A CASE OF RUPTURED TUBAL PREGNANCY,

INFECTION; CŒLIOTOMY; LARGE INJECTIONS OF SALINE SOLUTION WITH BRANDY; RECOVERY.[1]

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I SAW the patient, a married woman, aged 24 years, at Dr. R. H. Clay's request on Feb. 14th, 1899. The following was the history of the case. The patient had been married for seven years. She had had two children, six and five years ago. There had been no miscarriages. She had not missed a period for five years till Jan 15th last, when the period then due did not appear. On Feb. 3rd there had been "pain in the back and both passages." Retention of urine had been relieved by catheter. Pain had been again experienced on the 6th and there was a little blood per vaginam. There had been "straining in the back passage" on the 10th, with diarrhœa and a few clots per vaginam. On the 13th there had been diarrhœa and pains like those of labour. When I saw the patient on the morning of the 14th I ascertained that except for the amenorrhœa she had no reason to think herself pregnant. She had had leucorrhœa before and after marriage, but had had no abdominal pain during the last five years. The abdomen was slightly distended and tender, with an indistinct sense of resistance above the pubes. It was resonant except in the flanks, where dulness was most marked on the left side. The dulness was not affected by change of position. Per vaginam the uterus was found to be depressed and close behind the pubes. The cervical canal was closed. In Douglas's pouch was a smooth, very tender mass resembling an ovary; the rest of the pelvis felt full and boggy. Per rectum the same boggy mass was felt. The upper rectum felt constricted. The lips were somewhat anæmic. The pulse was 110 and the temperature was 101° F. Since 5 o'clock on the previous evening the patient had taken 80 minims of solution of morphia; she had been free from pain and had slept well. I diagnosed ruptured tubal pregnancy with coagulation of effused blood. When seen again at 9 P.M. it was found that in spite of the administration of 40 minims of solution of morphia there was great pain in the lower abdomen which was much more distended and which had a dull area above the pubes corresponding with a distended bladder. The temperature was 103° and the pulse was 124. The face presented a drawn appearance and the breath had an odour suggestive of grave sepsis. I considered that the blood in the abdomen was becoming infected and that possibly a fresh hæmorrhage was taking place, so, with Dr. Clay's concurrence, I decided to operate without delay.

At 1 A.M. on Feb. 15th chloroform was administered by Dr. Bushnell. The abdomen was scrubbed with soap-and-water and the pubes was shaved. 15 ounces of urine were withdrawn by catheter. Assisted by Dr. Pethybridge I opened the abdomen by a six-inch incision through the right rectus. The omentum and small intestines were partly adherent to the pelvic contents. On separating these handfuls of dark clot and fluid blood appeared. The uterine vessels were at once clamped at each cornu. After emptying the pelvis of blood the right Fallopian tube was found to be ruptured at its centre leaving a ragged cavity. The right ovarian vessels were tied at the pelvic brim and the uterine vessels at the uterine cornu. I had intended cutting away the tube and ovary and then uniting the two layers of broad ligament by a continuous suture, but as by this time the radial pulse had vanished I transfixed the broad ligament and tied off with the usual interlocking suture. On removing the clamp from the left uterine vessels some oozing occurred which was controlled by a double ligature not interlocked. Several large clots were removed from the right loin; the left flank appeared to be empty. The omentum had several clots incorporated with it, as had also the rectum and uterus. These clots were so adherent that they were left. Some difficulty was experienced in replacing the distended intestines. Mass sutures were used to close the abdomen. Before tying the last suture five and a half pints of hot saline solution were poured into the abdomen through a funnel and tube directed under the liver. This revived the patient slightly but she was still pulseless at the wrist. So far the operation had lasted an hour and five minutes. The chloroform administration was now stopped. On looking for a superficial vein into which to inject saline solution none was visible. The venæ comites of the right brachial were then exposed in the middle of the right arm. They were so small that it was impossible to introduce a cannula. The incision was then prolonged up to the fold of the axilla where the veins united and into this vein eight and a half pints of hot saline solution with an ounce of brandy were injected. Chromic catgut ligatures were used throughout. The patient who from excess of fluid was very bloated and blanched was warm and had a small radial pulse of 140. Just before being put into bed she had several rigors the result of the saline injections. She had a fair night with no sickness, sleeping fairly and passing a pint of urine. When seen at 10 A.M. her right forearm was very swollen and her face suggested severe nephritis, being very white and œdematous. The bowels had acted slightly. On the 16th she had taken small quantities of milk, brandy, and soda water. The bowels were acting freely. She had passed four pints of urine in the 24 hours following the operation. From the 17th onwards the patient picked up rapidly. She took food freely and the bowels and kidneys acted well. Some pain in the right iliac region due to the formation of an abscess which opened through the abdominal incision on the sixth day was relieved by occasional 20-minim doses of solution of morphia. The temperature, which varied between 99° and 102°, reached normal on the tenth day. The pulse after the first week rapidly declined and on the twenty-fifth day it had come down to 78.

When I saw the patient on the evening of the first day (Feb. 14th) she was evidently becoming acutely septic, as evidenced by the distended and painful abdomen, an increased pulse-rate in spite of more morphia, a temperature of 103°, diarrhœa, and the peculiar odour of breath so often associated with grave sepsis. Filling the abdomen with hot saline solution converts the peritoneal cavity for the time being into an internal hot-water bag and may in addition help to prevent adhesions. It also prevents any large secretion by the peritoneum which in one case appeared to me to be the cause of fatal collapse four hours after a laparotomy for a ruptured tubal pregnancy. The eight and a half pints of saline solution with brandy injected intravenously, partly compensated for the fluid lost by hæmorrhage and diarrhœa and gave the kidneys a chance of eliminating the septic material which had been absorbed. In a similar case of sepsis not complicated by hæmorrhage I intend, if by injecting a large amount of saline solution intravenously I can get a pulse of good volume, to relieve the patient of blood to the amount of a pint or more, with the object of directly reducing the poison present in the circulation. Everything used at the operation was boiled immediately before being used, with boiled saline solution for the instruments, &c. Antiseptics were not used. I consider it illogical and useless to apply antiseptic solutions in the strengths usually employed to a wound which is considered capable of union by first intention. If there had been time I should have tried to disinfect the skin with ether followed by a compress of a strong antiseptic. Normal saline solution does least damage to cut tissues and corresponds fairly well to the lymph which circulates in the part. As the five-inch incision in the arm healed by first intention the abscess in the abdominal incision was probably caused by infection from within and not from without. On examining the ruptured tube the fimbriated end was found to be blocked by a partly organised and adherent blood-clot, the sequence of events probably being partial tubal abortion, blocking of the abdominal ostium by clot, increased tension within the tube, and rupture into the cavity of the peritoneum.

The interval of five years since the last pregnancy is explained by the husband's admission of the use of artificial means to prevent conception. It would be interesting to know if the above explanation is applicable to other cases of tubal pregnancy which have been recorded as following a long period of sterility. I attribute recovery to the large

[1] A paper read before the Plymouth Medical Society on March 18th, 1899.

amount of fluid injected—one and three-quarter gallons in all—which in the first place relieved shock and afterwards enabled the emunctories, especially the kidneys, to dispose of the poison, and also to the very able assistance which enabled me to complete the operation in just over the hour.

Plymouth.

THE CONTENTS OF THE STOMACH IN THE GASTRIC CRISES OF LOCOMOTOR ATAXIA:

A CLINICAL AND CHEMICAL STUDY.

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GASTRIC crises hold a prominent position among the visceral symptoms of locomotor ataxia and no one observing a patient suffering from them when accompanied by much vomiting can fail to be struck with the enormous quantity of fluid which is often rejected, even when the patient is taking little by the mouth. As a rule, first of all the stomach is emptied of what food it contains; then there is vomiting of a clear liquid which later may be stained with bile or occasionally with blood. Marie[1] says: "There is vomiting, at first of food, but afterwards exclusively of slimy liquid or perhaps of a clear liquid mucous in character and more or less abundant. At times the vomited matter is coloured by bile and more rarely by blood; it may quite exceptionally present the appearance of coffee-grounds." With this general description that by Gowers agrees closely. He states that the vomiting is at first of food and then of clear liquid which may be very abundant; ultimately bile is vomited and sometimes blood. As regards detailed chemical examination of the vomited fluid there are on the whole but few reports, especially by English workers. Sahli[2] some years ago stated that he had found excessive acidity of the gastric juice in this condition, but his analysis is unreliable as he made no exact quantitative estimation but merely relied on the intensity of the colour reaction as judged by the eye. Marie quotes Rosenthal (1886) who found 0·3 per cent. of HCl during the first days of the crisis and 0·10 per cent. during the latter days, so that, taking Richet's figure of 0·174 per cent. as the normal, Rosenthal's figures show an increase in at least part of the attack. The same was found by Simonin of Lyons (1886). Hoffmann in one of his patients found the gastric juice to be secreted in excess and to contain temporarily an excess of acid. The quantity of HCl varied, often amounting to twice the normal quantity and rarely to less than the ordinary amount, and this only when owing to the abundance of the matter vomited the acid was somewhat largely diluted. The only other point in this connexion to which reference is made by Marie is that in some cases small and varying amounts of lactic acid have been found. Grasset[3] gives no details regarding the composition of the fluid, while Möbius[4] considers hyperacidity to be the exception in these cases and that the amount of acid is usually small.

In a recent paper Michell Clarke[5] gives a good account of the stomach contents in a patient suffering from early tabes both between and during the crises. The chief points as regards the chemistry are: 1. In the intervals gastric digestion was rapidly performed and there was excess of HCl during the process (sometimes 0·4 per cent.), whereas in the intervals between meals there was no evidence of secretion of HCl. 2. During the whole duration of the gastric crises free HCl was absent from the stomach contents altogether while lactic acid was abundantly present. In his analyses, however, no exact quantitative estimation of the different acids was made, the result being simply deduced from the amount of dilution which the fluids would stand while still exhibiting the colour reaction for the given acid. In Dr. Michell Clarke's case, as in the one which I am about to describe, there was marked hæmatemesis. In drawing any conclusions as to the presence of hyperacidity it must be remembered that the amount of acid secreted may vary, according to Verhaagen,[6] considerably even in health. He found that there might be variations of one per mille, 1·5 per mille, and even two per mille, the acidity from two to two and a half hours after a test meal ranging from 3 to 4·8 per mille. As regards lactic acid our latest knowledge of the gastric chemistry shows that it may occur in the fluid rejected in locomotor ataxia as in any other condition provided the stomach is emptied within an hour of eating and provided the last meal contained at least some carbohydrate. Here it is formed as a result of bacterial action upon maltose taking place within the stomach. This formation of lactic acid, which is quite physiological, ceases with the secretion of HCl and the lactic acid appears to be absorbed—at all events, it disappears from the stomach. Various observers, however, have found, as I myself did, that lactic acid may be present during gastric crises hours after food has been ingested and subsequently vomited. In the absence of dilatation of the stomach it is difficult to explain this, as recent observations point to the fact that when HCl is not secreted lactic acid does not take its place.[7]

Having recently had the opportunity, through the kindness of Dr. James Finlayson, to whom I desire to express my obligation, of studying the gastric crises in a case of early tabes where much hæmatemesis occurred I made a number of analyses of the stomach contents which I shall give in detail after a short account of the general features of the case.

A man, aged 27 years, was admitted to the Western Infirmary, Glasgow, on Oct. 14th, 1898, under the care of Dr. Finlayson, complaining of sickness and vomiting and weakness of the legs. The history of the patient's illness is taken in part from the ward journal. Eighteen months previously he had noticed some weakness of the legs and unsteadiness on going downstairs or when he was in the dark. Six months later girdle pain was experienced, but only for a fortnight. At the same time attacks of vomiting began. When the attacks were about to come on he had a feeling of nausea and uneasiness in the epigastric region; also great restlessness, so that he always tossed about in bed. When vomiting began he first rejected any food that was in the stomach and as emesis proceeded he brought up mucus mixed with blood and bile. The vomiting had no special relation to food either as regards the nature of the latter or the time of ingestion. The attacks occurred at first every fortnight but latterly every two or three days and might last from one hour to a couple of days. Eructations occasionally occurred before the attacks but as a rule the latter began suddenly. For nine months he had had some bladder trouble and a tendency to constipation. As regards his previous health, he had had gonorrhœa but not syphilis, nor was there any history of any stomach disturbance or of any gastric symptoms in the past. He was temperate in the use of alcohol. As regards his state on admission, the patient was pale, ricketty, and undersized, but fairly well nourished. His weight was 7 st. 7 lb. As to his nervous system it may be briefly stated that there was some impairment of the tactile and temperature sense in the toes and in an area extending from the umbilicus to the fifth rib. Romberg's sign was well marked; the superficial reflexes were present and were normal; the patellar reflexes were absent. The pupils were equal and medium in size; the Argyll-Robertson phenomenon was present; ptosis existed and was most marked on the left side; the fundus was normal. Examination of the blood between the attacks gave 5,500,000 red cells and 9000 leucocytes per cubic millimetre, 80 per cent. of hæmoglobin, and a specific gravity of 1060. The urine was clear, amber-coloured, and acid, and its specific gravity was 1026; no albumin, sugar, blood, bile, or albumoses were present; occasionally it was neutral or alkaline even apart from the attacks, and it then showed a deposit of pus and ammonio-magnesium phosphate. As regards the alimentary system the patient after admission exhibited marked ptyalism, sometimes rejecting from a pint to a pint and a half of colourless, slightly

[1] Lectures on Diseases of the Spinal Cord, 1892, p. 266.
[2] Ueber das Vorkommen abnormer Menge freier Salzsaüre bei den Gastrischen Krisen eines Tabetikers, Correspondenzblatt für Schweizerische Aerzte, 1885, No. xv., S. 105.
[3] Traité pratique des Maladies du Système Nerveux, Paris, 1894, quatrième édition, p. 513.
[4] Twentieth Century Practice of Medicine, 1897, vol. xi., p. 837.
[5] A Contribution to the Clinical Study of the Gastric Juice, Brit. Med. Jour., vol. ii., 1898, p. 1863.
[6] La Physiologie et Pathologie de la Sécrétion Gastrique, Paris, 1898, p. 9.
[7] Schäfer: Text-book of Physiology, vol. i., 1898, p. 358.