

If the total quantity which is available is a very small quantity we shall therefore employ an extremely fine capillary pipette. If, on the other hand, we have a relatively considerable quantity of serum available we shall employ a capillary pipette of correspondingly larger bore. In any case, we shall do well to arrange matters so that the quantity of serum which we have at disposal for our first titration shall occupy not less than 1-2 cm. of the stem of our capillary pipette. We may assume for the sake of fixing our ideas that it occupies a length of exactly 2 cm. In such a case we make a mark with a red wax pencil upon the stem of the pipette at a point about  $2\frac{1}{2}$  cm. above the end of the pipette. Having done this, we turn up the point of the pipette and allow the serum to run down the stem of the pipette up to the point which is indicated by the red pencil mark. The object of this manœuvre is to take an air-bubble index into the point of the pipette. Having done this, we proceed to dip the end of the capillary pipette into a drop of our twenty-fold dilution of normal sulphuric acid, and we allow the acid to run up into the stem of the pipette until the upper margin of the air-bubble which separates the serum from the acid just reaches the red mark upon the stem of our pipette. It is obvious that when the air-bubble index reaches this point the quantity of acid solution which has been run up into the stem of the pipette will be exactly equivalent to the amount of serum which was drawn off into the pipette. Having thus obtained exactly equal quantities of our serum and our tritrating acid, our next step will be to mix the contents of our capillary pipette. We can do this either by drawing up the contents of our capillary tubes two or three times through our mixing chamber, or, if our tube is unprovided with a mixing chamber, by blowing out the contents of our capillary pipette into a clean watch-glass and then re-aspirating them, after complete mixture has been effected, into our pipette. Mixture of the contents of our pipette, having been accomplished, we have only to blow out the mixture of serum and tritrating acid in a series of separate drops on to the surface of a strip of our red litmus paper. In the case of the particular mixture we are considering—i.e., in the case of a mixture consisting of equal volumes of serum and a twenty-fold diluted normal sulphuric acid—we should almost certainly find that we had in our mixture an excess of acid. We should then proceed in a precisely similar manner to titrate with each other equal volumes of serum and a thirty-fold diluted normal acid. If we were dealing with normal blood this mixture might or might not have a slightly acid reaction. If this mixture proved to be still acid we should of course proceed to test equal volumes of serum and a forty-fold diluted normal acid. This mixture would, in the case of normal blood, almost to a certainty be alkaline. Supposing that it was alkaline, and that the reaction of the mixture of equal volumes of serum and thirty-fold diluted acid had proved to be acid, we should then proceed to mix in a clean watch-glass equal volumes of a thirty- and a forty-fold diluted normal acid. We should titrate our serum again with this thirty-five-fold diluted normal acid. If we found that this quantity of acid just sufficed to neutralise the acidity of our sample of serum, it is obvious that the alkalinity of our serum would be

most simply expressed by the fraction  $\frac{N}{35}$ . For a serum which is exactly neutralised by an equal volume of a thirty-five-fold diluted normal acid would obviously correspond in alkalinity to a thirty-five-fold diluted normal solution of alkali.

In a future communication I hope to draw attention to some results of therapeutic and diagnostic importance which have been obtained by the exploitation of this method of measuring the alkalinity of the blood. For the present it will suffice to put on record for the guidance of others who may be disposed to employ this method of measuring the alkalinity of the blood that the maximum value which has been found by this method for the alkalinity of the normal

serum has been  $\frac{N}{25}$ . This result has been obtained only once.

In the case of thirteen other normal persons the serum has been found to possess an alkalinity which has always varied between the values of  $\frac{N}{30}$  and  $\frac{N}{45}$ . On the average the alka-

linity of the serum has been equivalent to about  $\frac{N}{35}$ .

Netley.

## 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.

#### ROYAL FREE HOSPITAL.

A CASE OF DILATATION OF THE STOMACH ASSOCIATED WITH PERIPHERAL NEURITIS.

(Under the care of Dr. WALTER CARR.)

A MAN, aged twenty-eight years, was admitted into the Royal Free Hospital on Nov. 20th, 1896. He said that since he was about sixteen years old he had been subject to occasional attacks of abdominal pain, lasting about twelve hours and not specially related to food. For some years these attacks were not attended by sickness, and the symptoms from which he suffered do not suggest that he had a gastric ulcer; neither had he ever swallowed any corrosive fluid. Since 1890 the attacks of pain had recurred about every two months, had been more severe, and had been terminated by copious vomiting. Since about Christmas, 1895, they had become much more frequent, sometimes occurring two or three times a week, with very severe pain; exertion seemed to increase their frequency. The patient said that before the attacks his stomach felt very uncomfortable and he could feel it "working" (evidently the peristaltic movements). After the sickness he was much relieved for a time. In June, 1896, his legs had begun to get weak, with much pain in the calf muscles, numbness and tingling in the legs and slightly in the hands; since August he had been unable to walk and had wasted rapidly. No history was obtained of any acute illness except influenza five years ago. He entirely denied any alcoholic excess, saying that he had rarely taken more than one or two glasses of beer a day. On admission the patient was seen to be a well-built man, about 5 ft. 11 in. in height, but greatly emaciated; his weight was 9st. 7lb. He had not been sick for a week, but the day after admission he brought up  $7\frac{1}{2}$  pints, and vomited from 1 pint to  $2\frac{1}{2}$  pints nearly every day for the next week; he also brought up much flatus. The vomit contained abundant sarcinae, but no blood. The tongue was clean, the appetite fairly good, and the bowels acted nearly every day. On examination of the abdomen peristalsis was frequently visible, starting in the left hypochondrium and passing across the umbilical region to the right. On palpation no tumour, thickening, or resistance could be detected in the neighbourhood of the pylorus or elsewhere. The stomach was distended by giving bicarbonate of potassium and citric acid, and was found to be greatly dilated, reaching well below the umbilicus, though the exact limits could not be defined. The liver dulness was normal. In regard to the nervous system there was no complete paralysis of any limb, but his legs were very weak, especially the left, and he was unable to extend the left toes. He could only walk very feebly, requiring a great deal of support. He could just stand alone, but not when the eyes were shut. No other evidence of ataxy was detected. All the muscles of the legs were much wasted. In the upper limbs wasting was especially marked in the supra- and infraspinati and the interossei. In the muscles below the knees no reaction was obtained to faradaism, but all the muscles reacted to the galvanic current. He complained of tingling and numbness in the legs, and the calf muscles were very tender. There was very slight impairment of sensibility below the knees, chiefly about the feet. No girdle sensation was present. The knee-jerks and plantar reflexes were absent. There was no loss of sexual power or of control over the sphincters. His pupils were normal and no ophthalmoscopic changes were found. His chest was healthy. The temperature was normal throughout his stay in the hospital. The urine contained no albumin or sugar.

The patient was kept in bed and carefully dieted; he was given hot water to drink before each meal, little or no fluid being allowed with the food; strychnine

was given by the mouth. From Nov. 28th the stomach was washed out every morning, at first with simple warm water and afterwards with a solution of bicarbonate of sodium (two grains to the ounce), which seemed to act rather more beneficially. At first a large quantity of very offensive sour-smelling mucus and grumous material was brought away, but this rapidly diminished, and after a few weeks the washings each day returned quite clear. The effect of the treatment was immediate, the vomiting ceased after the first washing, only recurring occasionally afterwards, chiefly when the patient began to get up, movement seeming at first to excite it. The appetite improved, the flatulence diminished, and he began to gain weight. The nervous symptoms also steadily improved, the legs got stronger, so that the patient began to walk alone, sensation became normal, and the tenderness of the calf muscles slowly subsided. He was discharged on March 7, 1897, greatly improved in every respect and having gained 18 lb. in weight. He was seldom sick, and his appetite was good; there was still some flatulence, but much less than before. He could walk well and all the nervous symptoms had disappeared, except that there was still some weakness of the extensors of the left toes and the knee-jerks had not returned. He had learned to wash out his stomach himself without any difficulty and was directed to do so every morning before breakfast. He was seen on May 3rd and had then not been sick for six weeks; he was feeling well and much stronger; he could move all the toes quite freely; the knee-jerks were still absent; his weight was 11 st. 4 lb., a further gain of 7 lb. since leaving the hospital.

*Remarks by Dr. WALTER CARR.*—The above case presents several features of interest. The diagnosis of dilated stomach was obvious from the quantity and characters of the vomit as well as from the examination of the abdomen. Its causation, however, is obscure. The marked visible peristalsis, indicating hypertrophy of the muscular coat, points doubtless to the existence of definite obstruction at the pylorus, and hence mere chronic catarrh as a possible cause of the dilatation is probably excluded, even were it at all likely to lead to such great enlargement in a young and previously very robust man leading an outdoor life. All the ordinary causes of pyloric obstruction can also with reasonable confidence be excluded; there is nothing to suggest that there has ever been any peritonitis leading to displacement or narrowing of the pylorus; malignant disease is, of course, out of the question; a gastric ulcer near the pylorus might no doubt have led in healing to narrowing of the orifice, but the patient's early gastric symptoms, so far as he remembers them, were not suggestive of ulceration, which is, moreover, exceedingly improbable in an otherwise healthy lad of sixteen, at or before which age abdominal pain, without any vomiting, was first complained of. It is well known that when gastric dilatation occurs in comparatively early life the cause is often obscure, and I think that in this, as in some other similar recorded cases, a congenital obstruction or kink at the pylorus may possibly be present. The narrowing is probably but slight, and consequently symptoms only arose slowly, some hypertrophy of the muscular coat being sufficient for a long while to overcome the obstruction; but as soon as food began to be retained for a considerable time fermentation would occur, and the gases evolved, by distending the stomach, would lead to more rapid increase in the dilatation and so to the development of the characteristic symptoms. Some such theory as this best explains, I think, the commencement of gastric symptoms in early life, the gradual but steady increase in their severity, and the very great dilatation ultimately attained. There is no need to emphasize the excellent results of the treatment by lavage; improvement began after the first washing, and all kinds of medicinal and dietetic treatment had been tried previously without the slightest benefit. If there be permanent pyloric obstruction the washing out will doubtless have to be continued for an indefinite time, but this is not a serious matter, as the patient seems rather to enjoy it than otherwise.

As to the nervous symptoms, the partial but symmetrical affection of both motion and sensation, the tenderness of the muscles, the loss of the knee-jerks, together with the absence of girdle pain or of any affection of the sphincters or pupils, presented a sufficiently characteristic picture of peripheral neuritis, a diagnosis confirmed by the complete recovery. The chief interest of the case, however, centres in the causation of this neuritis; undoubtedly, the symptoms strongly suggest the alcoholic

variety, but the patient's appearance and his behaviour whilst in hospital certainly did not point to any alcoholic excess, and I see no reason for doubting his statement that he rarely took more than a glass or two of beer a day. In this opinion his regular medical attendant (Mr. Greeves, of Willesden), under whose care he had been for a long time, entirely coincides. The patient had not suffered from any of the acute specific fevers, nor had he been exposed to any other probable cause of multiple neuritis so far as could be ascertained; and from the way in which the nervous symptoms developed as the gastric troubles increased, and began to improve as soon as the stomach was washed out, I am disposed to attribute the neuritis to absorption of the products of bacterial fermentation from the greatly dilated stomach. There seems nothing improbable in this view, although I am not aware of any previous cases having been recorded. For a long period, probably some years, large quantities of toxic substances must have been formed in the stomach, and some would doubtless be absorbed; and there seems no reason why they should not exercise a special action on the nervous system similar to that of the many poisons, organic and inorganic, which are recognised as possible causes of multiple neuritis. Of course, it may be argued that if the neuritis arose from this cause other similar cases ought by this time to have been recorded; but be it remembered, in the first place, that excessive dilatation of the stomach, persisting for any considerable time, is by no means a common condition (the most frequent cause of great dilatation is probably pyloric cancer, and this is necessarily fatal in a comparatively short period); and secondly, that alcohol and the toxins of the acute specific fevers (except diphtheria) cause multiple neuritis in only a very small minority of the cases in which they are present, and that similarly the poisons generated in a dilated stomach may only attack the nerves in an extremely small percentage of cases. Dr. Henry Waldo, of Bristol, has recorded<sup>1</sup> a case of peripheral neuritis in a girl, aged twenty-one years, following upon symptoms of poisoning after eating tinned salmon. He attributed the neuritis to the salmon and not to any metal. If this conclusion be correct, there seems no reason to suppose that if the products of decomposition of food outside the body can lead to neuritis, those formed as a result of its retention in the stomach should not at times exercise a similar action.

## MELBOURNE HOSPITAL.

### A CASE OF RUPTURE OF THE LIVER; OPERATION; RECOVERY.

(Under the care of Dr. W. MOORE.)

IN the less severe ruptures of the liver, which are not accompanied by such an amount of hæmorrhage as to prove rapidly fatal, collections of blood and bile tend to form, and should the laceration of the organ have been on its upper surface, adhesions between the liver and the anterior abdominal wall may so shut in the effused fluids that the general peritoneal cavity escapes. The implication of the right pleural cavity is by no means unusual in the partial ruptures of the upper surface of the liver. A case in many respects similar to the one recorded below was brought before the West London Medico-Chirurgical Society<sup>2</sup> by Mr. W. H. Battle in 1894.

A boy, aged eleven years, was admitted into the Melbourne Hospital under the care of Dr. W. Moore on May 31st, 1897. The patient had been kicked by a horse on the right side—the lower part of the (lateral) thorax—two days prior to admission. He was seen soon after the accident by a medical man, who found him completely collapsed and complaining of great pain in the right side; the diagnosis made was rupture of the liver. In four or five hours after the accident the abdomen had become distended, and this condition lasted about five days, but during these five days there were no other marked symptoms; the bowels acted after enemata. Four days prior to admission vomiting set in and had since continued. It was on account of this condition that he was sent to the hospital, as the medical attendant thought that the vomiting was due to

<sup>1</sup> Brit. Med. Jour., vol. i., 1889, p. 589.

<sup>2</sup> THE LANCET, May 19th, 1894, p. 1252.