was present. Had the plasma pH been determined it is likely that a state of uncompensated metabolic alkalosis would have been revealed. This is known to occur in conditions associated with a low serum potassium—for example, Cushing's syndrome (Kepler, Sprague, Mason, and Power, 1948; Teabeaut, Engel, and Taylor, 1950)—and rarely in post-operative patients (Kepler *et al.*, 1948).

Myasthenia

The distribution and nature of the muscular weakness observed with diminished tendon reflexes was similar to that described in familial periodic paralysis in which a low serum potassium content is associated with muscle weakness. Responses to direct muscle electrical stimulation, using a Ritchie-Sneath stimulator, showed a raised threshold during the phase of muscle weakness with all pulse lengths used; this was lowered appreciably when the serum potassium returned to normal. These findings are of course contrary to those of tetany as exemplified in Erb's sign, and constitute a paradox in the presence of Trousseau's sign.

Electrocardiographic Changes

The records taken on June 6 and July 15 show striking changes compared with that of August 1, when the serum potassium had returned to normal. Subsequent records showed no further change. It is, however, remarkable that the changes seen on June 6 had occurred long before symptoms of hypokalaemia had appeared, or the serum potassium was found to be abnormal. This may be related to changes in intracellular potassium, which at the time were not reflected in the serum. Cates (1949) likewise noted that electrocardiographic changes appeared before the serum potassium fell. At no time were any clinical signs of cardiovascular abnormality found other than the slight rise of blood pressure when the serum potassium was low.

Hypopotassaemia

This has been reviewed by Darrow (1948) and Danowski (1949). Diminished intake of potassium and excessive loss from vomiting, diarrhoea, fistulae, and intestinal aspiration are regarded as prominent causes. Cushing's syndrome, D.C.A. intoxication, and familial periodic paralysis rarely may be responsible. Diabetic acidosis which is being treated with insulin, especially when sodium is also being administered, is likely to show a serum potassium deficit. Occasionally in advanced renal disease potassium depletion may occur. In the two cases described there was no evidence that impairment of renal function could have been responsible for the depletion of their serum potassium.

Summary

Two cases showing marked muscular weakness associated with treatment with sodium P.A.S. and liquorice extract are reported.

In one case the association of such weakness with a low serum potassium was demonstrated.

The occurrence of Trousseau's sign in this condition is noted.

The mechanism involved in the production of such a serum potassium deficit is examined.

I wish to thank Miss E. Gilchrist for the biochemical investigations involved; Dr. H. Mowat, house-physician, for her careful records; and Dr. W. I. Card, in whose unit the patient was treated, for his helpful advice and criticism at all times. Miss S. H. Cameron, physiotherapist at the Northern General Hospital,

kindly measured the electrical reactions. I also wish to thank Dr. F. E. de W. Cayley for information about his patients and for drawing attention to Cates's (1949) report, and Mr. J. B. Marshall, of the Department of Public Health and Social Medicine, for his advice regarding the statistics summarized in Table II.

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INCIDENCE OF BACT. COLI O GROUP 111 IN SPORADIC INFANTILE GASTRO-ENTERITIS

BY

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Since the publication of the results of Taylor, Powell, and Wright (1949) into the incidence of *Bact. coli* O group 111 (one hundred and eleven) (*Bact. coli* D433) in gastro-enteritis, some of whose work was carried out at this hospital, we here have continued investigations on similar lines for the past two years. Our results are presented below.

Material and Methods

The period covered was from June 26, 1949, to March 3, 1951. During this time 264 cases of infantile diarrhoea and vomiting were admitted to the gastro-enteritis ward. Rectal swabs were taken from every case on admission and thereafter at intervals of two to four days until their discharge. Thirty individual colonies were picked from the rectal swab plate and tested for slide agglutination with a *Bact. coli* O group 111 K serum prepared in rabbits. If no agglutination was found after 30 colonies had been tested, a mass suspension was made by sweeping a platinum loop across the thickest part of the inoculum and subjected to the agglutinating serum. If any agglutination occurred with such a suspension the remaining discrete colonies were tested until a positive result was obtained. One in ten of the colonies previously identified as *Bact. coli* O group 111 was put up to titre by the standard tube technique.

Results

Cases positive on admission, 89, or 33.4%.

Cases negative on admission, 175, or 66.6%.

Cases positive on discharge, 81, or 30.7%.

Cases negative on discharge, 183, or 69.3%.

Cases positive throughout, 14, or 5.3%.

Cases negative throughout, 106, or 40.2%.

Cases positive on admission, negative on discharge, 42 or 15.9%.

Cases negative on admission, positive on discharge, 33, or 12.5%.

Cases positive on admission, varying throughout, 33, or 12.5%.

Cases negative on admission, varying throughout, 36, or 13.6%.

Cases Positive on Admission.—89 cases, or 33.4% of total, of which 14, or 15.7%, remained positive throughout; 42, or 47.2%, became negative and remained so; 33, or 37.1%, varied throughout.

Cases Negative on Admission.—175 cases, or 66.6% of total, of which 106, or 60.6%, remained negative throughout; 33, or 18.9%, became positive and remained so; 36, or 20.5%, varied throughout.

Cases Positive on Discharge.—81 cases, or 30.7% of total, of which 14, or 17.3%, had been positive throughout; 33, or 40.7%, were originally negative; 34, or 42.0%, varied throughout.

Cases Negative on Discharge.—183 cases, or 69.3% of total, of which 106, or 57.9%, had been negative throughout; 42, or 22.9%, were originally positive; 35, or 19.2%, varied throughout.

The complete variability of these findings appears to us to need no comment.

Control Series

During the period September 16, 1950, to March 3, 1951, each time a new case was admitted to the gastro-enteritis ward a similar case admitted without gastro-intestinal upset to some other ward was swabbed as a control. Each control was swabbed as soon after admission as was practicable, and for the sake of completeness two control series were run—the first under the age of 2, as were all our cases of gastro-enteritis, and the second over the age of 2. The results obtained are shown in Table I, the period having been broken up into four weekly periods. It will be seen that there is notably little difference between the isolations of *Bact. coli* O group 111 from the cases of gastro-enteritis and those from the controls.

TABLE I.—Cases and Controls from which *Bact. coli* O Group 111 was Isolated on Admission

4-weekly Period	Cases of Gastro-enteritis	Under 2 Controls	Over 2 Controls
1	2 of 12 (16.7%)	2 of 12 (16.7%)	4 of 14 (28%)
2	2 " 12 (16.7%)	3 " 12 (25%)	1 " 12 (8.3%)
3	1 " 15 (6.7%)	2 " 15 (13.4%)	0 " 15
4	2 " 15 (13.4%)	3 " 15 (20%)	3 " 15 (20%)
5	3 " 16 (18.8%)	3 " 16 (18.8%)	0 " 16
6	2 " 15 (13.4%)	4 " 15 (26.8%)	3 " 15 (20%)
Totals	12 " 85 (14%)	17 " 85 (20%)	11 " 87 (12.6%)

L.C.C. Notifications of Gastro-enteritis

During the period from June 26, 1949, to August 19, 1950, we were able to obtain from the L.C.C. Health Department the number of cases of gastro-enteritis notified to them. The cases admitted to the Hospital for Sick Children were essentially comparable in that they were drawn from the same area, but possibly not so

comparable in that naturally the more severe cases were likely to have been sent to hospital, and therefore our cases would be more severe than the average notified. It seemed worth while, however, to correlate the numbers of notifications with the number of hospital cases showing *Bact. coli* O group 111 on admission. Since the number of beds available for cases of gastro-enteritis was limited we have expressed the positive Great Ormond Street cases as a percentage of the total admitted. The L.C.C. cases were included in the four-week period in which the actual illness arose, and not according to the date of each notification. The total L.C.C. cases and Great Ormond Street percentages are shown in Table II. The Chart shows a completely inverse relationship between the total number of cases of gastro-enteritis in the L.C.C. area and the percentage of our sample of this total from whom *Bact. coli* O group 111 was recovered on admission.

TABLE II.—Total Cases Notified to the London County Council, and Those Admitted to Great Ormond Street, for Each Four-week period from June 26, 1949, to August 19, 1950

Period	Total L.C.C. Notifications	Total G.O.S. Cases	G.O.S. Cases with <i>Bact. coli</i> O Group 111 on Admission	
			No.	Percentage of Total
1	102	15	6	40.0
2	77	8	5	62.5
3	108	14	5	35.7
4	87	13	7	53.8
5	104	9	4	44.4
6	106	13	3	23.1
7	88	12	3	25.0
8	60	16	5	31.3
9	49	6	2	33.3
10	66	13	3	23.1
11	41	9	4	44.4
12	46	10	7	70.0
13	59	9	5	55.5
14	44	8	5	62.5
15	30	9	6	66.7

Graph showing reverse relationship between cases of gastro-enteritis in the L.C.C. area and the percentage of cases at Great Ormond Street Hospital from which *Bact. coli* O group 111 was recovered on admission.

Discussion

We are well aware of the findings of specific agglutinable types of *Bact. coli* in association with infantile diarrhoea and vomiting, recently epitomized by Taylor (1951) and Rogers (1951), and the support which has been forthcoming for their work from almost every other country in the world. It is true that we have investigated only the incidence of *Bact. coli* O group 111, and

have not taken into account the beta and other agglutinable types of *Bact. coli*. Even so, we are perplexed by the discrepancy between our findings and those of others, and record the figures for what they are worth.

The incidence of this particular strain of *Bact. coli* in our cases is much lower than we had expected, and we thought that our 30 individual colonies slide-agglutinated would have given us a higher percentage of positive results than the three or four colonies reported as having been examined by some other workers. A false positive slide agglutination is more likely than a false negative, and were our findings to be explained on technical grounds we would have expected an even higher incidence than was obtained. Similarly, from a study of the published work we expected our control series of cases—as comparable as we could get them, except that there was no gastro-intestinal disorder—to have a much lower incidence than was found.

Taylor (1951) and others have emphasized that their greatest isolation of *Bact. coli* O group 111 was obtained in epidemics, whereas our cases, with the exception of the few arising *de novo* in another ward of the hospital and subsequently transferred to the gastro-enteritis unit, cannot be regarded as epidemic cases. The L.C.C. cases are probably mostly sporadic; and here again, to put it no stronger, our figures show that there is no correlation between the total number of cases notified and the percentage incidence of *Bact. coli* O group 111 in our hospital cases.

We are unable to offer any explanation why our findings are so different from those of others, unless such is to be found in some difference between the epidemic and sporadic disease, or an enhancement of virulence of a causal organism leading to an epidemic. Even so, we are disappointed that an organism impliedly associated with the pathogenesis of epidemic gastro-enteritis has in our experience been so infrequently found in sporadic cases of what appears to be the same disease. We are perturbed, also, by the incidence of the same potential pathogen in the rectal swabs of healthy babies of the same age group, an incidence for all practical purposes identical over a period of six months with that found in our cases of sporadic infantile gastro-enteritis.

There has been a tendency recently to regard these agglutinable strains of *Bact. coli* as the cause of gastro-enteritis, and, while we do not suggest that there is no relationship between the organism and the disease, our results do not support the idea that the particular organism we have investigated is causal.

Summary

Figures are presented of the isolation of *Bact. coli* O group 111 (D433) from 264 cases of sporadic infantile gastro-enteritis observed over a period of two years.

In a shorter control series this organism was isolated as often as in the cases of the disease.

No correlation was found between the total number of cases notified to the L.C.C. and the percentage of cases in which the organism was found on admission to the Hospital for Sick Children.

We are grateful to Dr. Joan Taylor for advice, to Sir Allen Daley for the L.C.C. notifications, and to the physicians of the Hospital for Sick Children for access to their cases.

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SO-CALLED ACCIDENTAL MECHANICAL SUFFOCATION OF INFANTS

BY

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During 1940–9 in England and Wales the average annual number of deaths ascribed to the accidental mechanical suffocation of infants aged under 1 year was 620. There has for some time been considerable doubt whether the deaths so registered are in fact due to the cause alleged. Brend (1915) believed that many supposedly suffocated infants died from natural causes, and gave some statistics that lent support to his suggestion. Davison (1945), coroner for the City of Birmingham, ordered post-mortem examinations to be carried out by skilled pathologists on every case in which asphyxia was considered to be a possible cause of death in an infant; he found that out of 318 cases only 24 died from asphyxia caused mechanically. In the U.S.A., Werne and Garrow (1947) investigated the deaths of 167 infants where accidental mechanical suffocation was alleged to be the cause. In 43 the gross findings were adequate to establish a natural cause of death; of the remaining 124 cases, microscopical studies showed natural causes of death in many. In Australia, Bowden (1950) recorded the post-mortem findings in 40 cases of sudden death in infants, nearly all in bed. Morbid changes were found in all of them.

Though the pathological evidence indicates that some infant deaths ascribed to accidental mechanical suffocation are much more probably due to disease, it does not enable us to estimate what proportion. Suffocation may leave no signs discernible in an infant after death, and it is possible that an infant suddenly smitten with pneumonia, for example, might succumb to asphyxia from pressure of its sleeping mother's breast or from a pillow in its cot, while a healthy infant would have no difficulty in surviving either hazard. But certainly a considerable proportion of the cases described showed lesions sufficient to account for death without any need to invoke mechanical suffocation as a cause when there was no firm evidence of it.

The numbers of deaths in England and Wales recorded by the Registrar-General in the periods 1921–30, 1931–9, and 1940–9, distinguished by sex of infants and circumstances of death, are shown in Table I. Because of changes in the method of classification, the actual numbers of deaths in the first two periods are not strictly comparable with those in the third.

TABLE I.—Deaths from Accidental Mechanical Suffocation in Infants in Three Periods, 1921–49, England and Wales

Years	In Bed		In Cot, Cradle, etc.		By Food		Other		Total	
	M	F	M	F	M	F	M	F	M	F
1921–30	1,810	1,622	171	112	214	146	203	185	2,398	2,065
1931–9	1,031	791	311	192	313	248	45	32	1,700	1,263
1940–9	1,165	892	1,014	638	1,258	907	185	144	3,622	2,581

Sex Ratios

A feature of any cause of death is the distribution of the deaths between males and females—that is, the sex ratio of the deaths. This distribution is as characteristic of a particular cause of death as, for example, the age