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.