every half-hour for four and a half hours before the load and for one and a half hours afterwards. Urine was collected for a period before the hypertonic saline injection and for one and a half to two hours afterwards: the volume was noted and the sodium and chloride concentrations were estimated. Samples of blood for estimations were collected before the hypertonic saline injection and at intervals afterwards.

To test the hypothesis that these children might be suffering from a potassium deficiency two children were given potassium orally in amounts equal to an arbitrarily chosen deficit of one-quarter of their total body potassium. The potassium was given in divided doses over a period of five and four days respectively as a mixture of the citrate and chloride in a ratio similar to the ratio of bicarbonate to chloride in the extracellular fluid. This was done to avoid, so far as possible, any secondary effects on the acid-base state. In both these cases the diet was checked so that potassium intake did not suffer large fluctuations. Urine was collected during the four days before administration, during the time potassium was given, and for a period of four days afterwards.

All cases in this study were receiving *para*-aminosalicylic acid in the form of the sodium salt. The dosage was always approximately 0.5 g./kg., and it has been calculated that each case received around 60 mg./kg./day of sodium in this way.

Analytical Methods

Chloride was estimated on 0.2 ml. of plasma by Sendroy's (1937) method. Plasma CO₂ content was estimated on plasma which had been collected and centrifuged under liquid paraffin in either the volumetric or manometric apparatus of Van Slyke (Van Slyke, 1917; Van Slyke and Neill, 1924). Sodium was precipitated from a serum filtrate as sodium zinc uranyl acetate according to the modification of Butterworth (1951). The method of Jacobs and Hoffman (1931) was used for the determination of serum potassium. All the urine potassium determinations and many of the sodium estimations were made on an E.E.L. flame photometer.

Results

1. Electrolyte Patterns in Blood and Urine

Details of 21 cases are given in Table I. The cerebral condition was assessed in most cases by the presence or absence of papilloedema. Although many cases had air

TABLE I.—Data of 19 Cases of Tuberculous Meningitis and 2 Cases of Miliary Tuberculosis

Case No.	Age (Years)	Sex	Blood (mEq/l.)			Urine (mEq/l.)	Duration of Illness from Onset	Cerebral Condition	Vomiting in Preceding Fortnight	Period of Treatment
			Plasma Cl	Plasma CO ₂ Content	Serum Na					
1	9	F	89	30	131	24	13 days	Papilloedema	++	6 days
2	10	F	92	31	135	57	33 "	Normal	++	23 "
3	1½	M	82	34	140	18	4 weeks	Papilloedema	+++	4 "
4	5	M	87	29	138	98	4 months	Papilloedema and hydrocephalus	+	3 months (P.P.D.)
5	8	M	92	28	137	—	26 days	Papilloedema	++	5 days
6	11	F	83	31	132	34	15 "	"	+	8 "
7	2	M	80	23	133	123	16 "	Normal	++	2 "
8	5	F	88	27	135	37	15 "	Papilloedema	+	3 "
9	3½	M	89.5	26	124	56	11 "	Normal	+	3 "
10	9½	F	91	20.5	138	55	23 "	"	+	2 "
11	13	M	102	25	142	140	8½ months	Normal	—	7 months (completed for 1½ months)
12	3½	M	101	26	143	98	12 "	"	—	11 months
13	1½	M	98	24	144	—	8 "	"	—	6 months (completed for 2 months)
14	13	F	101	25	150	130	28 "	"	—	25 " (" " 3 ")
15	1½	M	101	23	146	116	3½ "	"	—	3 " (" " 3 weeks)
16	1½	F	99	24	141	50	3 "	"	—	3 months
17	10	F	99	28	141	63	4 "	"	—	3½ "
18	7½	M	99	25	138	135	5½ "	"	—	5 months (completed for 1 month)
19	7	M	102	28	146	134	9 "	"	—	6 months (all treatment completed for 2 months)
20	6	F	99	23	—	117	6 days	"	+	3 days
21	5	F	97	22	142	137	4 weeks	"	+	4 "

+ = Occasional vomiting. ++ = Vomiting once or twice a day. +++ = Vomiting more than twice a day.

studies, only one showed definite hydrocephalus, most of the patients with papilloedema being too ill for air studies to be done.

Ten patients (Cases 1-10) had a plasma chloride concentration of 92 mEq/l. or less, whereas nine patients with tuberculous meningitis (Cases 11-19) and two with miliary tuberculosis (Cases 20 and 21) had plasma chloride values within the normal range (95-106 mEq/l.). The serum sodium concentrations also showed a definite difference, although this was not so marked as in the case of the chloride values. Although only one child in the group with hypochloraemia had a serum sodium as high as 140 mEq/l., all but one of the other group had values above this figure. In nine of the children with hypochloraemia the chloride content of one specimen of urine (collected at the same time as the blood) was estimated. In all of them there were more than minimal amounts of chloride (18 mEq/l. or more) in the urine, in the presence of low concentrations of chloride in the plasma. The occurrence of serious vomiting in the fortnight preceding these investigations is confined to the group with an abnormal electrolyte pattern, as is abnormality in the cerebral condition (Cases 1-10). On the other hand, all 11 children with a normal electrolyte pattern had a normal cerebral condition (Cases 11-21). Nine of them had been treated for periods of three months or longer and had had no recent vomiting. The remaining two children in this group had been ill for a shorter time. They were vomiting occasionally but had miliary tuberculosis only and no meningitis (Cases 20 and 21).

2. Changes in the Electrolyte Pattern with Changes in Clinical Condition

Table II gives the results of determinations of electrolytes in serum or plasma, together with any changes in cerebral condition and vomiting, in seven cases of hypochloraemia,

TABLE II.—Follow-up Blood Electrolytes in Seven Cases

Case No.	Plasma Cl (mEq/l.)	Plasma CO ₂ Content (mEq/l.)	Serum Na (mEq/l.)	Urine Cl (mEq/l.)	Cerebral Condition	Vomiting in Preceding 2 Weeks	Days Between Investigations
1	89	30	131	24	Papilloedema	++	58
	93	26	140	84	Slight papilloedema	+	106
	97	25.5	147	—	Normal	+	
2	92	31	135	57	Normal	++	58
	95	25	143 (?)	35	"	—	106
	97	29	145	—	"	—	
3	82	34	140	18	Papilloedema	+++	32
	87	30	143	59	"	—	120
	97	25.4	140	—	Papilloedema and hydrocephalus	—	
4	87	29	138	98	Papilloedema and hydrocephalus	+	41
	89	31	137	100	"	+	116
	97	26	139	—	"	—	
6	83	31	132	34	Papilloedema	+	6
	89	33	143 (?)	24	"	+	100
	95	31	144	—	Normal	—	
8	88	27	135	37	Papilloedema	++	22
	88	34.5	137	31	"	++	
	89.5	26	124	56	Normal	+	14
9	88	27	141 (?)	116	"	+	55
	94	27	140	—	"	—	

in none of which had there been any persistent attempt to adjust the electrolyte disturbances. The second set of determinations (in Cases 3, 4, and 6) were just prior to the load of hypertonic saline, but this load is unlikely to have affected the subsequent determinations, which were done after intervals of at least 64 days. Similarly, it is unlikely that the third set of determinations (in Case 9) were affected by potassium therapy, which finished 50 days before.

A progressive improvement in the electrolyte values is seen in all children except Case 8. The increase in the concentration of chloride and sodium in the serum or plasma coincided with improvement both in the general condition and in the state of the meningitis, and simul-

taneously there was a diminution in vomiting. Case 3 was developing hydrocephalus during the investigations, but the meningitis healed and his general condition improved and vomiting ceased. Similarly, in Case 4 there was an improvement in the electrolyte pattern at the third estimation, when vomiting had ceased, but hydrocephalus was still present. One patient (Case 8) was extremely ill. She did not respond to treatment, deteriorated rapidly, and died three days after the second set of determinations recorded in Table II.

3. Load of Hypertonic Saline

The injection of hypertonic saline produced a rise in the concentration of plasma chloride and serum sodium in all cases at the second estimation, which was made within one hour of the injection. A third estimation 27-30 hours later was made in two cases of tuberculous meningitis (Cases 6 and 8) and one control. Case 8 was very ill and died a few days later. Homeostasis must have been seriously disturbed, as very little urine was passed through the indwelling catheter for 174 minutes after the injection. In the other two (Cases 6 and Control 2) the serum chemistry returned to the pre-injection levels within 30 hours. Case 6 had a normal serum sodium, but a low plasma chloride, which after a transient rise returned to a low value, indicating an inability to maintain the plasma chloride at a level approaching normal (Table III).

TABLE III.—Results of Sodium Chloride Loads (Electrolyte Levels)

Case No.	Pre-injection Levels (mEq/l.)			Post-injection Levels (mEq/l.) Time after Injections in Hours							
	Na	Cl	CO ₂	Time	Na	Cl	CO ₂	Time	Na	Cl	CO ₂
3	134	93	25	1.0	136	102	23	—	—	—	—
4	137	89	31	0.5	143	101	27	—	—	—	—
6	143	89	33	0.75	147	96.5	30	30	143	92	31
8	137	88	34.5	0.75	139	94	34	27	139	96	37
Control 1	149	96	26	0.5	150	105	23	—	—	—	—
Control 2	142	99.5	28.5	0.5	144	109	27	29	144	100	29

The basic excretion of chloride in mEq/min. during the pre-injection period was assumed to be the same throughout the whole study, and this amount was subtracted from the total excretion in any period. Table IV shows the

TABLE IV.—Results of Sodium Chloride Loads (Excretion)

Case No.	Hydration Prior to Injection	Weight in Kg.	Dose of 15% NaCl in ml.	Time of Study after Injection in Minutes	% Excreted in Period of Study	
					Na	Cl
3	N/5 saline	8.2	17	122	27	4.5
4	Water	15.0	35	162	17	18
6	"	27.7	50	129	17	9
8	"	18.6	30	174	0	0
Control 1	"	24.1	40	228	21	11
" 2	"	22.7	35	177	64	47

percentage of the dose of sodium and chloride that was excreted in the four cases and two controls. The excretion of sodium and chloride during the pre-injection period in Case 3—hydrated with N/5 saline—was much higher than in any other cases, and there was little increase in the excretion of chloride after the hypertonic saline injection. Control 1 showed a response indistinguishable from that of Cases 4 and 6, whereas Control 2 excreted a much greater percentage of the injected dose in 177 minutes. In Case 8 the child was extremely ill and unconscious, and excreted none of the injected dose within 174 minutes. A few days later she became grossly oedematous and died.

4. Administration of Potassium

The effect of potassium administration on the plasma pattern and on the excretion of potassium in the urine of two children with tuberculous meningitis and hypochloraemia is shown in Figs. 1 (Case 6) and 2 (Case 9). Case 6 was given 143 mEq of potassium a day, and

excreted 74% of the ingested potassium during the five days of administration and the subsequent four days. About 26% of the dose was therefore retained, unless it

FIG. 1.—Case 6. Effect of potassium administration on the plasma pattern and the excretion of potassium.

FIG. 2.—Effect of potassium administration in Case 9.

two cases of hypochloraemia resulted in retention of potassium and an improvement in the electrolyte pattern in the case with a normal serum sodium level, while it caused an insignificant retention of potassium and no change in the plasma pattern in the case with a low serum sodium level.

Discussion

This investigation shows, as was to be expected, that the occurrence of an abnormal electrolyte pattern in a group of children suffering from tuberculous meningitis was confined to those in whom the illness was of recent origin or in whom the cerebral condition was abnormal. On the other hand, children who had been under treatment for longer periods or who had miliary tuberculosis only did not show any abnormality in the electrolyte composition of the plasma. The clinical feature, however, which distinguished the two groups in the period preceding the collection of the blood was the food intake and the vomiting. The children with tuberculous meningitis and an abnormal electrolyte pattern had a low food intake and had been vomiting for two or more weeks before the initial estimation of the electrolyte content of the plasma. The importance of vomiting as the immediate cause of the electrolyte disturbance is further demonstrated by the fact that the most marked and most consistent of the changes was the hypochloraemia.

A direct effect of an abnormal cerebral condition on the causation of hyponatraemia and hypochloraemia becomes unlikely in view of the progress of Cases 3 and 4. In both these cases vomiting had ceased, but hydrocephalus was still present at the time that the electrolyte pattern had returned to normal.

In view of the suggestion by Rapoport *et al.* (1951) that defective kidney function accounts for the electrolyte disturbance in tuberculous meningitis, an attempt was made in the present investigation to assess the role of the kidneys. The experiments with the load of hypertonic saline were unfortunately inconclusive because of the different response of the two control cases, one of whom showed no difference from the response of the children with tuberculous meningitis.

Although it is realized that the estimation of the chloride content of one isolated specimen of urine may not give a true picture of the chloride excretion over 24 hours, it is thought that the presence of more than minimal amounts of chloride in the urine on more than one occasion in all the children with hypochloraemia is of significance. Such an occurrence might be due to a hypofunction of the electrolyte-regulating hormones of the adrenal cortex—that is, an Addisonian syndrome. A similar disturbance, however, is observed in potassium-deficient patients, and an experiment with potassium loads orally was planned to test this hypothesis. The retention of 81% of the potassium by a child with hypochloraemia and with a normal serum sodium resulting in a normal electrolyte pattern, and the retention of only 26% of the administered potassium by a child with a low serum sodium, suggests that in some cases at least there is a deficit of potassium and also that large doses of potassium cannot be retained in the presence of a sodium deficit and therefore cannot correct the electrolyte disturbance. It seems that the position is analogous to that in which, in the presence of a deficit of potassium, large amounts of saline do not restore the plasma pattern to normal until potassium is given as well.

Our present study suggests that vomiting and reduced food intake leading to a deficit of the main electrolyte ions Cl, Na, and K can explain the electrolyte disturbance in children with tuberculous meningitis. This study has not excluded the presence of a "disturbance in renal electrolyte control" (Rapoport *et al.*, 1951), neither has it excluded "a disturbance of the mechanisms governing ionic equilibrium between cells and extracellular fluid" (Harrison *et al.*, 1952). We think, however, that it is unnecessary to postulate that such disturbances exist, unless it can be proved that the observed abnormalities in electrolyte metabolism cannot be accounted for by the more simple phenomena of vomiting and reduced food intake.

Although this work is inconclusive, it seems to us that the maintenance of adequate electrolyte intake in the early stages of tuberculous meningitis may be an important factor in assisting these children to recovery.

Summary

The electrolyte disturbance in tuberculous meningitis has been studied. In a group of 19 children with tuberculous meningitis and 2 with miliary tuberculosis only, 10 were found to have an abnormal electrolyte pattern.

A correlation was found between the abnormal electrolyte pattern (hypochloraemia, hyponatraemia, and an increased CO₂-combining power) and the occurrence of serious vomiting.

The abnormal electrolyte pattern was not seen in children who had been treated for long periods, and it disappeared after cessation of vomiting, on improvement in the cerebral condition, and on increase of the oral intake to within normal limits.

Injections of hypertonic saline in four cases did not increase the plasma chloride permanently. Transient rises were noted.

Potassium as a mixture of chloride and citrate was administered to two children by mouth and balance studies were made. In one child with a normal serum sodium 81% of the potassium given was retained. In another child, who had a low serum sodium, only 26% was retained.

It is suggested that the electrolyte disturbances in tuberculous meningitis can be explained by the serious vomiting and prolonged low food intake in the early stages of the disease. The abnormality is not due to a deficiency of any one electrolyte, but is probably due to a combined deficit of sodium, chloride, and potassium. Ensurance of an adequate electrolyte intake may be an important contributory factor to the recovery of patients with tuberculous meningitis.

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CLINICAL AND CHEMICAL STUDIES IN HUMAN LACTATION

VII. THE EFFECT OF DIFFERENCES IN YIELD AND COMPOSITION OF MILK ON THE INFANT'S WEIGHT GAIN AND THE DURATION OF BREAST-FEEDING

BY

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Although it is recognized that there are individual differences in the yield and composition of milk, the practical effect of these differences in terms of successful breast-feeding has received almost no attention. In Sweden, Sydow (1952) investigated the relationship between the milk yield on the sixth post-partum day and the duration of breast-feeding, and found that if the sixth-day yield was under 300 ml. there was a considerably diminished chance of breast-feeding. This was an insensitive index, however, since only 25% of cases were in this low-yield group. Moreover, under "breast-feeding" were included those who were only partly breast-feeding, so that the overall figure of over 90% still breast-feeding at the end of two months, which is well in excess of any reported in this country, is difficult to interpret.

In an earlier study, Sydow (1944-5) gave an indirect indication of the effect of differences in fat content on the weight gain of the infant. Infants whose mothers donated to a milk bank milk which was stripped from the breast after feeding and therefore contained a high proportion of the fat gained less weight than those

whose mothers gave milk which was removed before the feed and contained relatively little fat. The average weight gains were 765 ± 62 g. and 866 ± 42 g. a month respectively. Sydow states that the material was unsuitable for comparison, since the birth weights and ages of the infants were not taken into account, and claims no significance for the results.

Kon and Mawson (1950) found no relationship between the weight gain of the infant after the eighth week and the fat content of the milk; but the milk yield was not known and, moreover, their technique of manually expressing a single morning sample gives no accurate indication of the daily fat output, and no conclusions can be drawn from it.

Gunther and Stanier (1951) found a significant relationship between the amount by which the baby was below its birth weight on the eighth day, and the eighth-day milk volume, but none with the fat content of the milk. The interpretation of this finding is not simple, since the initial weight loss varies very widely and is not related to food intake.

The terms "lactation" and "breast-feeding" must be differentiated. Lactation is a largely automatic physiological function showing wide individual variation. Successful breast-feeding is obviously dependent in the first instance on adequate lactation, but the efficiency with which this basic capacity is exploited depends on such factors as a desire to breast-feed, social custom, intelligence, and general living conditions. It is, in many ways, a test of the capability and character of the mother.

The various circumstances which determine the difference between the number of women having successful lactation and the number who breast-feed successfully will be discussed in subsequent papers, but, in order that the significance of the figures presented here can be appreciated in their proper context, the "official" attitude towards breast-feeding in this community must be described, since the proportion of women breast-feeding can to some extent be influenced by pressure of opinion from doctors and health visitors.

In Aberdeen, preparation of the breast as recommended by Waller (1946) is not practised, although Waller's shields are sometimes used for the treatment of inverted nipples. In hospital, unless there are definite medical contraindications, or unless a woman has firmly stated her intention to bottle-feed, it is tacitly assumed that all women will breast-feed, and much care is taken and time spent encouraging the mother in the lying-in wards. Excess milk is manually expressed after feeds as a routine during the first four or five days. Engorgement of the breast is treated along orthodox lines, which include the use of oestrogens. If the puerperium is uncomplicated the mother remains in hospital for eight to ten days.

If breast-feeding is not fully established in hospital, and if the woman is anxious to persevere, she may be sent home partly bottle-feeding, but there is an increasing tendency, if lactation is obviously poor, to begin artificial feeding in hospital so that efficiency in this technique can be acquired in the security of the hospital rather than in the general turmoil of failing lactation at home. Complementary feeds are given to breast-fed infants in hospital at the ward sister's discretion. There are no rigid rules governing their use, but they are generally given after the third day if the baby is still losing weight or when, after feeding, it still appears