6 per cent. of the women and 12½ per cent. of the men over 30 years of age admitted into the London Hospital for hæmatemesis from ulcer of the stomach. Cancer, according to Hauser, follows in 6 per cent. Pyloric stenosis, according to Gerhardt and Warren, occurs in at least 10 per cent. What may be the proportion in which other complications occur, so far as I know, has never been worked out, but few of those who have once had a chronic ulcer escape altogether, and fewer still remain exempt from pain and chronic dyspepsia even when their position in life is such as to enable them to take every care.

The operation, so long as the case is still uncomplicated, presents no serious difficulty. The risk of the ulcer being overlooked, if the stomach is carefully and systematically examined in the way I have already described,[1] is exceedingly small. Even if the diagnosis proved incorrect and there was no ulcer present, but merely chronic gastritis, there would be no cause to regret the operation. Gastritis that obstinately resists all ordinary remedies and continues with such persistence as to be mistaken for chronic ulcer is best treated by performing gastro-enterostomy and by giving the stomach a prolonged period of rest without reducing the patient's strength by starvation. The mode of dealing with the ulcer presents no difficulty. It may be excised or ligatured according to its depth and its extent. The wall of the stomach from its strength and thickness is even better adapted to suturing than the wall of the intestine. Difficulty only occurs when, owing to the length of time the ulcer has been allowed to continue, it has reached such a size, or such a depth, or has caused such dense adhesions to be formed to other organs, that simple excision or ligature is no longer practicable.

Taking acute and chronic cases together I have excised or ligatured in various ways an ulcer of the stomach in 13 instances, not reckoning those in which perforation had occurred. (These have all been published.) In many of them the loss of blood had already been extreme. In six transfusion had to be performed on the table or immediately after. Two of these patients, both of whom were in an absolutely desperate condition at the time of the operation, died; the rest recovered without a bad symptom and were cured. I cannot help thinking that if these two had been operated upon before they had lost so much blood they would have stood as good a chance of recovery as the others, and I feel convinced that if only ulcers of the stomach were operated upon as soon as it was recognised that they had become chronic there would be many fewer deaths from hæmatemesis and perforation and that such troubles as pyloric stenosis, hour-glass contraction, pain and dyspepsia from adhesions, perigastric and subphrenic abscesses, dilatation of the stomach, *et hoc genus omne*, would become far more rare than they are now.

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CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS.

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BUT a short while ago congenital hypertrophic stenosis of the pylorus was regarded by everyone as a very rare condition and those who had seen cases considered themselves not a little fortunate and exhibited their wares with some satisfaction to their less favoured brethren. Now all is changed—the pendulum has swung the other way and we are told that the condition is by no means uncommon. At a meeting of the Royal Medical and Chirurgical Society held on Dec. 9th a paper dealing with this disease was read by Dr. E. Cautley and Mr. C. T. Dent,[2] and in the discussion which followed many speakers expressed this opinion of its prevalence, one member having seen no less than nine cases within the past five months.[3]

Now, either the condition is rare or it is not rare, it cannot be both, and if the matter is observed more closely we find that those who consider it more common speak from clinical experience of living patients, and it is noteworthy that these observers also consider the condition less fatal than has been hitherto held. To the morbid anatomist, on the other hand, it still remains, as heretofore, a rare disease. This means either one or other of two things. Either the clinician is wrong in his diagnosis and mistakes cases of spasm arising from other causes for this condition, or the condition really is less fatal than was originally supposed and may often be recovered from without surgical assistance. May I urge the former explanation as the more likely?

In support of the latter explanation Dr. H. Ashby at the meeting of the Royal Medical and Chirurgical Society already mentioned, suggested that many vomiting marasmic infants met with are really instances of the disease, and this suggestion would probably be readily accepted by those who consider the condition one of not uncommon occurrence. If this were so, surely we should have some evidence of the fact. Seeing how readily all forms of gastro-enteric disturbance in the early weeks of life, especially among the poorer classes, lead on to a marasmus of fatal issue, and seeing, as a consequence of this, what an enormous number of such cases are examined post mortem in the London hospitals every year, surely, if the condition were anything but rare, cases would more often be met with accidentally on the post-mortem table. As a matter of fact, this occurs but seldom. The recorded cases of true congenital hypertrophic stenosis of the pylorus take little time to number, but there is another condition which occurs in certain cases and which may perhaps cause similar symptoms and lead to errors of diagnosis during life—namely, a simple hypertrophy of the pyloric muscle, probably produced after birth as a result of spasm. I have performed post-mortem examinations on two cases, at any rate, in which there was noticeable hypertrophy of the pyloric ring, and the condition occurred in marasmic infants dying with symptoms presenting some resemblance to those occurring in the more serious condition. Whether the hypertrophy in these cases was due to pyloric spasm and whether the symptoms depended on the pyloric hypertrophy must remain for the present a matter of speculation. Mr. Harold J. Stiles, at the meeting of the Royal Medical and Chirurgical Society above referred to, showed that great variety may occur in the amount of muscle present in the sphincter. This he regarded as accidental, but was he right? I am inclined to think that an increase of muscle may be due to spasm and when marked (as in the two cases mentioned above) may be associated with symptoms resembling those present in congenital stenosis. If this is so it is probable that the condition will be found more often if sought for.

How very different, indeed, is the appearance of the pylorus in these two conditions (and this is the point which I wish to emphasise here)—in the acquired variety a ring of muscle triangular in cross section, a mere exaggeration of the normal pyloric ring; in the congenital condition a long, firm cylinder. This difference in appearance is so striking that it seems to me to constitute in itself a point in favour of the origin of congenital hypertrophic stenosis as a developmental error, a primary hyperplasia, as opposed to its origin in ante-natal spasm, a theory upheld by many. In all the cases which I have seen recorded the pylorus is described as a sausage-like swelling fully one inch long, and the similarity of the descriptions is striking. No intermediate forms seem to occur; the condition is much the same in all.

Now is this a condition which is likely to be caused by spasm—not a mere hypertrophy of the existing ring but a pylorus deformed as a whole, a condition strikingly different from a hypertrophy occurring after birth (providing the cases I have mentioned above were such)? In answer to this it may be argued that its ante-natal origin may account for this difference, but this does not seem to me to be sufficient. Hypertrophy of the heart muscle in cases of congenital morbus cordis does not, so far as I am aware, present any marked difference from hypertrophy occurring after birth to compensate acquired lesions, and here we have the same cause at work—namely, increased function—as in the presumed pyloric spasm.

In most of the recorded cases of congenital hypertrophic stenosis of the pylorus the neighbouring parts, stomach,

[1] THE LANCET, Oct. 20th, 1900, p. 1125.
[2] THE LANCET, Dec. 20th, 1902, p. 1679.
[3] THE LANCET, Dec. 20th, 1902, p. 1692.