Among the precipitating factors were change of surroundings—as removal to hospital—and restricted diet.

The blood bromide was over 200 mg. per 100 c.cm., as compared with the normal level of 2.5 mg.

Treatment is by sodium chloride, 30 to 60 gr., repeatedly by mouth, or intravenous saline infusion; a favourable response, with lowering of the blood bromide, confirms the diagnosis.

I wish to acknowledge with thanks the permission of many colleagues at Manchester Royal Infirmary to publish cases under their care, and to express gratitude to my colleague, Dr. M. C. G. Israëls, for his helpful criticism and advice.

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INDUCTOTHERM TREATMENT OF SULPHAPYRIDINE ANURIA

BY

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Disturbance of renal function developing during treatment with the sulphonamide series of drugs has been described in a considerable number of cases by various authors. Clinically this disturbance may range from transient haematuria to anuria with resulting death from uraemia. The pathological changes in the urinary tract have been shown by experiments on animals (Antopol and Robinson, 1939; Gross, Cooper, and Lewis, 1939) and by post-mortem examinations in fatal cases (Backhouse, 1939; Stryker, 1940; Pepper and Horack, 1940; Plummer and McLellan, 1940) to be due to the precipitation of crystalline derivatives of the sulphonamides in the renal tubules and pelves, and in the ureters. It has been pointed out that owing to the greater solubility of sulphapyridine and its acetyl derivatives in an alkaline urine the possibility of this condition arising may be minimized by the administration of alkalis and abundant fluids to cases being treated with this drug (Lebel, Schroeder, and Simesen, 1940).

In cases in which this complication has resulted in anuria the most successful therapeutic measure, apart from the administration of abundant fluids by mouth and of intravenous solutions of non-threshold salts (Leedham-Green, 1941), has been catheterization and lavage of the renal pelves (Tsao *et al.*, 1939; Lebel, Schroeder, and Simesen, 1940; Scurry and Wittenborg, 1940). Recently we have had under our care such a case in which inductotherm therapy of the kidneys has yielded a satisfactory result.

Case Report

An airman aged 36 was admitted to a hospital for venereal diseases on Aug. 25, 1941, suffering from a urethral discharge. Treatment with sulphapyridine was started on the 26th, when 8 g. was given. On the 27th a further 7 g. was administered. That evening at 8 p.m. he passed a small quantity of brown urine. Sulphapyridine therapy was discontinued. Thereafter, although a feeling of urgency remained, he was unable to pass urine until 11 a.m. on the 28th, when he voided 1 oz. of deeply blood-stained urine. An hour later he developed intense aching pain in both loins radiating to the testicles, more severe on the left side. The pain rapidly became more intense and the sense of urgency increased. Intractable vomiting set in at about 3 p.m. No further urine was passed, and at 10 a.m. on the 29th he was catheterized. No urine was obtained. The patient was then transferred to this hospital. On admission he was vomiting, and complained of very intense pain in the loins. He was afebrile; the tongue was dry and coated; and there was marked tenderness in both renal angles. Routine examination revealed

no other abnormality. Blood pressure was 135/85. Fluids were given in large quantities, but absorption was minimal owing to the vomiting.

At 3.20 p.m. inductotherm treatment was started. The apparatus used was the General Electric Corporation's inductotherm, model B. Treatment was given to both kidneys alternately. Two coils were used behind and three in front. The immediate progress of the case is illustrated in the chart.

It will be noted that the passage of 6 to 8 oz. of urine followed about an hour after the termination of each of the first three applications, and that during the night of Aug. 29-30 the passage of urine, though in small amounts, indicated the return of renal function. 12 oz. of urine was passed after the final application; thereafter diuresis set in. The first specimen of urine was heavily blood-stained and acid in reaction. Unfortunately it was disposed of before it had been examined microscopically. The second specimen was "smoky" in colour and acid in reaction, and contained red blood cells in profusion. No crystals were seen. Subsequent specimens were clear, and red blood cells had disappeared from the urine by the 31st. The passage of urine after the first application was followed by marked relief of pain, which ceased shortly after the last application. By the evening of Aug. 31 vomiting had subsided. Thereafter convalescence was rapid and uneventful.

We are indebted to Major J. Bull, R.A.M.C., for the following report on subsequent investigation of the case by excretion urography.

Radiological Investigation

Sept. 4.—The kidneys were normal in size, shape, and position. Excretion urography (20 c.cm. of pyelactan intravenously) showed the right kidney excreting with fair concentration into the pelvis and ureter after 5 minutes. The calices were not seen throughout the examination. Excretion from the left kidney was abnormal. Not until 20 minutes was it seen to be filling with dye. It was assuming a white appearance. At 90 minutes the whiteness was very pronounced, but at no time were the calices, pelvis, or ureter outlined with dye. The right renal pelvis still contained dye. On no occasion was compression applied to the ureters. The examination was not pursued after 90 minutes.

Sept. 15.—A second intravenous pyelography (20 c.cm. of pyelactan) was now carried out. After 5 minutes the right calices, pelvis, and ureter were excreting the dye in fair concentration. The left calices and pelvis contained rather less dye. Compression of the ureters was applied for from 7 to 30 minutes. Just before releasing compression a film showed that there was a moderate concentration of dye in both calices, pelves, and ureters. Both kidneys were white. Radiographs taken at 3 hours, 6 hours 40 minutes, 60 hours, and 84 hours showed the kidneys to be partially white, the intensity decreasing with time.

Radiological Conclusions

At the first examination on Sept. 4 the right kidney was excreting moderately. The left renal apparatus at no time showed excretion into the calices, pelvis, or ureter. The kidney itself, however, became homogeneously white, indicating that the dye had entered it. The conclusion is that the dye was being held up proximal to the calices. Thus the excretory mechanism of the left kidney was functioning very poorly indeed, whereas that of the right was moderate. At the re-examination after 11 days the calices of both kidneys contained the dye even before ureteric compression was applied. Compression improved the concentration. Both kidneys were partially white, indicating that the dye was being held up to some extent in the

proximal portion of the excretory apparatus. Even after 84 hours the kidneys cast an abnormally dense shadow, indicating that some of the dye was still being retained.

Volhard's test was carried out on Sept. 19, with the following result: Specific gravity of urine during 24 hours with forced fluids: minimum reading 1000. Specific gravity of urine during 24 hours without fluids: maximum reading 1028.

Discussion

Prominent in the consideration of the treatment of this case was the assumption that the renal tubules were blocked with sulphapyridine crystals. With the aim of promoting active hyperaemia of the glomeruli and increasing secretion it was decided to employ inductotherm (short-wave) treatment. This method of heating deep tissues, the most efficient in use, has been applied previously in cases of renal disease (Stark, 1939), but we have been unable to find any reference to its employment in anuria resulting from the use of the sulphonamide series of drugs. The results in this case indicate that it may be of considerable value in the treatment of cases in which the condition is due primarily to mechanical obstruction of the renal tubules. It is unlikely that this treatment would be equally effective when glomerular damage is present.

It would appear that in cases with oliguria or anuria arising from the use of the sulphonamides this method of treatment is worthy of trial before submitting the patient to ureteric catheterization and lavage of the renal pelvis, a procedure which may be inadvisable when an infection of the urinary tract is present. Catheterization will of course be required when the ureteric lumen is completely occluded.

The x-ray findings in this case indicate the presence of intrarenal changes, gradually diminishing during convalescence. It is therefore apparent that, even though ureteric catheterization may have to be carried out, short-wave therapy may be a useful adjunct in clearing the renal tubules to the greatest extent at the earliest opportunity.

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CEREBROSPINAL FLUID IN ACUTE ANTERIOR POLIOMYELITIS

BY

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Paralysis in infancy does not constitute infantile paralysis (acute anterior poliomyelitis), and we need to be in a position to diagnose this, "the saddest of diseases," when we are confronted with paralysis in childhood. The nature of the paralysis and the tenderness of the muscles are characteristic features in this disease, in which the diagnostic picture with its pre-paralytic stage and "back signs" can be very typical. Many cases, however, are atypical; how are we to diagnose them?

The Literature on Diagnosis

H. Howton Merritt and Frank Fremont-Smith (1937) state that before the onset of paralysis the diagnosis of anterior poliomyelitis cannot be made with certainty on the basis of the examination of the cerebrospinal fluid because numerous diseases of children and young adults are accompanied by similar cerebrospinal fluid changes. Fortunately, about the time of the onset of the paralysis changes begin to occur in the cerebrospinal fluid which are very characteristic, but these can only be fully appreciated if two or three specimens are obtained during

the first three weeks of the illness. These changes are: (1) an early rise in cell count, falling to normal by about the end of the second week of the illness; (2) a protein content which is almost normal until some days after the onset of paralysis, and which rises to a maximum in the third week. These very characteristic changes were first demonstrated by Peabody, Draper, and Dochez in 1912, and have often been confirmed since then.

As to the nature of the cells which cause the early rise, Howton Merritt and Fremont-Smith state that 80% of their cases showed a lymphocytic predominance within five days of the onset of the paralysis, although the early counts might consist largely of polymorphonuclear cells. Similarly, Peabody, Draper, and Dochez state that "polymorphonuclear cells, except in small numbers and probably due for the most part to a slight admixture of blood, were, except in a very few instances, absent after the first week of the disease."

This glance at the literature gives us a clear idea of the possibilities and limitations of the findings in the cerebrospinal fluid as an aid to diagnosis.

Findings in a Series of Seven Cases

This short paper is designed merely to stress the importance of these known facts by reporting the findings in seven cases of infantile paralysis admitted to the Bristol Children's Hospital during a small epidemic which occurred in the autumn of 1938. It was found that the cerebrospinal fluid had been examined on various occasions between the fifth and twenty-first days of the illness. The paralyzes had in every case developed between the second and seventh days, and the fluid was in all cases examined during the paralytic stage.

In those cases in which the fluid was examined within the first twelve days the cell counts were high, reaching 50 in two cases, while in those examined after the second week of the

Chart showing the cerebrospinal fluid findings in the seven cases described. o = Cell count. • = Protein content. (Cerebrospinal fluid from A.P. examined on two occasions.)

illness the cells never exceeded 6. No fluid was examined before the fifth day of the illness, which explains why no very high cell counts were recorded.

The protein content was below normal in two fluids examined on the seventh and tenth days, but there was one specimen on the fifth day which gave a reading of 70 mg. per 100 c.c.m. Three estimations of 300 mg. and one of 200 mg. were, however, obtained during the third week, revealing the magnitude of this late protein rise. On the twenty-first day a specimen showed a cell count of 5 and a protein content of 70 mg., suggesting that at this stage the protein content was also on the wane.

One case was subjected to two lumbar punctures, and the cerebrospinal fluid findings were: on the seventh day—cells 50, protein 80 mg. per 100 c.c.m.; on the fifteenth day—cells 5, protein 200 mg. Such findings are diagnostic of acute anterior poliomyelitis.

It must be noted that a single specimen of fluid taken towards the end of the second week may give a reading which will show little that is abnormal, because the cell count will