CONGENITAL DUODENAL ATRESIA.

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In a recent issue of the Journal* I recorded an example of congenital hypertrophic pyloric stenosis and, while discussing the question of differential diagnosis, I drew attention to the fact that other congenital lesions might give rise to somewhat similar symptoms. Among these other congenital lesions duodenal atresia and stenosis take first place, and the following record of an example of the former condition, which has just been under observation, will therefore be not without interest.

The patient was a three-day-old female infant, the first-born of healthy young parents. The pregnancy had been uneventful and had terminated spontaneously with the birth of an apparently healthy child weighing 6 lbs. Breast feeding was instituted and the child was able to suck quite vigorously and always seemed ready for its feeds, but from the first vomited almost immediately afterwards apparently all the milk which had been ingested. The vomiting was said not to be expulsive in character and the vomited material was reported to consist simply of the unaltered milk. There had been no bowel movement since birth and it was even doubtful if any meconium had been passed.

On coming under observation the child was noted to be small (4 lbs. 4 ozs.), of good colour (hæmoglobin 114% and red blood cells 6,400,000 per c.mm.) with, however, a definite icteric tinge of the skin. Although the child apparently had lost weight, there was no marked dehydration and the elasticity of the skin was good. The abdomen was retracted and lax, there was no evident distension of the stomach; visible peristalsis could not be elicited and palpation failed to reveal any pyloric tumour.

The vomiting after every feed continued and the vomitus was seen to be definitely bile-stained. The urine on the day after admission to hospital contained 0.06% of chlorides (normal .26%), but it should be mentioned that a subcutaneous injection of 250 c.c. of normal saline solution had been given on the day previously. Physical examination still failed to reveal any distension of the stomach or pyloric tumour, but on radiological examination great distension of the stomach with a small air bell in the region of the duodenum was noted and a complete absence of air in the gut. After a small barium meal the much distended stomach was very apparent with a smaller opaque ball to the right of the middle line and connected with the main stomach mass by a narrow band (Fig. 1B), and quite definitely due to a dilated duodenum. That there was a complete duodenal obstruction (atresia) was certain from the fact that 4½ hours after the ingestion of the barium meal there was no appearance of any of the opaque substance in the small intestine.

Since operation gave the only hope this was carried out by Mr. H. S. Wright under local anæsthesia. The state of matters found was just as anticipated, a greatly dilated stomach with ballooning of the first part of the duodenum, complete obstruction at the junction of the 2nd and 3rd portions, and collapse of the rest of the intestine. Anterior gastro-jejunostomy was performed, which the child seemed to stand fairly well, but she collapsed some hours later and died 23 hours after the operation. A post-mortem examination showed atresia at the junction of the 2nd and 3rd portions of the duodenum and that the operation had been satisfactorily performed, the gastro-jejunostomy opening functioning quite efficiently.

* Post-Graduate Medical Journal, 1935, xi, 352.

Commentary.

Although there is a close similarity, as one would naturally expect, between the symptoms of duodenal atresia or stenosis and hypertrophic pyloric stenosis, there are features which point more to one condition than to the other.

Vomiting is usually an early symptom, indeed from the first feed in the case of duodenal atresia, whereas this is the exception with pyloric stenosis. Moreover, bile-stained vomit is also common and may be a very marked feature when the obstruction is situated distal to Vater's papilla. It is usually stated that the vomitus in pyloric stenosis is never bile-stained, but this is not correct. Bile-stained vomit is certainly rare in pyloric stenosis but it is not unknown, although when it does occur it is only slight in degree.

FIG. 1A. Girl aged 3 weeks with duodenal stenosis. Radiogram 21 hours after a barium meal. Note distended duodenum partially filled with opaque meal and evidence of opaque meal in small intestine.

FIG. 1B. Patient with duodenal atresia. Radiogram after barium meal. Note distended duodenum filled with opaque meal and absence of meal in small intestine.

It is, however, on the physical signs that the differential diagnosis really rests. In the first place, it depends on whether the duodenal obstruction is partial or complete, and the stage of the illness at which the child comes under observation, whether some of the signs so characteristic of hypertrophic pyloric stenosis may also be present. If the obstruction is complete there is little likelihood that gastric peristalsis will be visible because there will not be time for hypertrophy of the stomach musculature to develop and without this peristalsis cannot result. As a rule the stomach simply dilates. If, however, the obstruction is partial (duodenal stenosis) not only is life considerably prolonged but there are present the conditions essential for hypertrophy and consequently gastric peristalsis may be as apparent as in any example of pyloric stenosis.

On the other hand, as there is no pyloric hypertrophy in duodenal obstruction, a pyloric tumour, which is so characteristic and indeed pathognomonic of hypertrophic pyloric stenosis, is not palpable. Too much stress cannot be laid on this point, as in our experience a pyloric tumour is possible of detection in 100 per cent. of examples of hypertrophic pyloric stenosis.

Examination of the urine, although of value in the diagnosis of obstruction high up in the gut, is of little value in deciding whether that obstruction is due to hypertrophic pyloric stenosis, duodenal atresia or duodenal stenosis. In all these conditions there is a diminished chloride content of the urine, indeed, not infrequently chlorides are entirely absent from the urine.

It is by the use of X-rays, however, that the greatest assistance is given in this matter of differential diagnosis. While X-ray examination may reveal little that is abnormal in the case of hypertrophic pyloric stenosis, and at most some delay in emptying of the stomach, in duodenal stenosis or atresia, on the other hand, the most characteristic pictures are obtained. In duodenal obstruction, from whatever cause, dilatation of the portion of the bowel proximal to the obstruction is present, and this is seen in the X-ray picture as an air-bell a short distance from the pyloric end of the stomach immediately to the right of the vertebral column. After a barium meal the distended duodenum is immediately partially or completely filled with the opaque meal and its passage or not into the small intestine shows whether the obstruction is complete (atresia), Fig. 1B, or not (stenosis), Fig. 1A.

Before concluding the discussion of the matter of diagnosis mention should perhaps be made of another congenital anomaly with a somewhat similar symptomatology to duodenal atresia and with which it might be confused. The condition referred to is oesophageal atresia. Here vomiting also occurs from the first attempt at sucking, but the child, though anxious to drink, can only swallow a few mouthfuls before coughing is set up and the milk, mixed with mucus, is immediately returned. The presence of this malady is easily determined by passing a stomach-tube, when it will be found to be held by $4\frac{1}{2}$ inches from the gums, or a spoonful or two of an opaque meal may be administered when on X-ray examination the upper part of the oesophagus will be found dilated and terminating in a rounded end about the level of the 3rd thoracic vertebra.

Treatment.

As it is impossible on clinical grounds to express any opinion regarding the nature of the obstruction further than that it is complete or incomplete, and thus one cannot say whether its remedy would be an easy or difficult surgical undertaking, all cases should be submitted immediately to operation. The partial obstruction may result from pressure by a band on an aberrant vessel, or from simple narrowing of the bowel, whereas the complete obstruction may be caused by a diaphragm, or there may be a complete hiatus in the continuity of the gut, but as recoveries have been recorded even in examples of this latter type no case should be regarded as hopeless. Before undertaking the operation the almost constant dehydration should be treated by liberal saline and glucose transfusions, and afterwards, if there has been any hæmorrhage, a blood transfusion may be given with distinct advantage.

The skiagrams illustrating this communication were taken by Dr. G. T. Calthrop, Radiologist, Princess Elizabeth of York Hospital for Children.