second patient prolonged drainage was employed to cure the cholecystitis, and incidentally appears to have acted beneficially on the pancreas. In the third case the gall bladder was drained, but only for a short time, and the pancreatic trouble subsequently increased until a large cyst formed. In the fourth patient, for the reasons mentioned in the notes of the case, I joined the gall bladder to the duodenum with resulting cure of the jaundice and the other symptoms complained of, but the pancreas still remains somewhat enlarged.

In the paper referred to Mr. Robson commends prolonged drainage of the gall bladder in preference to cholecystenterostomy and in his hands it has given very satisfactory results. His objection to cholecystenterostomy is chiefly the likelihood of the anastomotic opening closing without giving such prolonged drainage of the gall bladder as will suffice to cure the pancreatitis. A more important objection I think is offered by the greater risk implied in an anastomosis as compared with mere drainage of the gall bladder. Probably in the majority of cases the simpler method will be employed, anastomosis being reserved for those cases in which the nature of the pancreatic enlargement and its subsidence are doubtful, or in which prolonged drainage fails to cure.

The occurrence of thickening about the head of the pancreas with jaundice has in the past had a dire significance, it being constantly inferred that malignant growth was present. The possibility of a more hopeful explanation is offered by such cases as I have described and by those contained in Mr. Robson's paper, whilst the value of exploratory opening, when there is any room for doubt, is strongly emphasised.

REFERENCE.
Lancet, July 28th, 1900.

A CASE OF CONGENITAL HYPERTROPHY WITH STENOSIS OF THE PYLORUS.

BY

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A FULL term male infant, born of healthy parents, weighed at birth $8\frac{1}{2}$ lbs., and did not present any outward deformities or abnormalities. The mother's milk was not available, but the child's feeding was most carefully supervised. The first symptoms—vomiting and convulsions—arose when the infant was a fortnight old; diarrhoea and vomiting occurred a week later; temporary relief followed careful dieting, but after another week's interval profuse sickness set in, the vomited matters being highly acid, and apparently completely digested. Vomiting now continued on and off, the amount returned being sometimes in excess of that recently taken, constipation was troublesome, and the child lost weight. When six weeks' old death appeared imminent from continued convulsions. Probably these convulsions depended on the absorption of toxic material from the stomach, and may be considered as analogous to tetany in gastric dilatation. On rectal injections of saline solution containing albumin, rectal feeding by enules, and abstinence from any food by the mouth, the infant rallied, and even put on a little weight.

Five days before death the vomited matters contained blood, as shown by microscopic examination. Three days before death dilatation of the stomach was made out, this, though carefully sought for previously, had not been found. At no time could any tumour in the region of the pylorus be detected. It had been looked for, inasmuch as the condition found after death was suspected during life. Death was preceded by increasing asthenia and emaciation. The infant was 7 weeks and 5 days old. The infant was under the care of Dr. Crofton-Atkins, and was seen twelve days before death by Dr. Rolleston.

Necropsy.—A very limited examination only was possible. There was great hypertrophy, with almost complete occlusion of the lumen of the pylorus. A transverse section of the thickened pylorus showed a mere radiating fissure representing the canal of the pylorus, which barely

admitted the point of a pin. It is probable, as pointed out by Pritchard, that the degree of stenosis found after death cannot be regarded as an accurate gauge of that existing during life. The thickness of the wall of the pylorus was 8 mm., and was manifestly largely due to increase in the circular muscular coat, which was 4 mm. thick, the longitudinal coat being 2 mm., and the mucous and submucous coats 2 mm. thick. There was hypertrophy of the muscular coats in the antrum pylori and neighbouring part of the stomach, and intense injection of its mucous membrane.

Microscopic sections were taken through the pylorus in the situation of the accompanying photograph, and from the

Fig. 1.—Transverse section of the hypertrophied pylorus, actual size. The lumen of the pylorus is seen as a radiating fissure. (From a photograph by H. G. Drake-Brockman, M.R.C.S.)

middle of the stomach. At the pylorus the mucous membrane was well preserved and healthy; the submucosa was condensed, probably merely a mechanical effect of pressure; the circular muscular coat was greatly increased in thickness, and showed a few foci of small cells between some of the muscular bundles. The mucous membrane in the piece of the stomach wall examined showed catarrhal inflammation. It is probable that spasmodic closure of the pylorus prevented this catarrhal change from spreading into the pylorus. The condition of the oesophagus was not examined. There was, therefore, stenosis of the pylorus of extreme degree, and marked hypertrophy of the circular muscular coat. The gastritis was probably a late or terminal phenomenon, and showed itself clinically by blood in the vomit five days before death.

REMARKS.

In the *BRITISH MEDICAL JOURNAL* of April 28th, 1898, one of us (R.) published a case, in conjunction with Dr. L. B. Hayne, and referred to 22 previously recorded cases. Since then published reference has been made to 19 other cases: Meltzer (5),¹ Still (3), Cautley (2), Fletcher (1), Batten (1), Pritchard (1), Adler (1), Stern (1), Thomson (3), and Fisher (1); in addition, 2 cases referred to by Ashby in 1897 but omitted in the collection of 22 cases, must be included.

We have therefore references to 44 cases already published, though in some the bare statement of the observation only exist, making with the present case 45 in all.

It is noteworthy that the great majority (about three-fourths) of these cases have been recorded since 1897, when J. Thomson drew special attention to the subject. It is therefore probable, as pointed out by several writers, that the disease is less rare than might be thought. Further, it is a significant fact in the short history of this disease that not uncommonly an observer who has seen one case, and thus become familiar with the existence of the condition, is able to report others. [Hirschsprung, Henschel, Gran, Thomson, Soltan Fenwick, Schwyzer, Meltzer, Cautley, Still, Ashby]

In 30 cases in which we have been able to obtain information as to sex, 18 were males and 12 females. Drs. J. Thomson and J. Raglan Thomas (case recorded by Morley Fletcher) kindly gave us information on this point. Most of the cases were under 4 months old.

The causation of the condition has attracted a great deal of attention and speculation. Of the explanations given, two are the most prominent: (a) That it is a congenital redundancy; "Nature, in her extreme anxiety to provide an efficient pyloric sphincter, has over-exerted herself, and produced too great a quantity of muscular tissue" (Cautley); and (b) that of "congenital gastric spasm," that is, there is a functional disturbance of the nervous mechanism of the stomach and pylorus leading to over-action, and so to hypertrophy (Thomson). It seems reasonable to combine these views so far as to believe that there is some congenital hyperplasia of the pyloric sphincter, and that spasm supervenes on this, and is largely responsible for the symptoms manifested.

Diagnosis.—Obstinate vomiting associated with constipation in an infant whose dietary is carefully supervised may suggest the disease, but a diagnosis cannot be made in the absence of a tumour. In this case, though we were attracted by the idea, it did not seem justifiable to make a positive diagnosis of congenital hypertrophic stenosis of the pylorus. Such a conclusion would carry with it the question of surgical interference, and in the absence of a palpable tumour it would be impossible to exclude all other possible causes of vomiting in an infant.

Prognosis.—Cases in which this condition has been diagnosed, and in which recovery has occurred, have been mentioned by Finkelstein (3), Senator (1), Batten (1), and Barlow (1). Possibly some of these instances are, as suggested by Still, developmental variations in the amount of muscular tissue in the pylorus, in which the stenosis is not great. In Batten's case, where a male child had signs and symptoms of the disease when 11 weeks old, and recovered under careful treatment, but died from acute broncho-pneumonia and gastro-enteritis when 11 months old, the stenosis was not extreme.

A point of interest is whether these slighter cases, that do not prove fatal in early life, may subsequently present themselves as cases of simple pyloric stenosis. In the 31 cases described as congenital stenosis of the pylorus collected by Maier, and Landerer the ages ranged from 12 to 80 years, and though the type is certainly a different clinical one, it is possible that they are the outcome of slightly marked muscular hypertrophy in early life. It is conceivable that a primary muscular excess might in course of time undergo fibrous transformation, and result in fibrous stricture of the pylorus.

Treatment.—Rectal feeding led to temporary improvement in this case, but the condition of the pylorus, as shown in the woodcut, could hardly have been successfully treated by any means short of surgical.

Slighter cases, when recognised, should be treated, as Batten suggests, by nasal feeding, which, by obviating the peristalsis set up by deglutition, probably both (1) allows digestion and absorption to go on, and (2) tends to minimise the spasmodic stenosis of the hypertrophied pylorus. Probably a combination of rectal and nasal feeding, with a cautious and gradual return to ordinary means would be the best plan.

Six cases have been submitted to operation. Recovery followed Loreta's operation by Nicoll; the others were unsuccessful. Pylorotomy was performed in a case of Thomson's, and gastrostomy in Meltzer's, Stern's, Adler's, and Thomson's cases.

For the photograph illustrating this paper we wish to express our thanks to H. G. Drake-Brockman, M.R.C.S.

REFERENCE.

¹ In the earlier of his two contributions Meltzer speaks of having seen five cases. His second and usually quoted paper is devoted to a detailed account of one case.

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FURTHER OBSERVATIONS ON EPIDEMIC ARSENICAL PERIPHERAL NEURITIS.

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It has been very gratifying to me to have had my observations on the symptoms and cause of epidemic peripheral neuritis amongst beer drinkers so fully confirmed, and even more pleasing to know that the source of the arsenic has been traced, as was first done by Professor Delépine to the glucose and invert sugar, and ultimately to the commercial sulphuric acid, so that there is

Fig. 1, Case 1.—Paralysis of extensors of wrist and fingers.

every probability of a stop being put to the outbreak. At first Dr. Brooke and myself were of opinion that the source of the arsenic would be found in some connection with sulphur, and from the symptoms presented by a private patient of Dr.

Fig. 2, Case 1—showing "dropped feet" from paralysis of anterior tibial and peroneal muscles; slight keratosis of soles of feet.