in adults and in children, but these have not been included in the present survey. It offers a pointer to useful therapeutic measures to be adopted and we have extensively used such measures in the treatment of our cases.

Recognition of such radiological change as an aspiration segmental lesion has led us to perform further detailed investigations, and in this connexion over 200 cases have been examined by bronchography in order to study the changes in local bronchial pattern at the time of illness. The wide range of bronchial abnormality which may occur, some of which may be serious and irreversible, draws attention to the need for careful supervision and treatment of the original illness. These findings also contradict the usual view that "primary atypical pneumonia" is an essentially benign condition which resolves without serious sequelae.

This latter section of our work will form the subject of a separate publication.

Treatment

In the past no therapeutic measures were described as influencing the course of a "primary atypical pneumonia." More recently chloramphenicol and aureomycin have been widely advocated (Meiklejohn and Shragg, 1949).

When the aspiration basis for the lesions is appreciated it becomes evident that more simple measures may assist in the resolution of the pulmonary disease. Thus percussion postural drainage has been extensively used and has frequently assisted in the re-aeration and drainage of an infected segment. On occasion additional agents such as steam inhalations and short-wave diathermy have been employed.

The widely varying bacterial flora of such aspiration lesions gives some explanation for the varying response to chemotherapy and penicillin. Chloramphenicol and aureomycin can be regarded only as of use in those cases in which there is a considerable infective element due to organisms insensitive to penicillin. Their indiscriminate use as agents to combat the "virus of atypical pneumonia" must be regarded as unjustified; in any case no antibiotic can have significant effect upon the mechanical element of such segmental lesions.

Summary and Conclusion

The general features of a condition believed to be segmental aspiration pneumonia are described and a review of previous descriptions of primary atypical pneumonia is given. Comparison of the two conditions reveals no significant difference between them.

Reasons are given for doubting the viral origin of the pulmonary lesions. It is considered that they result from aspiration of products from an associated upper respiratory tract infection.

The significance of such a point of view is discussed and mention is made of possible complications and treatment.

Our experiences with this series of cases has led us to believe that the name and diagnosis "primary atypical pneumonia" is meaningless, as it is not a specific condition but merely represents a segmental aspiration pneumonia.

Without the co-operation of the medical authorities of the Royal Air Force and, in particular, Group Captain Rumball, the consultant in medicine, this work would not have been possible. We are grateful to the Director-General for permission to publish our findings. Dr. Robert Coope and Dr. P. H. Whitaker, whose clinical assistants we are, have given us every encouragement and placed the full facilities of their departments at our disposal. We are also grateful for much material assistance from the University of Liverpool.

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SERUM POTASSIUM DEFICIENCY DURING TREATMENT WITH SODIUM P.A.S. AND LIQUORICE EXTRACT

BY

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Cayley (1950) reported the occurrence of serum potassium deficiency in the course of treatment of three patients with pulmonary tuberculosis using *p*-aminosalicylic acid (P.A.S.). Heard, Campbell, Hurley, and Ferguson (1950) found evidence of this complication in 12 patients undergoing treatment with the drug.

Two patients with pulmonary tuberculosis have recently shown marked myasthenia while being treated with sodium P.A.S., and in view of the apparent rarity of the complication, judging from its absence in the numerous reports of the use of the drug, an account of it may be of interest. The implications of the metabolic changes found are examined.

Case 1

A girl aged 18 suffering from a breaking-down primary tuberculous lesion in the left upper lobe was receiving streptomycin, 0.5 g. twice daily intramuscularly, and sodium P.A.S., 4 g. five times daily by mouth, each dose containing 20 min. (1.2 ml.) of ext. glycyrrhiz. liq. *B.P.* as a flavouring agent. After 11 days' treatment and for the succeeding five days she suffered from intermittent pyrexia. She had previously been afebrile.

The tonsils showed a follicular exudate; the right tonsillar gland was found to be enlarged, and an erythematous rash appeared over the shoulders. Treatment was continued, and these signs disappeared. By the thirtieth day of treatment she complained of generalized stiffness in all limbs and of weakness and cramping pains in her legs. She remained

afebrile; her pulse rate was 80 and blood pressure 120/80. She did not appear to be overbreathing. There was marked weakness of all muscles of the neck, trunk, and limbs. Tendon reflexes were weak but could all be elicited, plantar reflexes were flexor, and no abnormality of sensation was found. Trousseau's sign was positive after 40 seconds' application of the inflated sphygmomanometer cuff, but, by contrast, Chvostek's sign was never elicited. Examination of several specimens of urine with Sulkowitch's reagent gave a normal reaction for calcium with a moderate precipitate. The serum calcium content was 8.8 mg. per 100 ml. and CO_2 combining power 85 vols.%.

Immediately after removing blood for examination she was given 10 ml. of 10% calcium gluconate, and a few minutes later Trousseau's sign was still found to be positive after three minutes. In spite of stopping sodium P.A.S. and giving oral ammonium chloride and further intravenous calcium gluconate, the muscular weakness and Trousseau's sign persisted, though becoming less marked, for 10 days. On the ninth day after onset of the weakness the serum calcium was 9.2 mg. per 100 ml., serum potassium 15 mg., and CO_2 combining power 62 vols.%. No cardiac abnormality was noted throughout, and for this reason, and because the possibility of a serum potassium deficit was not recognized until late, no electrocardiogram was made. Streptomycin was given throughout this complication and subsequently, with continued satisfactory progress.

Meanwhile Cayley's (1950) paper had appeared, and, while the changes he described might have accounted for the muscular weakness, it was not clear why such a marked Trousseau's sign had been found in our patient. The incident, however, led to fuller consideration of similar findings in another patient.

Case 2

An ex-miner aged 54 was admitted to hospital on May 12, 1950. He was known to have had pulmonary tuberculosis with tuberculous laryngitis for at least three years; and in November, 1949, he had had one testis removed for tuberculosis. He had not been treated at any time with streptomycin or P.A.S. For two months before admission he had complained of headache, particularly in the morning, that he staggered to the left on attempting to walk, and that when he tried to read "the lines ran into each other." He had noticed a sensation of "pins and needles" in the tip of his tongue and nose as well as round his mouth for about one month. His appetite had recently been poor, and he vomited from time to time.

Examination showed optic atrophy on the right due to a fractured skull 16 years previously. Anosmia was complete and taste was absent except for quinine. There was a right sixth-nerve palsy due to the old head injury. Nystagmus, maximal on looking to the left, was present, and there was slight past-pointing with the left arm, but no intention tremor or "rebound." Rombergism was not present, but on attempting to walk he staggered to the left. No other abnormality was found in the C.N.S. Cerebrospinal fluid pressure was 50 mm.; the fluid was clear and contained 42 lymphocytes per c.mm. The protein content was 80 mg., chlorides 744 mg., and sugar 64 mg. per 100 ml. The Wassermann reaction was negative (also in blood). Tubercle bacilli were not found on direct examination or on animal inoculation. The erythrocyte sedimentation rate (Westergren) was 22 mm. in the first hour. On admission the urine was acid, specific gravity 1027, and contained some albumin and pus cells. Tubercle bacilli were subsequently isolated by culture from a 24-hour specimen of urine. The albuminuria cleared up soon after admission, and was found later only when haematuria was occurring. A diagnosis of disseminated tuberculosis with cerebellar involvement was made and treatment with streptomycin, 0.5 g. twice daily intramuscularly, and sodium P.A.S., 3 g. six times daily, was begun on May 22. The P.A.S. was given in a mixture containing 20 min. (1.2 ml.) of ext. glycyrrhiz. liq. *B.P.* per dose as a flavouring agent. He had been vomiting intermittently since admission, and during the first few days of treatment lost several doses of P.A.S. in this way; no further vomiting occurred after June 5, and sodium P.A.S., 3 g. five times daily, was continued.

On June 29 he complained of numbness of the thumb and index and middle fingers of the left hand, but no objective changes were found. Tendon reflexes were all present. On July 12 he complained of stiffness and weakness in both arms, so P.A.S. therapy was stopped, but the streptomycin injections were continued.

On July 13 he was very weak and unable to sit up or feed himself; he had no difficulty in respiration or swallowing, and, objectively, power in muscles of cranial innervation, as in the legs, was good compared with that in the arms. Reflexes in the arms were weak; only the right upper abdominal reflex could be elicited, but those in the legs appeared normal. Trousseau's sign was present after two minutes' compression, but Chvostek's sign remained absent throughout. The only sensory abnormality found was depression of pain sensibility over the tips of the fingers of the left hand. The pulse rate was 84, and no abnormality was found in the cardiovascular system except that the blood pressure was slightly raised to 150/100.

As the condition showed some superficial resemblance to myasthenia gravis the patient was given 1 mg. of neostigmine hydrobromide with atropine sulphate, 1/100 gr. (0.65 mg.) intramuscularly, without any apparent effect. The serum potassium was found to be 8.5 mg. per 100 ml. (duplicate estimations). Subsequent changes in the serum potassium concentration are shown in Table I.

TABLE I.—*Serum Electrolyte and CO_2 Changes*

Date	Weight		Potassium mg./100 ml.	Calcium mg./100 ml.	Sodium mg./100 ml.	CO_2 Comb. Power Vols.%	Chlorides (as NaCl) mg./per 100 ml.
	lb.	kg.					
15/6/50			18·0				
17/6/50	116	52·6					
1/7/50	117	53·0					
8/7/50	116	52·6					
13/7/50			8·5	10·0			
15/7/50	114	51·7	7·6		323	88	454
17/7/50			8·1		324	97	
20/7/50			10·7		336	63	
21/7/50			8·1				
24/7/50			7·6			79	
25/7/50					333	75	
28/7/50			10·4			82	
29/7/50	111	50·3	15·8	9·2		78	
31/7/50			22·1			79	
2/8/50			21·8			73	
3/8/50				10·5			
6/8/50	106	48·1					

On July 14 he developed marked haematuria. This persisted for several days, and was thought to be due to renal tuberculosis, as tubercle bacilli had been identified in his urine on June 9. The haemorrhage may have been precipitated by hypoprothrombinaemia, as this constantly follows the administration of P.A.S.

On July 15, in view of the persistent Trousseau's sign and raised CO_2 combining power, he was given ammonium chloride, 1 g. two-hourly by mouth during waking hours. This was continued until August 6.

On July 19 he developed prolapse of the rectum, probably due to weakness of the muscles of the pelvic floor. At this time he could raise his hands to feed himself, though power and reflexes in the legs were weaker than previously. From July 26 to August 1 he was given potassium chloride, 3 g. four times daily by mouth. On July 31, when the serum potassium had risen to 22.1 mg. per 100 ml., power in all limbs was much improved, reflexes were more readily elicited, and Trousseau's sign was positive only after four minutes' compression. Within a further two days the myasthenia had entirely disappeared, though the paraesthesiae in the left hand persisted without objective sensory

changes. Throughout the incident muscle and reflex weakness had been more marked in the arms and trunk than in the legs, whereas muscles of cranial innervation were conspicuously unaffected.

Electrical reactions in the right quadriceps and right forearm flexor groups of muscles were examined almost daily from July 22 to August 15, using a Ritchie–Sneath stimulator. The results are summarized in Table II. The

TABLE II.—*Muscle Electrical Thesholds*

Muscle Group:	Right Quadriceps				Right Forearm Flexors			
Pulse length (millisecs.)	100	10	1	0·1	100	10	1	0·1
Hypokalaemic phase:								
Mean threshold voltage	34·6	35	49·4	51·6	26·75	28·0	43·0	45·0
Range of threshold voltage	32–7	32–8	40–64	42–64	22–30	22–30	35–50	35–50
No. of observations	5				4			
Standard deviation	2·55	3·0	10·85	10·74	4·02	4·02	6·28	7·08
After recovery:								
Mean threshold voltage	30·5	30·7	41·3	43·3	26·5	26·5	35·2	36·1
Range of threshold voltage	28–35	30–5	35–48	36–48	22–8	22–8	25–42	25–48
No. of observations	10							
Standard deviation	1·84	1·63	4·68	3·68	2·54	2·54	5·55	6·72
Difference of means	4·1	4·3	8·1	8·3	0·25	1·50	7·8	8·9
Standard error of difference	1·28	1·43	5·1	4·94	2·22	2·22	3·2	3·6

In the case of the quadriceps, when the pulses were of 100 and 10 m.secs. duration the observed differences were more than three times their standard error, whereas with pulses of 1 and 0·1 m.sec. the differences were less than twice their standard error. For the forearm flexor group the reverse is true, and only with the shorter pulses of 1 and 0·1 m.sec. were the differences significant (2·4 times their standard error).

voltage required to elicit a contraction at a given pulse length, frequency remaining constant at approximately five per second, is expressed as the mean of daily observations made, first, when the muscle weakness was present, and subsequently when muscle power was normal.

It will be seen that after muscle power returned to normal the voltage required to elicit a contraction at all pulse lengths examined was diminished, and, further, that in the thigh muscles the difference was more striking with the longer pulses, and in the arm with the shorter pulses. This may be an expression of the different behaviour of muscles in various parts of the body as a result of potassium deficiency, which was evident on clinical examination and which is shown to be capable of measurement.

The patient's condition in December, 1950, was satisfactory in that he showed none of the earlier evidence of cerebellar disorder, he had put on weight, and tuberculous activity elsewhere was apparently in remission.

Discussion

In Case 1 the markedly positive Trousseau's sign with the complaint of stiffness and cramps led to the conclusion that the weakness was the result of tetany, though the absence of Chvostek's sign and the normal urinary excretion of calcium raised doubts about this. The serum calcium, however, was found to be in the lower range of normal (8.8 mg. per 100 ml.); this is above the usual renal threshold for calcium, and is in accord with the positive Sulkowitch reaction in the urine. The high CO_2 combining power (85 vols.%), together with the slightly lowered serum calcium, was thought to account for the presence of Trousseau's sign, and only when this sign and the muscle weakness failed to respond to the administration of ammonium chloride and calcium gluconate was the possibility of potassium deficiency considered. By this time the serum potassium was 15 mg. per 100 ml.

The experience with Case 1 led to early estimation of serum potassium in Case 2, and an E.C.G. (see Fig.) was recorded (June 6) sixteen days after starting sodium P.A.S. therapy. It will be noted that this was already abnormal in form. Once more the onset of weakness with paraesthesiae in the fingers suggested tetany, and Trousseau's sign was found positive, though again Chvostek's sign remained absent throughout. These signs persisted until the serum potassium returned to normal after the administration of potassium chloride.

It seems clear that the muscle weakness was related to the low serum potassium, as in the second patient both findings were corrected simultaneously by giving potassium chloride. Why this potassium deficit should have occurred with the administration of sodium P.A.S. in these two patients while it has not been observed in others is difficult to explain. Borst *et al.* (1950) described a liquorice extract with deoxycortone-like action. This in retrospect seemed to offer a possible explanation, as both of the patients reported had been taking sodium P.A.S. in a mixture containing 20 min. (1.2 ml.) of ext. glycyrrhiz. liq. *B.P.* per dose; and Cayley (1950, personal communication) has stated that his three patients had all had liquorice with their P.A.S. In some respects the metabolic changes induced resemble those produced by deoxycortone (D.C.A.). The serum potassium was lowered, blood pressure was raised, and withdrawal of sodium P.A.S. was followed by a diuresis, loss of weight, and lowering of blood pressure. The serum sodium, however, was never found to be abnormal, but plasma chlorides were low on the single occasion when they were measured.

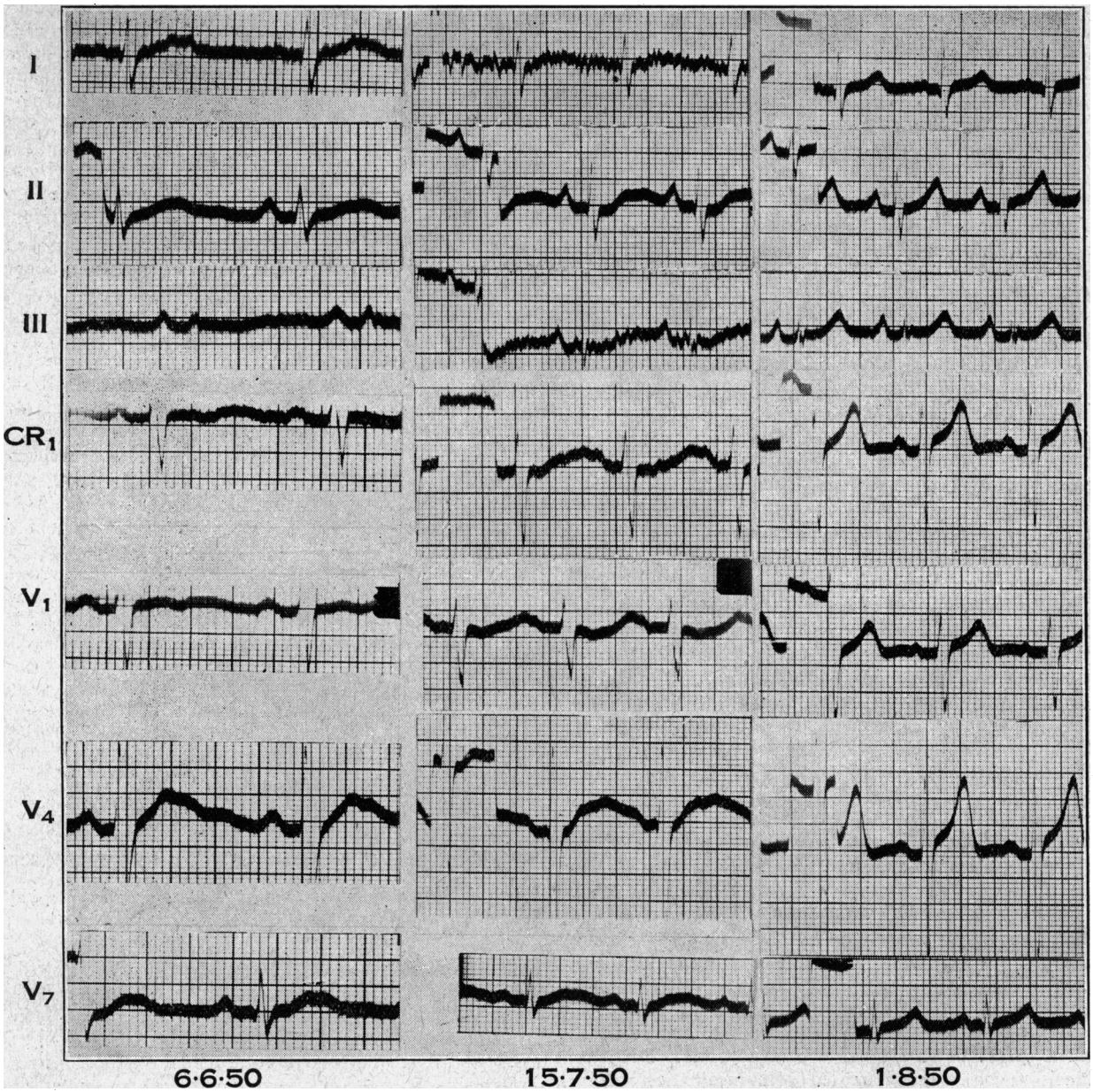
Campbell and Neufeld (1951) attributed the hypokalaemia developing during P.A.S. therapy to an impurity in certain commercial preparations of P.A.S. They found that ext. glycyrrhiz. liq., given in four 1-drachm (3.5-ml.) doses daily with P.A.S., caused a slight lowering of the serum potassium concentration, and that the lowest levels were encountered when impure potassium P.A.S. was administered with liquorice.

If, then, liquorice was responsible for the low serum potassium and the related symptoms, it seems surprising that no other cases than those mentioned have been reported, as liquorice was widely used as a flavouring agent for P.A.S. and other drugs. Individual idiosyncrasy may be an important factor, and, as Borst *et al.* (1950) further point out, not all preparations of liquorice appear to possess this property, which may be related to their origin or to conditions of preparation and storage. P.A.S. is now commonly sold in cachets, so the need for a flavouring agent no longer exists.

It is of particular interest that Groen, Pelser, Willebrands, and Kamminga (1951) have reported satisfactory control of two patients with Addison's disease using liquorice extracts.

Cates (1949) has reported on the occurrence of oedema and potassium loss in combined sodium *p*-aminohippurate and penicillin therapy during the treatment of a case of subacute bacterial endocarditis; he was, however, giving the equivalent of 25.5 g. of sodium by intravenous infusion daily, whereas the two cases here described were having 15 g. of sodium P.A.S. daily, which would contain approximately 2 g. of sodium.

Fluid retention could scarcely be attributed to such a small increment of the sodium intake, nor is it likely to have been responsible for the gross serum potassium deficit.



The most striking changes in the records of June 6 and July 15 are in the prolongation and flattening of the T waves in all leads. The corrected Q–T interval (Ashman and Hull, 1941) was 0.47 sec. on June 6; this had increased to approximately 0.60 sec. on July 15, and had diminished to 0.425 sec. on August 1. Owing to superimposition of P waves on T waves in the record of July 15 it is impossible to measure the Q–T interval precisely, and the figure should therefore perhaps be even higher than that given. The E.C.G. abnormalities found on June 6 evidently occurred before the serum potassium was lowered, as the latter was normal on June 15. Records taken later were identical with those made on August 1 and it is assumed that they represented the normal for the individual. (The normal QTC in the adult male may extend to 0.422 sec.)

Trousseau's Sign

The serum and urinary calcium were normal whenever examined and cannot therefore have accounted for the presence of this sign, unless the plasma protein concentration had been grossly raised.

The relation of Trousseau's sign to hypocalcaemia and hypopotassaemia has been examined in detail in two cases of malabsorption syndrome by Engel, Martin, and Taylor (1949), who found that raising the serum potassium while the calcium content remained low regularly precipitated overt tetany, which could then be abolished by the infusion of calcium. This is in contrast to Case 2, in which Trousseau's sign disappeared when the serum potassium was restored to normal, and it is more likely, therefore, that a change in *p*H was the factor concerned rather than purely a change in the serum calcium:potassium ratio. Lowering of the serum potassium with a normal or raised plasma *p*H would be expected to diminish the tendency for tetany to occur (Lehmann, 1937).

Engel *et al.* (1949) suggest that anoxia, by releasing potassium from cells, may lower the threshold to conduction in the limb below the pressure cuff, and thus convert latent to overt tetany. This would provide an explanation for the absence of Chvostek's sign when Trousseau's phenomenon could be readily elicited. In the presence of a normal serum calcium Trousseau's sign probably occurs only if the plasma *p*H is raised. In the cases described the CO_2 combining power was either raised or in the upper range of normality while this sign

was present. Had the plasma *p*H 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 examp'e, 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.