

looking up such books and articles on the subject that lay at hand. As already stated, Walsham and Spencer<sup>1</sup> mention "external violence" as a cause of intussusception. Adams,<sup>2</sup> in a review of 100 cases admitted to St. Thomas's Hospital, states that there was a history of injury in only one case. Thompson and Miles<sup>3</sup> make no mention of trauma as a cause of intussusception. Warren<sup>4</sup> does not mention it. Miles,<sup>5</sup> writing in "Choyce's System of Surgery," does not mention it. Walton<sup>6</sup> makes no mention of it. Souttar<sup>7</sup> quotes Elliot and Corscaden as giving trauma as a definite cause in a few cases.

It appears likely that trauma may cause an intussusception in two ways. Firstly, it may cause a hæmorrhage into the gut wall, which is then caught and forced onwards in the same way as an adenoma or an inverted Meckel's diverticulum. That hæmorrhage into the gut wall may cause an intussusception has been shown by Lett,<sup>8</sup> Sutherland,<sup>9</sup> and Collinson,<sup>10</sup> who reported cases caused by hæmorrhage in Henoch's purpura. The hæmorrhage caused by trauma is, however, more likely to be under the peritoneal coat, where it would not easily lead to intussusception. Secondly, intussusception may perhaps originate in a temporary local paralysis of gut caused by violence applied through the abdominal wall. Walton<sup>6</sup> quotes the experiments of Nothnagel in which he induced intussusception by electrical stimulus, "invaginatio spasmodica," and by inducing local paralysis by crushing, "invaginatio paralytica." As clinical illustrations of "invaginatio paralytica," Walton quotes a case following a Richter's hernia and a case reported by Sherren,<sup>11</sup> in which a gangrenous portion of the transverse colon acted in the same way.

It seems not unreasonable to suppose that violence applied through the abdominal wall may occasionally crush a small portion of gut so severely as to cause a temporary local paralysis and yet leave no obvious sign of injury. Such is, I believe, the explanation of the case reported here and, perhaps, of other cases where a less convincing history of trauma is given.

**References.**—1. Walsham and Spencer: Theory and Practice of Surgery, 1903. 2. Adams and Cassidy: Acute Abdominal Diseases, 1913. 3. Thompson and Miles: Manual of Surgery, 1909. 4. Warren: A Text-book of Surgery, vol. ii., 1915. 5. Miles: A System of Surgery by Choyce, vol. ii., 1912. 6. Walton: The Practitioner, August, 1911. 7. Souttar: The London Hospital Gazette, June, 1911. 8. Lett: THE LANCET, Feb. 20th, 1909. 9. Sutherland: Pediatrics, vol. ix., 1896. 10. Collinson: THE LANCET, March 12th, 1910. 11. Sherren: Clinical Journal, 1906.

Plymouth.

## A CASE OF SYPHILITIC NEPHRITIS.

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THE following case of acute nephritis complicating secondary syphilis seems worthy of record, since the response to specific treatment was unusually rapid and complete.

H. M., an Egyptian tram-conductor, aged 20, was admitted to Kasr-el-Aini Hospital on Dec. 6th, 1919, for nephritis. He stated that a fortnight previously he had begun to suffer from loss of appetite with occasional vomiting, and that a week later œdema had appeared, first in the scrotum and then in the face and whole body. On admission he presented the usual signs of nephritis. There was extensive œdema, but no free fluid in the serous sacs; the urine averaged 1000 c.cm. per diem (vide chart), had a sp. gr. 1015, and contained a considerable amount of albumin (13 per 1000 Esbach). Microscopically, there were numerous casts, granular and leucocytic, as well as bilharzia ova, with attendant blood and pus corpuscles. Over the patient's body were scattered papules of a deep coppery hue. There were snail-track ulcers on the tonsils and a barely healed hard chancre on the penis. Blood examination gave a positive Wassermann reaction. The patient admitted noticing the chancre about two months before, but had taken no medical treatment, and was anxious to conceal his disease.

Treatment consisted of intravenous injections of novarsenobillon, 0.45 g. being given on the fifth and a similar dose on the twelfth day after admission. The result was immediate and very striking. Within a few hours of the first injection the urinary flow increased four-fold and the diuresis with frequent micturition continued as shown on the chart.

The œdema quickly subsided and inside a week the albumin had diminished to 1 part per 1000. Two days after the second dose of novarsenobillon, the patient had an attack of fever lasting three days which caused a temporary fall in the diuresis but did not increase the albuminuria which had so rapidly declined. A fortnight after the treatment had begun the patient insisted on taking his discharge as he felt perfectly well and could not afford to lose his employment. At this time the urine measured 2500 c.cm. per diem,

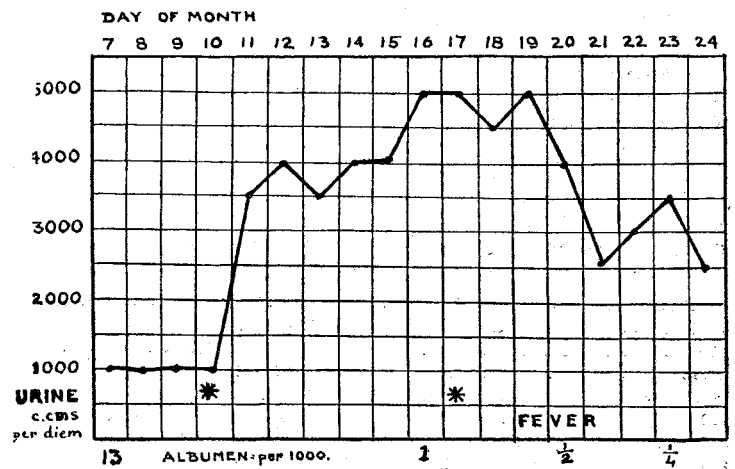


Chart showing quantity of urine passed per day in case of syphilitic nephritis. \* Indicates intravenous injection of 0.45 gm. novarsenobillon.

had a sp. gr. 1013, and contained but a trace of albumin (under  $\frac{1}{2}$  per 1000) with scanty granular casts. The rash had largely faded, but no effect on the bilharziasis was apparent; ova were plentiful and hatched as freely as on admission.

The diuresis appeared to be entirely derived from the retention of fluid in the body. For within the space of 12 days the patient lost 20 kilos. in weight (over 3 stone)! This amount—20 litres—closely corresponds to the excess of urine passed if we allow 2 litres a day of urine as the normal maximum output.

The immediate effect of salvarsan therapy on this patient was so striking that there can be little doubt that the nephritis was entirely syphilitic in origin. As in the majority of cases reported, nephritis appeared in the early secondary stage, about three months after infection. I attribute the success of treatment (so far as it could be carried out) to the early use of adequate doses of the salvarsan compound instead of mercury. It seems improbable that a nephritis appearing during specific treatment of syphilis would respond so favourably as this case where no previous treatment had been received.

## PERICARDIAL EFFUSION FOLLOWING THE INJECTION OF ANTIDIPHTHERITIC SERUM.

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THIS case is of sufficient interest to be worth recording.

The patient, a boy, aged 10, presenting clinical and bacteriological evidence of diphtheria, was given on the fourth day of the disease an injection of 7000 units of serum. His temperature was then 101° F. The cardiac physical signs were normal. Thirty-six hours later the temperature rose to 103°. The fauces had cleared, but there had developed well-marked signs of a pericardial effusion; obvious præcordial bulging; dullness extending beyond the apex beat to the line of the anterior axillary fold. Breath sounds absent over the front of the upper lobe of the left lung. There was no pericardial friction audible at any time in the illness. The pulse-rate was 120, and its quality good and rhythm regular.

Dr. W. W. Jameson (medical officer of health for Finchley), to whom I showed the case as a curiosity, agreed in the interpretation of the signs, which suggested a passive effusion into the pericardial sac rather than an inflammatory pericarditis.

The signs of fluid gradually diminished and disappeared after ten days. The diphtheria itself proved a mild attack, and cleared up without any further complications. There was no serum rash.

Dr. Jameson informs me that he has been unable to trace in the literature any record of a similar occurrence following the injection of serum.

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