who has not been struck by the remarkable fact that tuberculous disease of the testicle so frequently follows blows or contusions of the organ which were unaccompanied by any open wound or breach of surface. Equally striking and suggestive is the very common occurrence of spinal caries following blows or sprains of the vertebral column. Applying this hypothesis to the elucidation of the mysterious frequency with which phthisis follows long after simple pneumococcal or streptococcal pleurisies and other non-tuberculous chest affections, the etiology of many examples of tuberculosis becomes a simple matter.

In conclusion I would venture to suggest that, though we still are hardly justified in assuming that phthisis never occurs from the inhalation of dried sputum-dust or from the breathing of the spray injected in the act of coughing, it appears to be conclusively proven that, as forcibly put by Ravel, the alimentary tract is a frequent portal of entry for the tubercle bacillus, which is able to pass through the intact mucous membrane of the bowel without producing any local lesion at the point of entrance; that this event is especially frequent in children, and that the milk of tuberculous cows is the common source in these cases. Probably at no distant date the contention of Calmette will be accepted that *in the immense majority of cases pulmonary tuberculosis is not contracted by inhalation*, but, as taught by von Behring, the germs enter through the intestinal tract. Future research may explain how in China, where the consumption of the milk of bovines is practically nil, tuberculosis is everywhere prevalent amongst the natives, as I have been informed by Dr. Duncan Main, of Hangchow.

The previously mentioned considerations of the etiology of human tuberculosis have their obvious bearing upon our action in regard to the introduction of measures of prevention which are now occupying the attention of the entire profession in its crusade against phthisis, and I am proud to say that nowhere in His Majesty's dominions is this crusade more vigorously active than it is in Ireland under the auspices and guidance of her Excellency the Countess of Aberdeen, about whose work I can the more unhesitatingly speak since I have been unable to take any direct part in it. The present movement is certain materially to diminish ultimately the appalling mortality of this dread disease, and we must not be cast down if immediate results are not obvious, bearing in mind that:

For while the tired waves, vainly breaking,
Seem here no painful inch to gain,
Far back, through creeks and inlets making
Comes silent, flooding in, the main!

Remarks

ON

CONGENITAL STENOSIS OF THE PYLORUS IN THE ADULT.

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Pyloric Stenosis.

In approaching the special subject with which this communication deals it is not inappropriate that the generally recognized causes of pyloric stenosis should be enumerated. No doubt the recognized causes are usually the common causes. On this ground the cause of stenosis here specially dealt with might be placed amongst the uncommon causes, for it has been sparingly and hesitatingly recognized even as a possibility; and yet it may soon be seen that it ought to be given a place in the common list. However that may be, at present the commonly-recognized causes are (1) ulcer, (2) cicatrix, and (3) malignant disease.

Pyloric Spasm.

That any of these three conditions may cause recurring spasm contraction of the pylorus, going on to muscle thickening, will not be questioned by any sound clinical pathologist. That the spasm is due to local irritation will be generally conceded; while the further proposition, that the spasm is an important factor in occluding the pyloric

outlet, will be readily accepted by clinicians who have watched such cases with any reasonable measure of interest.

That pyloric spasm may, in addition to the causes just enumerated, be also determined by the direct stimulation or irritation produced by the chemical constitution or mechanical composition of the gastric contents upon the unbroken pyloric wall will not meet with such general acceptance; while probably only few clinicians have confirmed Lauder Brunton's¹ observation that pyloric spasm may accompany migraine, and be but an additional manifestation of an explosive neurosis.

Effect of Pyloric Stenosis.

Without going into details, which are unnecessary, it may be noted that the main results or manifestations of stenosis are those of stomach dilatation or of stomach ptosis. The size and position of the stomach can, as a rule, be easily determined by physical examination; while the recurring vomiting demonstrates the retention in the stomach of material which, under normal circumstances, is readily passed on into the duodenum.

A further and a very important effect of pyloric stenosis is *visible stomach peristalsis*. This, when present, at once excludes the possibility of the dilatation or ptosis being due to muscular atonicity resulting from catarrh, anaemia, or nervous debility. It is only in pyloric obstruction, associated with the retention of considerable power in the muscular wall of the viscus, that this symptom will be noted; and in my experience such a loss of power only occurs in extreme cases, which are either cases in which there has been a long history of gastric trouble, or are malignant, with a relatively rapidly progressive debility and a correspondingly short history. Where definite pyloric swelling is palpable the merest tiro in clinical medicine has no difficulty in ascribing the gastric symptoms to pyloric obstruction.

Congenital Stenosis an Additional Cause of Pyloric Obstruction in the Adult.

From the preceding summary I hope it may be conceded that I approach the subject of congenital stenosis of the pylorus in the adult with sound views regarding pyloric stenosis in general, and I therefore turn now to the special theme of this communication.

The best way of expounding my subject is to begin from the historical side; and here we need not too seriously concern ourselves with the question as to who recorded the first case. John Thomson² ascribed it to Williamson of Leith in 1841, Osler³ has put it back to Dr. Hezekiah Beardsley in 1788; further research may put it back into the still darker ages of medicine. For our present purpose it is not profitable to go beyond the years 1879 and 1885. In the former year, 1879, Heinrich Landerer⁴ published the thesis submitted by him for the doctorate of medicine of the University of Freiburg. It was entitled *Congenital Stenosis of the Pylorus*. After referring to the hitherto known causes of pyloric stenosis and gastric dilatation, he proceeded to show that there was an additional condition to those he had enumerated—namely, one in which the stenosis was congenital. He supported this thesis by giving details of ten instances in which he noted pyloric narrowing. The first case observed was a man of 45 years, who had been for years in the hands of doctors on account of stomach disorder. After death it was found that he had an enormously dilated stomach without structural change, no thickening at the pylorus, and yet a pyloric outlet so small as to measure only 2 mm. This led to the careful examination of the condition of the pylorus in other bodies. The result was that he collected other nine instances of narrowing in persons ranging in age from 43 to 63 years. The degree of stenosis varied. Although it was only in the first case that a previous history was available, his clinical acumen led him to realize that this was a condition which must be of importance to the clinician.

In 1885 Professor Rudolf Maier⁵ of Freiburg published in *Virchow's Archiv* a contribution to the same subject. He gave a short account of 31 cases in which he had found a pyloric stenosis which he believed to be congenital. The observations were made at *post-mortem* examinations, his attention having been drawn to the matter by a striking example of the condition having occurred in one of his

patients. The cases varied in age from 12 to 75 years. The degree of narrowing of course varied considerably, but in none was the narrowing associated with what we would call a coarse structural lesion. As the result of these observations he separated his cases into two groups: First, a simple form; second, a combined form. The second group contained the cases in which, in addition to stenosis, there was thickening of the pylorus. With regard to the first group, where narrowing was present without other change at the pylorus, he did not think there was reasonable room for doubt that they were congenital; those in which there was pyloric thickening in addition to narrowing he examined more fully and argued more closely in support of their congenital nature.

This argument we need not follow now, for later developments have strengthened his contention that a combined form is met with in the adult. At the same time, the subject cannot be left without indicating that the description of the changes he observed will be found well worthy of attention from those specially interested in the exact anatomical appearances presented by them.

Congenital Hypertrophic Stenosis in Infants.

The next important step was the recognition that the condition now known as *hypertrophic pyloric stenosis in infants* was a pathological fact and a clinical entity. Hirschsprung⁶ in 1888 seems to have been the first to describe the condition; but many members of the profession probably first had their attention drawn to the subject by John Thomson's⁷ paper published in 1897. Since those dates the flood of literature on the subject has attained portentous proportions. It is no part of my present purpose to deal with that literature, or with the differences of opinion which have risen up around the etiology of the condition—that may be left in the hands of the protagonists already in the field. It may, however, be taken as established that there is met with early in infant life a stenosis of the pylorus due to, or accompanied by, marked thickening of the muscle of the pylorus; that the condition is commonly regarded as congenital, and is described as congenital hypertrophic stenosis in infants.

It does not appear, so far as a cursory look through some of the literature has disclosed, that any other form of stenosis has been observed in infants; that is to say, there are no examples of the class Maier called simple stenosis.

The hypertrophic stenosis is much the same as Maier's *Combined Form*. It is so easy of recognition in extreme cases that one wonders now that clinicians had not recognized it long before.

On that lack of observation on the part of clinical pathologists it is not necessary to enlarge. It is, however, necessary to claim that in a condition of this type there must of necessity be degrees of stenosis and degrees of thickening. All infants with hypertrophic stenosis of the pylorus do not die. Only the severe cases are seen by the expert, and it is only for them that surgical aid is required.

That all cases have not the same measure of pyloric narrowing, and therefore do not show equally urgent clinical symptoms will presumably be granted as a sound proposition. In support of the proposition there indeed emerges from the literature what to my mind is an illuminating difference of opinion. It is this: Controversy has arisen as to the part that spasm of the pylorus takes in the production of the hypertrophy and of the stenosis. Pfaunder⁸ goes far when he holds that the condition is entirely one of local cramp or spasm, and that it can be successfully combated by medicinal and dietetic measures. Pfaunder's contentions are based on clinical observation, and, as such, they belong to the data on which judgement has to be founded. At the same time, if I do not question his observations, neither do I question the observations of those who have had experience of cases unrelieved by dietetic or medicinal measures, and imperatively requiring surgical measures for the relief of a permanent and structural obstruction.

From this it must be inferred that there are degrees of stenosis, and that a pyloric stenosis is not incompatible with infant life; that in spite of its presence life may be continued into childhood and even onwards.

Simple Stenosis in Infants.

That, so far as I know, there is no recognition in infancy of what Maier calls simple stenosis is to me inexplicable,

and when cases begin to be recorded of simple stenosis of the pylorus being found in children of 6 years when on the operating table, it is at least suggestive that there is a clinical hiatus which ought not to be difficult to fill.

The Present Medical Position.

When we inquire into the position congenital stenosis of the pylorus in the adult occupies in the realm of clinical medicine at the present moment, there are facts to be noted which have considerable significance. The article on dilatation of the stomach⁹ in Allbutt's *System of Medicine* is from the pen of the erudite editor himself, but there is no mention of this condition amongst the causes of dilatation.

Sidney Martin¹⁰ and Hemmeter¹¹ do not mention the condition. Ewald,¹² quoting Landerer and Maier, includes congenital stenosis amongst the mechanical constrictions met with at the pylorus. Van Valzah and Nisbet¹³ recognize congenital pyloric stenosis in infants, and state that, with the observance of a proper diet, they may live into adult life.

Lambert and Fisher,¹⁴ writing in 1907 on benign stenosis of the pylorus, do not mention the congenital form.

Riegel,¹⁵ writing this year, only refers to congenital narrowing of the pylorus as due to muscular hypertrophy of the pyloric sphincter, and as leading to early death.

Boas,¹⁶ writing in 1907, merely mentions congenital pyloric stenosis in the adult, and passes on to the consideration of other causes of stenosis.

Osler¹⁷ asks if some of the cases of dilated stomach with thickening or hypertrophy of the pylorus may not be congenital.

The Present Surgical Position.

When we turn to the surgical side of the literature dealing with the abdomen, the following may be quoted:

Cautley and Dent¹⁸ refer to three cases as suggesting a congenital origin, occurring at the ages of 6, 11, and 22.

Mayo Robson and Moynihan,¹⁹ after reviewing and discussing the literature on congenital hypertrophic stenosis, conclude as follows: "There can be little hesitation in affirming that a congenital abnormality of the pylorus of some, at present indeterminate, character may after the lapse of few or many years be so altered, or added to, as to cause symptoms of pyloric obstruction. The conditions and the frequency of such cases require further investigation." Mr. Mayo Robson, in his large experience, had only operated on one such case at the date of writing, the patient being a young man of 24 years.

Maylard²⁰ of Glasgow in 1903 wrote a paper that has not received the attention in this country to which it was entitled, on *Congenital Narrowness of the Pyloric Orifice*. The paper is based upon observations made when the abdomen had been opened for "chronic gastric derangement." The condition of the pyloric orifice in those cases was not only sufficient to explain the symptoms which existed, but "to suggest that the condition itself was probably of congenital origin." In 1904 he recorded another case, a man aged 31, in whom at the time of operation he found a hypertrophic stenosis of the pylorus, which he believed to be congenital, presenting the "funnel-shape" type described by Maier.

In none of the cases recorded does it appear that any attempt had been made at a differential diagnosis before the patient was submitted to operation.

When Landerer and Maier wrote, the surgery of the abdomen had not entered upon the brilliant operating epoch included in the last twenty years; and although they both insisted on the great value of differential diagnosis, the success of surgical operations on the stomach may be said in the interval to have dulled the desire for diagnostic precision. That phase has already nearly passed.

The Existence of the Condition.

From all that precedes it seems to me that the occurrence in the adult of congenital stenosis must be accepted as an established fact. From the observations of Landerer and Maier and from the experience of Maylard it might even be predicted that, as soon as attention is definitely directed to the condition of the pylorus, cases of pyloric narrowing will be found to be a fairly common cause of the more intractable gastric symptoms. To again quote Maylard: "There exists a considerable class of patients in young adult life who owe their chronic gastric

trouble to a congenital narrowness of the pyloric orifice." That both the simple and the hypertrophic forms will be found also seems to be probable.

Three Cases.

As a modest contribution to the diagnosis of congenital simple stenosis of the pylorus I submit the following three cases which have fallen into my hands in the last two years.

CASE I.—Thomas C., aged 34, married, was admitted to the Edinburgh Royal Infirmary on August 28th, 1906. He complained of vomiting and pain in the abdomen, and stated that he had been very bad for the preceding six weeks. On going into his history it was ascertained that he had been troubled with his stomach for fifteen years, that is, since he was a youth of 19 years. The general description of his symptoms was that he had discomfort, fullness, and heaviness in the stomach, coming on usually about an hour after food. The discomfort was sometimes followed by and relieved by vomiting. Sometimes the vomiting was at night. The vomit when copious, he thought, consisted of what he had taken for some days. The vomit had sometimes been dark in colour and frothy. Twelve years ago he was treated in the infirmary for dilated stomach, and for the same condition two years later. Since that time he has been troubled off and on with his stomach. During the past year he has had comparatively little trouble until six weeks before admission, when discomfort, pain, and vomiting came on. The discomfort and vomiting became very severe, and, he was confident, worse than ever before experienced. He had lost flesh and, of course, weight. Constipation had been extreme, the bowels only moving once a week if left to themselves. There was no history of melaena, nor of vomiting recently poured out blood. The patient had a fresh complexion, but was very lean. The stomach was greatly dilated; the fundus was at the level of the fifth rib, and the lower border below the level of the umbilicus. The lesser curve was lowered in position, while the pylorus was high up under the liver edge. He on several occasions vomited as much as 3 pints, and as much had been removed by the stomach tube. The total acidity of the gastric contents was high, and there was much free hydrochloric acid. From the symptoms and from the stomach dilatation and ptosis there was no doubt about pyloric obstruction. The chemical examination of the stomach contents excluded malignant disease, and the symptoms were not those of gastric ulcer. From the high position of the pylorus I inferred that it was probably held up by old adhesions, and that the fixity with the addition of ptosis would so kink the outlet as to lead to obstruction. The patient had a gastro-enterostomy performed by Mr. Caird. At the operation no adhesions were found, and there was no evidence of recent or of old ulceration. The difficulty at the pylorus was due to an indurated and apparently fibroid condition of the pyloric ring. The patient made a speedy recovery, and was entirely relieved of his symptoms. It never occurred to me that the condition of the pylorus found in this patient might be congenital until Mr. Maylard, of Glasgow, wrote to me on the subject, pointing out that the condition corresponded with cases he had described. The suggestion at once appealed to my judgement, and I was surprised it had not instinctively occurred to me before.

CASE II.—James M., aged 42, married, was admitted to the Edinburgh Royal Infirmary on July 22nd, 1907. He complained of vomiting, constipation, giddiness, and weakness. He stated that he had lost much flesh during the preceding fortnight, and he was so ill that he had been a week in bed before admission. Careful inquiries into his history elicited the fact that he had suffered more or less from his stomach all his life. Even when he was a herd-boy he used often to vomit. The vomiting attacks came on at intervals of a few weeks, at other times after an interval of a few months. The attacks were preceded by "sickness" (nausea). After vomiting he was relieved, but the vomiting might continue for some days, but after that he would be all right again for a while. When asked about the character of the vomit, he said that he "always minds that it was very, very sour." At that time his food was the same as his brothers and sisters had, and they did not suffer as he did. These attacks of stomach disorder had recurred all through life, and it was only by great care as to his feeding that he was able to avoid their more frequent occurrence. He usually breakfasted at 9 a.m., and could not take another meal until 2, 3, or 4 p.m. His afternoon meal commonly consisted of rice-soup made with mutton, some of the mutton being cut up finely and put into it; or of fish or tripe with bread. He gave up taking potatoes and all other vegetables years ago, as they "always came up." He drank a good deal of skimmed milk and buttermilk. His symptoms had been severe since the middle of June; they were of the same type as formerly, but were more persistent, and he could not depend on any meal staying down. The stomach showed dilatation and ptosis; the greater curve was considerably below the level of the umbilicus, while the lesser curve reached the level of the umbilicus. The pylorus was in the right hypochondrium, not far from the costal margin; there was no thickening. Handling the abdomen set up visible gastric peristalsis. The gastric contents showed abundant free hydrochloric acid. The diagnosis in this case seemed to me to be perfectly clear, and I strongly advised the patient to submit to operation. A gastro-enterostomy was performed by Mr. Dowden. There was no evidence of recent or cicatrized ulcer; the pylorus had to my eye much the same appearance as the former one, and Mr.

Dowden could only put the tip of a finger into, not through, it. This patient also made an excellent recovery.

CASE III.—Mrs. H., aged 51, was admitted to the Edinburgh Royal Infirmary on October 25th, 1907. She was a small, spare, ill-nourished woman. Her complaints were referred to the stomach. She had suffered all her life from indigestion, but for the past four years her symptoms had been more pronounced. In fact, during that period her medical history was a varied one, although gastric symptoms, as pain and vomiting, appear to have predominated. She had never been able to eat vegetables; there was a history of having vomited material like coffee grounds shortly before admission to the infirmary. On examination the stomach showed dilatation and ptosis, both curves being displaced downwards, the greater one reaching to considerably below the level of the umbilicus. There was often visible gastric peristalsis. There was no thickening of the pylorus. The gastric contents contained no free hydrochloric acid. From the dilatation, ptosis, and peristaltic movements there was no doubt as to the existence of pyloric difficulty, but without pyloric thickening. The symptoms were not those of gastric ulcer. The absence of thickening at the pylorus led me to negative malignant disease notwithstanding the absence of free hydrochloric acid. In fact, the symptoms seemed to me to be due to the retention of stomach contents; and in view of the lifelong history of indigestion and the need of extreme care with regard to her food and great moderation in the quantity taken, I looked upon this case as another example of congenital stenosis. I advised this patient to submit to operation. Mr. Hodsdon operated. The case was, however, an unlooked for disappointment. The condition at the pylorus was as I had judged it to be; but there was in addition a malignant growth high up at the fundus which could not be removed. A gastro-enterostomy nevertheless was performed and gave the patient complete relief from her gastric symptoms. She made an excellent recovery, but the operation was followed by that curious feeling of "having no mind" which is one of the occasional and mysterious sequelae of such operations. There was no constant pain, no pain on the ingestion of food, no difficulty of deglutition, no mass to be felt, to guide us to the suspicion of malignant growth at the fundus. The absence of free hydrochloric acid suggested malignancy, but being satisfied that the pylorus was not malignant the idea was set aside. I concluded that the absence of free HCl was an individual peculiarity which is not rare.

I heard of this patient in the beginning of June, 1908. There had been no return of the gastric symptoms and no further signs of malignancy. The mental condition had not improved.

Diagnosis.

The diagnosis may be briefly summed up under the following heads:

History.—There is a long history of stomach trouble, or of such digestive difficulties that the patient has learnt to be very careful as to the character and the quantity of food taken, and to allow a sufficient number of hours to separate meals. On the intelligence with which diet has been regulated depends the further history. There may be a history of supposed bilious attacks dating back into childhood. In Case II the patient as a boy had at varying intervals of time attacks of vomiting, which lasted for a day or two. He had had these when his feeding was his share of the food provided for the family, yet none of his brothers or sisters had similar attacks. As he grew to manhood he had learnt great abstemiousness in eating and drinking, and thus saved himself from a frequent repetition of acute digestive disorder. In Case III the patient had similarly learnt by experience how to regulate her diet. In Case I, where possibly the stenosis may have been less pronounced, there was a definite history of preceding attacks of severe digestive disorder with stomach dilatation. In all three patients previous attacks had been overcome by ordinary dietetic and medicinal means; they came to hospital because ordinary means had failed, and ordinary means failed in our hands also.

Symptoms.—The symptoms are the symptoms of stomach dilatation or of gastric dyspepsia, vomiting only occurring in the more severe attacks. The attacks of stomach disorder tend to become intensified as they are repeated; and as Mayo Robson has observed, something happens, often not till life is fairly advanced, which brings out the pyloric difficulty and leads to all the symptoms of pronounced pyloric obstruction.

Physical Examination.—At the stage when these cases are likely to come to hospital stomach dilatation is easily made out on physical examination. In 2 of my 3 cases gastric peristalsis could be induced. In Case I the dilatation and gastric atony were too pronounced to allow of this sign being elicited. When gastric peristalsis can be induced the pyloric end of the stomach also contracts and is easily located. In my 3 cases there was no permanent

—that is to say, continuous—pyloric thickening. The cases being examples of the simple form accounts for the absence of this sign; whereas in the combined or hypertrophic form there is permanent thickening, although the degree of thickening will vary from time to time. The absence of continuous pyloric thickening is a most important diagnostic point, but the negation of course assumes that the pylorus can be located through the parietes.

Pyloric Spasm or Cramp.—This as a physical sign requires a little special reference made to it. Kussmaul²¹ appears to have been the first to draw attention to it. It is recognized by Ewald, Hemmeter, Van Valzah and Nisbet, and Boas. It is a condition that I knew clinically long before I knew that Kussmaul had noted it. When spasm occurs, the pylorus hardens and thickens; when spasm relaxes, the pylorus softens. The two phases can usually be easily followed by the hand placed over the pylorus, as the patients are lean with a lax abdominal wall. The spasm can be caused by the irritation of the stomach contents, by structural lesion at or near the pylorus, or even by nervous disorder. Spasm occurs both with and without permanent hypertrophic or hyperplastic thickening of muscular and fibrous tissue. Such permanent thickening is also no necessary accompaniment of pyloric narrowing; in fact, in my three cases there was no thickening, and reference has been made to similar observations by others. When spasm is present there is thickening, and in this way spasm often enables us to locate with certainty the position of the pylorus, while the knowledge thus gained of its position may enable us to definitely determine after spasm has relaxed whether structural thickening has remained. When structural thickening remains, the question of its cause remains to be decided.

Chemical Examination of Gastric Contents.—The chemical examination of the stomach contents, especially the determination of the presence or absence of free hydrochloric acid, should, of course, never be omitted, for the result materially influences the formation of an opinion.

Summary.—The diagnosis in the case of simple stenosis is determined by the existence of pyloric difficulty, the character and duration of the history, the absence of permanent pyloric thickening, the presence of free hydrochloric acid in the stomach contents, the exclusion of ulcer, of malignancy at the pylorus, and of a history that would fit in with cicatrix from previous ulceration. When there is permanent thickening the *benign* causes, when we exclude ulcer, are congenital hypertrophic stenosis, and cicatrix from healed ulcer. The field for differential diagnosis can thus be greatly circumscribed and simplified. In fact, once the possibility of congenital stenosis occurring in the adult enters the clinical field of vision, it will, I think, be found to be quite within the differential diagnostic skill of the physician.

Treatment.

The treatment might be summed up in the words of Lambert and Foster²² when discussing benign stenosis of the pylorus, namely: (1) Control excessive secretions, (2) reduce pyloric irritability, and (3) increase the muscular activity of the stomach wall. Space does not permit of these principles being elaborated here. When dietetic and medicinal measures are no longer sufficient to enable a patient to nourish himself, the physician can, fortunately with confidence, advise the patient to submit to surgical operation.

The Clinical Pathology.

I have not had the opportunity of examining *post mortem* the character of the anatomical change at the pylorus in simple stenosis. The changes in both the simple and the hypertrophic form have, however, been dealt with by Maier and Maylard. In the three cases which have been in my hands I have only seen the pylorus when it was exposed at the time of the operation and when the surgeon was satisfied as to the smallness of the opening and, at the same time, of the absence of ulcer, new growth, or cicatrix to account for it. Under these circumstances, I fail to see what view the clinical pathologist can hold but that the narrowing or stenosis is congenital. There is, so far as I know, no pathological process which produces this kind of change, while I am told that a more or less corresponding congenital condition occurs at the internal os of the uterine cervix, and a congenital tightness of the prepuce is common. That the

condition may, in some instances, be the true cause of the attacks of indigestion or dyspepsia seen in young people has to be kept in mind, for until the possibility of occurrence is present to the mind it cannot be diagnosed. In young or later adult life symptoms are more likely to become pronounced, for it is a common experience to find digestive disturbances emerging during this period. It must also be acknowledged that, if congenital narrowing be present, gastric catarrh, hyperchlorhydria, or pyloric spasm will all tend to present exaggerated symptoms. When, therefore, any one of these conditions is present, the definiteness of pyloric difficulty may be a surer guide to the existence of the congenital defect than we as yet know. Another factor which Maylard lays stress on, probably rightly, is that any condition which weakens the muscular coat of the stomach, such as anaemia or general debility, will bring out symptoms of digestive trouble, owing to the pyloric narrowing effectively preventing the passage of the gastric contents under the lowered pressure of weak stomach contraction. There are thus several explanations to account for the symptoms of congenital stenosis first appearing prominently in adult life, or at least assuming such prominence as to send the sufferer to seek medical help.

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CONGENITAL NARROWNESS OF THE PYLORIC ORIFICE A CAUSE OF CHRONIC GASTRIC DISEASE IN THE ADULT.

ILLUSTRATED BY TWELVE ADDITIONAL CASES.

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SOME years ago, when making exploratory operations upon the stomach for intractable disease of that organ, I encountered cases which presented a condition of the pyloric aperture that appeared to me at the time difficult to explain on the basis of any organic or functional derangement. When the index finger was introduced through an incision in the gastric parietes and made to explore the pyloric orifice, it was either found difficult to insert it into the aperture, or, when forced through, it was felt to be gripped by a uniformly narrowed ring. There was no indication of irregularity suggestive of cicatrization after ulceration; no thickening of the tissue in and around the pylorus; no bands of non-resistant fibrous tissue; and no evidence that the muscles were in a state of contraction, spasmodic or otherwise. The only construction I could put upon the condition was that it was a congenital abnormality, and this of the nature of an aperture that had not developed sufficiently to meet the normal requirements of easy access from the stomach to the duodenum. This view and other reasons for adopting it—derived both from anatomical, pathological, and clinical sources—I embodied in a paper read before the Clinical Society of London, published in its *Transactions* (vol. xxxvii, 1904, p. 63), and also reproduced in full in the *BRITISH MEDICAL JOURNAL* (vol. i, 1904, p. 417). I need not, therefore, repeat the arguments used on that occasion in support of the assumption that such a condition as congenital narrowing of the pylorus is a possible cause of intractable gastric symptoms occurring in young and middle-aged adults. But since the record of the seven cases given in illustration of the remarks made on the occasion referred to I have carefully been on the outlook for others which might reasonably be considered confirmatory of the opinions expressed; and I now add