DUODENAL ULCERS IN TWO INFANTS OF THE SAME FAMILY.

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Duodenal ulcers are not a common event in young children and therefore I think that the occurrence of the lesion in two successive infants in the same family is worthy of record.

So far there have been three children in the family: the first, now aged six years is alive and well. The second child died at the age of two months in the Royal Hospital for Sick Children, Edinburgh, and the details were reported by J. A. L. Loudon¹ in 1925. My case, the third child, was admitted to the Dundee Royal Infirmary in a dying condition early this present year, and was found to be suffering, like the Edinburgh infant, from a duodenal ulcer which was the immediate cause of death.

These two cases present some points in common and some of difference. The Edinburgh infant was a small baby weighing at birth $6\frac{1}{2}$ lb. and never throve. The baby was breast fed for two weeks, and then on a varied assortment of artificial foods. Vomiting was frequent, and wasting, at first gradual, then rapid, soon produced a serious condition. Three days before death a brown stool was passed in which altered blood was found. On the following day a typical mæna stool was passed, and though on admission to the Royal Hospital transfusion was at once performed, there was further hæmorrhage, and death occurred on the next day. At the post-mortem examination an acute ulcer was found, $\frac{1}{2}$ -cm. from the pylorus, on the posterior wall of the duodenum, and another smaller ulcer on the anterior wall.

The child whom I saw was admitted to the Dundee Royal Infirmary on February 5th, 1928, at the age of seven weeks. At birth it weighed 10 lb. and for a week appeared to be healthy. Then for six or seven days there was a little brown vomit each day. Breast feeding was in consequence abandoned and a constantly changing succession of artificial foods was tried. After the first vomiting there was no recurrence, but there was incessant crying, and green stools without blood in them were frequent. The child rapidly wasted so that by the time of its admission its birth-weight of 10 lb. had sunk to $6\frac{1}{2}$ lb. The Wassermann reactions both of the child and its mother were negative; there was no peculiarity in the blood-count and the stools betrayed no sign of bleeding.

An X-ray examination showed a somewhat dilated stomach, which emptied at a normal rate: in $3\frac{1}{2}$ hours the whole meal had passed into the bowel.

The history of the previous child, together with the story of the brown vomit, suggested the diagnosis of a duodenal ulcer. In spite of treatment death took place on February 10th. A post-mortem examination revealed the presence of three ulcers, one the larger on the posterior surface of the first part

of the duodenum, $\frac{1}{2}$ -cm. from the pyloric sphincter, and two smaller ones opposite on the anterior surface. The larger ulcer was punched out and had penetrated the muscular coat ; the two smaller involved the mucous membrane alone. The microscopic examination of the ulcers showed nothing remarkable ; there was no sign of round-cell infiltration and no fibrosis.

DISCUSSION.

Duodenal ulcers in infants have long been known especially as post-mortem phenomena in the disease known as 'melæna neonatorum,' but of recent years they have been reported much more frequently, especially by the American pædiatricians. Theile² in a monograph on gastric and duodenal ulceration in infancy and childhood, published in 1919, was able to collect 64 cases of gastric or duodenal ulceration in children dead of melæna, 31 of which were duodenal.

Fig. 1.

In the first part of the duodenum one large ulcer is seen and two small ones lying below it.

He also showed that they were almost as frequent in marantic infants who had not shown any signs of melæna, and gives details of 48 cases. Together with more recent cases recorded in America there are in all some two hundred cases on record. I have not been able to find any record of successive infants in the same family. In all these records the ulcers have always been in the first part of the duodenum and most often on the posterior wall. They are never found below the ampulla. They do not show signs of round-cell infiltration, nor of induration, and are usually of the punched-out variety. Very rarely a perforation of the ulcer has been the immediate cause of death.

Of course the suggestion has been made that these ulcers are not the cause of the ill-health and wasting, but are the result, that is are secondary and terminal conditions. This contention is probably true of some cases, yet it is

difficult to resist the conclusion in other instances that they are at least the primary cause of the fatal termination.

Let us look at some of the hypotheses as to the cause. First, that the ulcers are of infective origin, streptococci and their toxins producing an area

Fig. 2.

Arterial Supply of the First Part of the Duodenum ; showing Supra-duodenal Artery arising from the Right Hepatic Branch (after Prof. Wilkie).—Supra-duodenal artery supplies upper one-third of the anterior surface, and upper one- to two-thirds of posterior surface. Branches from the pyloric artery in 50 % supply first $\frac{1}{2}$ -in. of upper border and adjacent parts of anterior and posterior walls. Recurrent branch from either R. gastro-epiploic or sup. pancreatico-duodenal artery supplies lower one-third of anterior surface of first inch of duodenum. Retro-duodenal artery, branch of gastroduodenal artery, supplies lower two-thirds of posterior surface. Branches from sup. pancreatico-duodenal artery supply termination of first part of duodenum : anastomosis between those and ones mentioned above is by no means free.

of necrosis, and auto-digestion leading to ulcer. Secondly, that there is a focal sepsis, and infection is carried direct by the blood-stream. Thirdly, that there is thrombosis, due to the great feebleness of the infant. In considering these hypotheses one must remember the fact that the ulcers are situated in the first part of the duodenum above the papilla. The suggestion is that the acid gastric juice is squirted on to the first part of the duodenum, and produces an ulcer, and that the duodenum below the papilla is protected by the alkaline bile and pancreatic juice. Braithwaite found that if the papilla were transplanted to a lower position, ulceration took place in the lower half of the duodenum.

I believe that I am right in saying that in health the acid gastric juice impinging on the duodenum will not produce an ulcer, and I venture to suggest another explanation of the cause and position of these ulcers.

First let me take one or two points of anatomy in regard to the position and blood-supply of the duodenum. The text-books of anatomy fully describe the surface markings and the normal position of the duodenum. It is, however, well known that there is no normal position of the stomach and duodenum; and though it is stated that the duodenum has a mesenteric attachment of the first part only, one will often find on careful examination that it has a complete mesentery. Mr. Taylor, one of the Assistant Surgeons at the Dundee Royal Infirmary, out of his accurate knowledge of anatomy, has several times demonstrated this fact to me.

Next look at the blood-supply. Wilkie of Edinburgh has thoroughly explored this region. He describes an artery not mentioned in the text-books, which he has termed the supra-duodenal artery. This has a varying origin and supplies the upper two-thirds of the anterior surface and the upper one- or two-thirds of the posterior surface of the first part of the duodenum. This artery is small and is in addition practically an end-artery, with little or no anastomosis between it and the pyloric. In wasted children with great muscular enfeeblement and laxity of ligaments there is a possibility of dragging down of the abdominal organs, and this might cause the supra-duodenal artery to be put on the stretch, thus narrowing or obliterating its lumen and causing a necrosis of the tissue supplied.

Lastly, I may add a few words about the diagnosis of these ulcers. At present the majority are diagnosed in the post-mortem room. In the case of adults there has been a progressive improvement in the diagnosis of duodenal ulcer. Is there a possibility that a similar improvement may be attained in the case of infants now that we are aware that the presence of ulceration is by no means an uncommon event?

At present the only guide that we have is the presence in the vomit or the stools of blood, together with marked and progressive wasting. In adults the information which can be obtained from an accurate history is probably one of the safest guides; the indigestion, the periodicity and the definite cycle of pain. In infants one is denied this help. The child is often fed irregularly and on odd materials, and the pain of hunger especially and that due to the existence of a duodenal ulceration, are indistinguishable. The

vasosensory and vasomotor reflexes are in the infant, to me at any rate, of no assistance. X-rays so far have not helped me. Appearances vary much even in health ; the duodenal cap is not formed in infants, and, the ulcers being small and acute, there is neither deformity nor residue of barium in the ulcer.

Yet all our information goes to prove that we have in duodenal ulcer a real clinical entity, and probably with more accurate observation we shall be able to make in the near future an earlier diagnosis. Neff³ has recently stated that successful gastro-enterostomy has already been formed in some such cases, but gives no reference to support his statement.

REFERENCES.

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² Theile, P., *Ergebn. der Inn. Med. und Kinderheilkunde*, Berlin, 1919, XVI, 302.
³ Neff, F. C., *Clinical Pediatrics*, N. Y., VIII, 147.