

of deep inflammatory redness. On the swollen areola round the base of the nipple were situated six similar but smaller vesicles, each one being distinctly umbilicated. Four of them were on the inner side and the remaining two were on the upper and outer side of the base of the nipple. Over the greater part of the breast the skin was red and tense and the axillary glands were enlarged. There was great tenderness over the whole of the breast. The temperature, taken in the mouth, was 100·6° F. and the pulse was 120.

Although the vesicles were suggestive of vaccinia in their appearance and in the history of their development the diagnosis of the case would probably have been somewhat puzzling from the unusual situation of the lesions but for my recollection of the fact that on Dec. 4th I had vaccinated the four children of this patient, none of whom had been previously submitted to the operation. In each case good results were obtained and when the arms were inspected on the tenth day they each presented four perfect though somewhat slowly developed vesicles. In the case of the two-month-old baby there was a good deal of inflammation and I therefore saw the child again on the 16th, when I found the vesicles had been broken and there was then a considerable amount of discharge from them which the mother afterwards told me continued for some days. I advised her to bathe the arm frequently and to dredge over it some boric powder. On questioning her closely I learnt that she had misunderstood my directions and had merely wiped away the discharge with a small piece of dry wool and that she was in the habit of putting the child to the breast immediately afterwards without previously washing her hands. In doing so she no doubt conveyed the vaccine matter directly from the baby's arm to the abraded surfaces on her own left nipple and thus unwittingly infected herself. It may be added that she had not been vaccinated since she was two years old.

Commercial-road, E.

## A Mirror

OF

### HOSPITAL PRACTICE, BRITISH AND FOREIGN.

*Nulla autem est alia pro certo noscendi via, nisi quamplurimas et morborum et dissectionum historias, tum aliorum tum proprias collectas habere, et inter se comparare.*—MORGAGNI *De Sed. et Caus. Morb.*, lib. iv., Proœmium.

#### ST. GEORGES HOSPITAL.

##### A CASE OF PERFORATED DUODENAL ULCER; OPERATION; RECOVERY.

(Under the care of Dr. CYRIL OGLE and  
Mr. A. MARMADUKE SHEILD.)

DUODENAL ulcer is decidedly more common in men while gastric ulcer is more frequent in women but the symptoms produced by perforation of a duodenal ulcer resemble so closely those arising from perforation of a gastric ulcer that a diagnosis between the two conditions is rarely possible before the abdomen is opened. It is very desirable that all cases should be recorded in which an operation has been performed for a perforated duodenal ulcer, whatever may have been the result, so that we may obtain data on which to found trustworthy statistics. For the notes of the case we are indebted to Mr. T. Crisp English.

A man, aged 36 years, walked into St. George's Hospital on July 26th, 1901, giving the following history. Four hours previously, when at work, he had been suddenly seized with violent abdominal pain which completely "doubled him up." This was accompanied by vomiting, collapse, and profuse sweating. In a short time he recovered but as the pain and vomiting continued he went to the hospital. He attributed these symptoms to having eaten half a pound of unripe cherries on the previous evening. In the past medical history of his life there had been no similar attack nor had there been any dyspepsia or other gastric trouble. Free indulgence in beer and spirits was admitted. On examination the patient presented an alcoholic aspect with marked acne rosacea. There was nothing in his appearance

to suggest that he was seriously ill but he complained of intermittent colicky pain about the umbilicus and was sweating freely. There was slight tenderness in the epigastrium but there was no distension or rigidity of the abdominal wall which moved freely with respiration; the liver dulness was normal. There were no signs pointing to trouble in the region of the vermiform appendix. The pulse was 80, of good quality, and the temperature was 98·4° F. The only obvious symptoms, therefore, were colicky pain and slight epigastric tenderness and the diagnosis rested between intestinal colic resulting from the unripe cherries, biliary and renal colic, lead colic, and an acute peritoneal lesion in the stage of reaction.

The case was obviously one to be carefully watched and the patient was admitted by Dr. Cyril Ogle. Food by the mouth was withheld and the pain was treated by hot applications; enemata were administered and acted freely; no morphia was given throughout the case. The patient was at first much relieved but in the evening the pain again became severe, whilst the pulse-chart which had been carefully taken showed a progressive increase in frequency. At 6 P.M. it was 96, at 10 P.M. 110, at 12 midnight 116, and at 2 A.M. 128. The abdomen was now becoming markedly rigid and tender and vomiting occurred three times. The liver dulness, which as previously stated was normal on admission, now disappeared to the left of the right nipple line. On the following morning, after a consultation between Dr. Cyril Ogle and Mr. Marmaduke Sheild, a diagnosis of perforated gastric or duodenal ulcer was made and operation was determined upon. Mr. Sheild opened the abdomen in the middle line above the umbilicus (28 hours after perforation) and found free gas and a large quantity of bile-stained fluid in the peritoneal cavity, with early general peritonitis. A rapid examination of the stomach showed that this organ was intact, but on examining the duodenum a small perforation was found on its anterior aspect, just beyond the pylorus. This was closed with a double row of Lembert sutures which held readily and an omental graft was also sewn on the part of the operated area. The peritoneum was then thoroughly irrigated and cleansed with sponges, special attention being paid to the right kidney pouch and iliac fossa. The abdominal wound was closed, no drainage being employed. The operation lasted 25 minutes. The patient was kept on rectal feeding for four days, after which small quantities of peptonised milk were given and the amount was gradually increased. There was no return of pain or vomiting; the bowels were very freely opened by enemata containing one drachm of magnesium sulphate, one and a half drachms of glycerine, and water to four ounces. The wound healed perfectly and recovery was complete, the patient getting up at the end of the third week.

*Remarks by Mr. ENGLISH.*—Very few cases are on record of successful operation for perforated duodenal ulcer. Indeed, Mr. B. G. A. Moynihan<sup>1</sup> in an exhaustive examination of all the recorded cases, could only find eight instances of recovery. Within the last few years this subject has come prominently under the notice of surgeons and it is of importance that all cases, fatal or otherwise, should be reported, that in this way a reliable foundation may be built on which to extend our knowledge of the disease.

In connexion with the above case may be related another case of perforated duodenal ulcer in which death took place shortly after admission to the hospital, on May 11th, 1901. The patient was a dairyman, aged 32 years, who for one week had suffered from indigestion with vomiting. On May 10th at 11 A.M. he was seized with sudden agonising pain in the abdomen followed by severe vomiting and profound collapse. He was taken home and seen by a medical man. The pain and vomiting continued and the abdomen became distended but the patient was not sent to hospital until 9.30 P.M. on May 11th. On admission he was extremely pale and collapsed, the radial pulse could scarcely be felt, the temperature was 96° F., and the extremities were cold. Intense abdominal pain was complained of. The abdomen was greatly distended, tender, and rigid. Liver dulness was completely absent, a tympanitic note being obtained all over. In spite of full doses of strychnine subcutaneously, brandy, &c., he died in about half an hour, 35 hours after perforation. The necropsy revealed advanced general peritonitis, with much gas and fluid in the peritoneal cavity. In the upper aspect of the

<sup>1</sup> THE LANCET, Dec. 14th, 1901, p. 1656.

first part of the duodenum just beyond the pylorus was a sharply punched-out ulcer of about the size of a threepenny-piece, with a small circular perforation at the bottom of it. There were no adhesions or thickening round the ulcer.

The first case forms an excellent illustration of a fact which is, perhaps, not sufficiently emphasised—namely, that there frequently exists a well-marked stage of reaction in acute peritoneal lesions. Failure to recognise this has frequently led to errors in diagnosis and serious delay in treatment. Three distinct stages may be described in these cases. First, the stage of collapse, which is usually profound and which with the agonising pain and vomiting forms the clinical condition known as “peritonism.” A history of the sudden onset of these symptoms in a healthy individual is strongly suggestive of some very serious lesion within the abdomen and forms a valuable aid in the diagnosis of the condition. The second stage is that of reaction and this may prove very misleading. The patient rallies from the initial shock, the general condition improves, whilst the pulse and temperature may be normal and the abdomen may present no evidence of serious mischief. The patients may, as in the case related above, walk into a hospital with the contents of their intestines leaking into the peritoneal cavity. If first seen in this stage, as is frequently the case, a too hopeful view is apt to be taken, the patient is not carefully watched, and thus by delay the chances of recovery are minimised. Should morphia, as is usually the case, have been given freely the symptoms are still further obscured. The third stage is that of the onset of obvious peritonitis, in which the general condition progressively deteriorates, the pulse increases in frequency, and the condition of the abdomen points unmistakably to a widespread infection of the peritoneum. Progressive quickening of the pulse is a sign of the utmost importance.

It is noteworthy that vomiting in cases of perforated duodenal ulcer is usually pronounced and persistent from the beginning, contrasting with the vomiting in perforated gastric ulcer, which occurs as an initial symptom in about 40 per cent. of the cases and rarely persists. The mortality of the affection is necessarily very high. Mr. Moynihan places the mortality of cases operated upon at 84.6 per cent. It must, however, be remembered that it is certain that many of the fatal cases are not recorded, whilst, on the other hand, it is probable that some perforations are sealed by the formation of adhesions and that a natural cure results. In still other instances abscesses have formed and these have been known to “track down” behind the peritoneum. One of the principal operative difficulties is to find the ulcer when this is small and situated towards the posterior aspect of the gut; indeed, the lesion has repeatedly been “missed” by operators and has actually only been discovered with difficulty at the necropsy.

Dr. Cyril Ogle and Mr. Marmaduke Sheild kindly gave me permission to publish this case.

### EDINBURGH ROYAL INFIRMARY.

A CASE OF TABES WITH ACUTELY DEVELOPED ATAXIA IN WHICH GREAT AND RAPID IMPROVEMENT RESULTED FROM FRENKEL'S PLAN OF TREATMENT.

(Under the care of Dr. BYROM BRAMWELL.)

THE value of Frenkel's treatment of locomotor ataxia is by no means yet generally acknowledged and therefore the following case will be of interest. In the rapid onset of the ataxia and its almost equally rapid disappearance, even though Frenkel's treatment was employed, Dr. Bramwell's case differs markedly from most cases of tabes and therefore it does not, perhaps, prove very clearly the benefit of this form of treatment.

The patient was a married man, aged 35 years. For five years he had been a barman and for 16 years previously a ship-steward. He was admitted into the Edinburgh Royal Infirmary on Jan. 22nd, 1902, because of inability either to stand or to walk. He had enjoyed good health until the present illness commenced. He absolutely denied having had syphilis or gonorrhœa. There was no suspicion of alcoholic excess; his average allowance of alcoholic liquor was four glasses of beer per week; he did not take more than 12 glasses of whisky per annum. He was not a heavy smoker. For six months before the inability to walk

developed he had had some, but only slight, shooting pains in the legs, more especially the left leg, which was sometimes swollen at night, the result, he thought, of varicose veins. He had occasionally experienced a dull pain in the back and some little difficulty in micturition, having to “force” a little to get the urine away. The inability to walk developed rapidly on Jan. 13th, 1902. For a fortnight before this date he had felt some stiffness and “pins and needles” in his legs and feet but had no difficulty in walking in the dark. He was in the habit of walking from his place of business to his house—a distance of about a mile—every night at 12 o'clock. On Sunday, Jan. 12th, he walked three miles. On the 13th he had great difficulty in walking from one room to another and on the 14th he was quite unable either to stand or to walk.

On examination it was found that the inability to stand and walk was due to ataxia. There was no paralysis. The muscularity of the legs was good. The movements of the legs were wildly ataxic. There was no ataxia in the arms. When the eyes were closed there was total loss of the sense of position, both during and after passive movements, in the lower extremities. Occasional shooting (lightning) pains were complained of in the legs (thighs and calves). There was some varicosity of the veins of the left leg. The left calf measured 12½ inches and the right calf 11½ inches in circumference. The patient was a short, spare man. The knee-jerks and Achillis-jerks were completely absent, even after reinforcement; the deep reflexes in the upper extremities were present but not exaggerated; the jaw-jerk was present. The direct muscular excitability of the anterior and posterior muscles of the leg and of the quadriceps group was slightly increased; there was no myoidema contraction. The plantar reflex was very active in both legs, the toe movement being flexion; the cremasteric reflex was absent on both sides; the abdominal reflex was present but slight. The pupils were unequal in size and markedly dilated; the right measured eight millimetres and the left 10 millimetres; they were quite insensible to light and only contracted very slightly in the act of accommodation, the right pupil becoming six millimetres and the left eight millimetres in size; they did not dilate on powerful stimulation (pinching and faradic irritation) of the skin of the neck. There was well-marked thoracic and ulnar analgesia and there were considerable analgesia and anæsthesia of the lower extremities. Biernacki's sign was not present. There was also marked analgesia of the muscles of the calf. There was some, but only slight, hypotonia. There was no girdle sensation. A slight dull pain in the “small of the back” was occasionally experienced. Slight difficulty in micturition (necessitating “forcing”) was occasionally experienced: the bowels were regular; there was little or no loss of sexual desire and power. There had never been diplopia or ptosis; the ocular movements and the optic discs were normal; the special senses were normal; there were no crises and no trophic or vaso-motor derangements. The mental functions were unimpaired, there was no articulatory defect, and there were no facial or tongue tremors.

A striking and rapid improvement resulted from Frenkel's plan of treatment. On Jan. 22nd the patient was unable to stand or to walk, the movements of the legs being wildly ataxic. Frenkel's treatment was commenced on the 27th. In the course of 10 days (on Feb. 7th) the patient was able to stand and to walk with the help of a go-cart. A week later (the 14th) he was able to walk with the help of two sticks. On March 5th he could walk steadily and well with sticks and he could also walk across the room fairly well and without a marked ataxic gait without sticks—i.e., totally unsupported.

*Remarks by Dr. BRAMWELL.*—The case is interesting because of the following features:—1. The absolute and apparently quite straightforward denial of the patient that he had ever had syphilis or gonorrhœa. 2. The rapidity with which the ataxia was developed. I have lately analysed 155 cases of tabes which came under my notice and in only three of them was the ataxia very rapidly developed. 3. The dilated condition of the pupils. In 104 of the 155 cases of tabes which I have analysed the size of the pupils was mentioned in the notes. In 69 of these 104 cases, or 66.3 per cent., the pupils were contracted (below three millimetres); in 27 cases, or 25.9 per cent., they were of medium size (between five and three millimetres); and in only eight cases, or 7.6 per cent., they were dilated (above five millimetres). 4. The rapid improvement which resulted from Frenkel's method of treatment.