A CASE OF ECLAMPSIA TREATED WITH SPINAL ANAESTHESIA.*

BY

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RUGBY.

On April 29th, 1929, shortly after 8 p.m., I was called to assist my colleague Dr. Waugh in the treatment of a case of eclampsia. I suggested that the administration of chloroform, if it was not already in progress, should be started at once. On my arrival I found a young, massive woman lying on her side in bed. She was almost fully dressed, was under the influence of chloroform, comatose, and breathing stertorously. The room was dismantled for spring cleaning.

The patient, aged 22, a primigravida seven months pregnant, had been quite well till half-past seven that evening. She had been feeling sick occasionally, but had regarded this symptom as unimportant. At the ante-natal clinic, where she had attended once a month, her condition was considered to be good. She ate a great deal, and was habitually constipated. At seven o'clock she had gone upstairs to help in the spring cleaning of the bedroom where she was lying. She knelt down to clean a chair, and said that she felt sick. She was told to sit down, and was given a glass of water. She took a sip of water and was helped into the bed. No sooner was she on the bed than she had a fit, and as the fit or fits continued and her condition became alarming, Dr. Waugh, who had never seen the patient before, was sent for. He immediately injected a quarter of a grain of morphine (8.15 p.m.) and sent for me. The administration of chloroform was started at about 8.30.

As soon as I saw the patient I made preparations to induce spinal anaesthesia. To expose the patient's back her combinations were cut, a low corset being removed. With due precautions I made a lumbar puncture, drained off about two drachms of cerebro-spinal fluid, which was apparently under normal pressure, and then slowly injected 1.8 c.cm. of tropacocaine. This ended the treatment. The administration of chloroform was stopped; no vaginal examination was made, and not even a catheter was passed to collect urine.

It was now nearly 9 o'clock; the patient lay still, and her respirations were regular, loud, gurgling, and at the rate of about 24 a minute; the pulse, which previously had been about 72 to 80 in rate and bounding in quality, soon began to quicken and lose its volume; its rate increased to 104, and at times it could scarcely be felt at the wrist. On auscultation of the chest I could clearly hear the signs of fluid in the bronchi, and I thought that pulmonary oedema had occurred. For these reasons the foot of the bed was raised. Shortly before 10 o'clock the clinical picture changed. The patient turned her head, grimaced, and moved her arms as though to wipe her face. Mucopus which had collected about the mouth and nostrils was wiped away, and the patient settled on her side, apparently in sleep. The breathing was now quiet, and on auscultating the chest I found that respiration was normal and the mucous rales had disappeared.

I left soon after 10 o'clock, but when at 11 o'clock I returned, the nurse, who had arrived during the interval, informed me that the patient had had another, very slight, fit, which had lasted only three minutes, after which she had once more become comatose. I found the patient semi-conscious, but at moments restless and moving about. I gave instructions that she was to be kept on her side; that if the bowels did not move naturally an enema was to be given, with the patient still on her side; and that if she became conscious she was to have nothing but water by the mouth.

The next morning (April 30th) Dr. Waugh and I saw the patient again; she was completely conscious, and had passed a restless night, but no further fit had occurred. She had complained to the nurse of "uterine pain." The night report was as follows:

11.10 p.m. Temperature 97.4°; pulse 130.
11.40 p.m. Simple enema given, not retained; patient very restless.
12.30 a.m. Vomited.
1.0 a.m. Vomited.
1.40 a.m. Vomited again.
2.40 a.m. Regained consciousness; passed urine, 20 ounces.
3.0 a.m. Temperature 97.2°; pulse 104. Sips of water given.
3.15 a.m. Soap enema given; very good but very constipated result.
9.0 a.m. Temperature 98°; pulse 84.

Thus the patient had vomited, had had an action of the bowels, and had passed plenty of urine. The pulse and breathing were normal, and the blood pressure was 110/65. The patient was still a little confused, and had no knowledge of what had happened. On examination we found that the uterus was enlarged to the

size of a seven months' pregnancy, and we heard the foetal heart. There had been no vaginal discharge, and a vaginal examination was not made. No urine had been kept for analysis. We allowed the patient nothing but water with sodium bicarbonate, ordered another enema, and forbade visitors.

From that day the patient's condition steadily improved; she had no more fits, and various symptoms, such as backache, headache, and buzzing in the head, gradually disappeared. She was allowed up on May 7th, and on May 15th, when she visited the ante-natal clinic, her condition was found to be good, and the urine, though alkaline, was normal. She had her baby, a male weighing 7½ lb., on June 20th, and Dr. Waugh, who attended, reported that delivery was normal. I saw the patient on June 23rd; she was smiling and well. Her blood pressure was 120/80, and a catheter specimen of urine was normal. It is noteworthy that albumin was found once only (May 1st), and in much smaller amount than was expected. Whether it was present at the time of the attack (April 29th) or the next day cannot be stated. Unfortunately, the child died at the end of a week, but the mother was discharged well on July 3rd.

I wish to express my indebtedness to Dr. Waugh for giving me the opportunity of treating this case.

THE SIGNIFICANCE OF OXALURIA.

BY

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IN the routine microscopical examination of urinary deposits it is not uncommon to find octahedral crystals of calcium oxalate. Apart from the formation of calculi, this oxaluria is not considered by most modern authorities to be of clinical importance. It is said that the great majority of persons passing even large numbers of individual oxalate crystals suffer no inconvenience. This absence of urinary symptoms is all the more remarkable when we consider the known effects of oxalic acid and soluble oxalates on the kidneys. Numerous workers have reported that these substances, introduced either orally or subcutaneously into animals, produce tubular nephritis, and Shaw Dunn and his co-workers¹ have shown that in rabbits, guinea-pigs, and dogs the intravenous administration of oxalates causes an intense nephritis, which primarily attacks the first convoluted tubules, and which for some months seriously affects the power of the kidney to excrete urea. It is interesting to note that they conclude that the deposition of calcium oxalate in the tubules plays no important part in this process. In man the ingestion of oxalates is sometimes followed by urinary symptoms.

During the past five years four patients suffering from oxalic acid poisoning have been admitted into the Royal Infirmary at Leicester, and in the only fatal case there was suppression of urine. In 1915 three anonymous correspondents in the *Lancet*² reported symptoms observed in themselves after taking rhubarb, which were attributed to the oxalic acid in the vegetable. Of these, one had dysuria and blood in the urine, one had dysuria alone, and one had no urinary symptoms.

There is, in fact, a considerable amount of evidence that oxalates in a state of solution are highly toxic to the kidney, and it would appear that the absence of serious symptoms in individuals passing calcium oxalate is dependent on the extreme insolubility of the lime salt. It was formerly believed that calcium oxalate crystals only separated out after the urine had stood for some time, being held in solution by acid sodium phosphate. That this is not the case is shown by the frequent finding of oxalate crystals in freshly passed acid urine, and by the entire absence of impaired renal function in these cases.

Presumably, then, calcium oxalate is sometimes excreted as such by the kidney. It would nevertheless be surprising if these sharp, hard crystals should pass over the delicate renal tissues without causing some disturbance, and that mechanical injury can be caused in this way the following case appears to show.

A boy, aged 15, consulted me on August 1st, 1929, for haematuria. He had been seen eighteen months previously for liliun tuberculosis, but six months' sanatorium treatment had apparently completely cured this condition. The haematuria of which he now complained had had a sudden onset a few days

* Dr. Waugh reported this case briefly in the *British Medical Journal* of October 12th, 1929, p. 668.

beforehand; it was profuse and quite painless. He maintained that he felt quite well, and had absolutely no symptoms other than blood in the urine. A thorough examination revealed no abnormal physical signs. The urine was bright red, and a microscopic examination revealed, in addition to large numbers of red blood cells and a very occasional blood cast, the presence of many octahedral "envelope" crystals of a size rather larger than those usually seen in urine. He was put to bed, and given a milk diet with alkalis. On the following day there was much less blood in the urine, but the crystals were even more abundant. On the next day there was neither blood nor albumin in the urine, and only an occasional crystal was seen. The systolic blood pressure was 125 mm. of mercury, the diastolic 70. The blood urea was 23 mg. per 100 c.cm. No tubercle bacilli were found in the urine, and an x-ray examination showed no stone in the urinary tract. A week later there was nothing abnormal in the urine, and the boy has remained well since.

This patient had always had a fair amount of rhubarb in the summer, and during the past season had had more than usual. However, none had been taken for a fortnight before the onset of haematuria.

The Nature of the Crystals.

Although these crystals were of the octahedral form typical of calcium oxalate, it was thought advisable to investigate their chemical characteristics. So long ago as 1862 Neubauer and Vogel³ pointed out that very similar forms could be obtained by crystallizing out sodium chloride from a solution containing urea. Another possible mistake is the confusion of an unusual form of ammonio-phosphate of magnesia with calcium oxalate. A specimen of urine containing the crystals was centrifugalized, the deposit was shaken up in a tubeful of distilled water, and this was again centrifugalized. The process was repeated, and the crystals were found to be still undissolved; therefore they were not sodium chloride. They were then similarly washed in dilute acetic acid, but remained intact, so that they were not triple phosphate. They were, however, soluble in hydrochloric acid, but were recovered from this solution on neutralizing it with lime water. Thus there can be no reasonable doubt that they were truly crystals of oxalate of lime.

It would be foolish to generalize from a single case, but the history of this patient is, I think, suggestive. It suggests, first, that the passage of calcium oxalate crystals is not always as innocuous as supposed, and that some cases of unexplained spontaneous haematuria may be due to a similar cause; secondly, although calcium oxalate can cause some mechanical damage to the kidney, the absence of symptoms, the rapid recovery, the normal blood pressure, and particularly the normal blood urea, of this patient show that it is the safest form in which the body can excrete any abnormal accumulation of oxalates in its substance.

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- ² *Lancet*, 1915, i, 1006, 1110, and 1164.
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Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

ACUTE APPENDICITIS COMPLICATED WITH PARATYPHOID FEVER.

THE association of the above two diseases seems of sufficient interest to warrant recording.

A healthy-looking adult, aged 22, was admitted to hospital complaining of a sharp bout of pain in the right lower quadrant of the abdomen. His temperature was 100.8° F., and pulse 80 per minute. Tenderness was present over the appendix area and there was some muscular rigidity, more marked on the right side. Tenderness was elicited on the right side when he was examined per rectum. He had vomited once. Acute appendicitis was diagnosed and he was operated upon. The abdomen was opened by a gridiron incision. Some free peritoneal fluid was found. The appendix was long, thick, tortuous, and very much inflamed. It was removed and the wound closed without drainage. The appendix was opened and found to contain blood and pus.

Next day the temperature was 99° and pulse 72. On the second day after operation the temperature and pulse were

normal, and the patient felt very well and appeared to be making an uneventful recovery. However, on the third evening after operation the temperature suddenly started rising, and the next morning reached a maximum of 103.2°, while the pulse remained steady at 80. This was accompanied by diarrhoea of a dysenteric type. The stools consisted chiefly of greenish-yellow mucus, with here and there pale yellow "cheesy" masses containing pus cells and also bright red blood clots—about two or three per stool. The stool was examined bacteriologically.

One thought of the possibility of dysentery, or post-operative peritonitis, but the steady pulse, soft abdomen, absence of pain, and the general well-being of the patient were against the latter. That evening he had a sharp bout of epistaxis and later vomited some old blood; this latter was probably the result of the former, because neither epistaxis nor vomiting was repeated. He now complained of a sharp pain in the region of the umbilicus, which was relieved by going to stool. The motions (about six per diem) continued to show the bright red blood. The fever continued but the pulse remained steady. This lasted for six days, the fever gradually diminishing and the general condition of the patient always remaining surprisingly good. In the meantime, from the stool an organism had been isolated, cultured, and identified as *Bacillus paratyphosus* B. The appendix wound healed by first intention, and the patient made a good recovery from the paratyphoid fever.

It may be mentioned that the paratyphoid B is the common form of enteric in this part of Germany, but, as far as the British garrison is concerned, this was an isolated case. Apparently the sudden onset with rapid rise of temperature, dysenteric stool, infection of the colon causing bright red blood clots in the stool, steady pulse, etc., are all compatible with a paratyphoid B infection, but at the time this case caused great difficulty clinically in diagnosis.

The following questions are of interest. When did the paratyphoid fever start, and was the pathological condition of the appendix due entirely to *B. paratyphosus* B? If the condition of the appendix was entirely due to *B. paratyphosus* B, and if it had not been removed, would it have perforated, causing peritonitis? Unfortunately the appendix was not examined very carefully after operation, or sectioned, because it appeared an ordinary routine case. It may be mentioned, however, that at the operation there was no evidence of inflammation in the coils of small intestines seen, or in the commencing portion of the ascending colon, whereas the appendix was acutely inflamed. Finally, was free peritoneal fluid the result of a paratyphoid B infection in the lumen of the intestines?

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ACTIVE PULMONARY TUBERCULOSIS OF EXCEPTIONALLY LONG DURATION.

THE following case appears to be of sufficient interest to warrant publication.

X. Y. recently died of pulmonary tuberculosis at the age of 60. He gave a history of having suffered from "bronchitis" since he was 8 years of age. When he was 14 he commenced work as a compositor, but from the commencement he lost a great deal of time owing to cough and malaise, and he was told by his medical attendant that he was "in a decline." At the age of 27 he had an attack of pleurisy, followed by blood-stained sputum, and was definitely diagnosed as suffering from pulmonary tuberculosis. When 39 years of age he was sent to the Brompton Hospital, where he was found to have a cavity in the right lung.

He came under the Hertfordshire County Council Tuberculosis Scheme at the age of 47, and he was found to have signs of widespread disease in both lungs, and tubercle bacilli in the sputum. With an occasional course of sanatorium treatment he was able to continue at his occupation until the age of 53, when he was pensioned off. At no period of his life had he been able to live and work as a fit man, and he never remembered being without cough and sputum. The last two years of his life were spent more or less in bed.

From the above history it would appear that the patient had suffered from active pulmonary tuberculosis which was slowly progressive for forty-five or fifty years. Until the last two years of his life his general condition remained fairly good, but whether the marked chronicity of the disease was due to exceptional resistance of the patient or to an infection of low virulence is a matter of doubt.

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