ACUTE ULCERATIONS OF THE STOMACH IN CHILDREN

By Benjamin Rice Shore, M.D.
of New York, N. Y.

FROM THE SURGICAL SERVICE OF THE BABIES HOSPITAL OF NEW YORK

Acute, fatal ulcerations of the stomach in children are sufficiently rare to warrant the report of the following case, with a review of the literature of the condition.

Case Report.—C. C., female, aged twenty-two months, was admitted to The Babies Hospital January 26, 1930, with the complaint of frequent vomiting during the previous thirty-six hours. She was the only child of young and normal parents. She was a full-term baby, vertex presentation, and weighed seven and one-half pounds. The history failed to disclose any past serious illnesses, injuries or operations. There were no abnormal symptoms referable to the stomach or duodenum.

Present illness.—Two days before admission the child was slightly flushed and seemed feverish although she continued to be playful and ate well. At night, however, she was restless. The day before admission, she started to vomit and refused to take food. All fluids given by mouth were immediately vomited. The vomitus was always watery and never contained blood or fecal material. The baby slept poorly and was very restless although the mother thinks that the child had no fever. On the day of admission the mother noticed that the child's abdomen was swollen and tender but she never complained of abdominal pain. There had been no defecation for two days. An enema the night before admission and another on the day of admission gave small fecal returns. For several days the baby had had a running nose but there were no other symptoms of an upper respiratory or pulmonary infection. There were no genito-urinary symptoms.

Subsequent questioning of the father revealed the fact that three or four days before admission the child was playing with a glass bottle which broke. When the parents tried to assemble the pieces it was found that a small portion was missing. The baby had the habit of putting all sorts of things in her mouth and it is the belief of the parents that she may have swallowed this missing piece of glass.

The patient was a well-developed and well-nourished female of twenty-two months. The skin was pale, cold and dry. The eyes were sunken. She was restless, picked at the bedclothes, and pulled at her mouth and throat. The respirations were labored and grunting but not rapid. The legs were drawn up at the hips. The head, neck, lungs and heart showed no important abnormalities. The pulse rate was 200 per minute. The abdomen was very full and distended and small dilated veins were present over the upper part. The liver dullness was completely obliterated. The liver and spleen were not palpable and no masses could be felt. There was marked dullness in both flanks and above the pubis and this dullness shifted with change of position. A fluid wave was present. No localized tenderness could be elicited although palpation seemed to cause pain. The external genitalia were normal. Examination by rectum revealed no masses or tenderness. The patellar reflexes were normal.

The respirations were 24 per minute and the admission temperature was 99.6°F. The white blood corpuscles were 8,400 per cubic millimetre, and showed 60 per cent. polymorphonuclear leucocytes, 38 per cent. small lymphocytes and 2 per cent. large mononuclears.

A milk and molasses enema given shortly after admission showed a small fecal return.

The diagnosis of peritonitis with possible ruptured viscus was made and laporatomy was decided upon. Under novocaine anæsthesia, a right rectus incision was made and the abdominal cavity opened. A quantity of air, together with about 2,500 cubic centimetre of fluid, escaped. The fluid was dark brown in color and contained numerous food particles, curds of milk and globules of oil. The peritoneum was everywhere injected but there was no purulent exudate. The small intestines and colon were collapsed and contracted. The appendix was normal except for peritoneal injection. The lesser peritoneal cavity was bulging with undigested food.

The child ceased breathing during the exploration of the abdomen. The anterior stomach wall was opened and a perforation about three centimetres in diameter was found on the posterior wall of the fundus. The edges were ragged, soft, frayed-out and deeply hæmorrhagic. No foreign body was found. Sections for microscopic study

FIG. 1.—Photograph of a portion of the stomach. On the upper border is a part of the ulcer, showing the thin, hæmorrhagic and frayed-out edges.

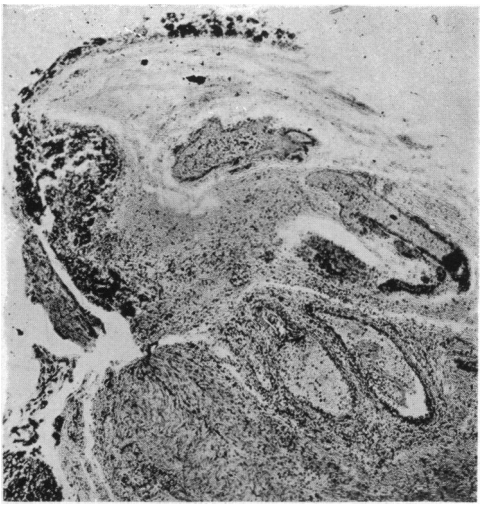

FIG. 2.—Photomicrograph of a section taken from the border of the ulcer at operation. Necrosis of the epithelium, together with œdema and marked polymorphonuclear infiltration of the submucosa is shown. The vessels contain fresh thrombi.

were cut from the edge of the perforation and fluid from the peritoneal cavity was saved for chemical analysis.

"The *autopsy* was done by the medical examiner who found nothing pathological in the heart and lungs. The peritoneal cavity contained a large quantity of fatty fluid with kernels of yellow corn. The stomach showed a round hole five centimetres in diameter in the posterior wall near the fundus (Fig. 1). Surrounding the ruptured area there was a dark red hæmorrhagic zone, but beyond that the mucosa was apparently normal. No foreign body was found in the stomach, intestines or abdominal cavity. The intestines were normal throughout and no blood was present. The liver was moderately fatty. The spleen weighed eighty grams. It was enlarged, firm, with prominent Malpighian bodies. The thymus was not enlarged. It weighed twelve grams.

Microscopic examination of a section which included the hæmorrhagic edge of the tear and a narrow adjacent area of gastric wall (Fig. 2), which appeared to be normal, revealed necrosis of the mucosa; swollen, œdematous submucosa in which lay several thrombosed vessels. In the adjacent area of mucosa thrombosed capillaries were present

between the glands which were in various stages of disintegration, merging into a normal mucosa. The muscle coats were œdematous and infiltrated with polynuclear leucocytes which were also present in large numbers around congested blood-vessels in the submucosa. A fold of mucosa with normal glands showed œdema of its submucosa and congestion of its vessels extending beyond the area of thrombosis.

Another section taken at the medical examiner's autopsy showed a fibrinous exudate on the serosa and polynuclear infiltration of all the coats with marked œdema and a fairly well-maintained mucosa.

This looks like an acute ulcer with secondary inflammation of the gastric wall, although the presence of a previous gastric lesion cannot be excluded." (Dr. Martha Wollstein)

The subject under discussion is acute ulceration of the stomach in children under ten years of age. By ulceration we mean a destructive process which involves not only the mucosa but also a portion of the submucosa and muscularis and which in certain instances may progress so as to involve the serosa. Erosions on the other hand never extend beyond the mucosa. It is this question of depth which separates the erosions and the true ulcers into two large and distinct groups.

Ulcers can further be divided into those which are acute and those which tend to run a chronic course. The acute ulcers are sudden in onset, produce marked and often alarming symptoms in previously supposedly well children and progress rapidly, either to healing or to perforation. Severe hæmorrhages are common.

The chronic ulcers of childhood are entirely different. They are similar pathologically to the ordinary chronic ulcers of adult life and also give rise to the same group of chronic but often more mild gastric symptoms. These chronic ulcers of childhood show the same indurated, infiltrated and sharp edge which is typical of those of adult life. The base is often composed of only a thin layer of muscle and serosa and perforation is not uncommon. The latter, however, differs from the perforations of the acute ulcers in that it is usually small and is situated in the center of a well-formed crater. In this paper, chronic, indurated gastric ulcers of childhood will not be discussed.

Etiology of acute ulcers.—Hæmorrhagic erosions of the mucosa and acute ulcerations of the stomach wall have long been considered to be closely related conditions. In both there is a local disturbance of circulation which in the true erosion is limited to the mucosa. In the ulcerative process, the vascular lesion extends deeper and involves the underlying layers of the stomach wall. To this disturbance of circulation is added complete or partial digestion of the infracted area. The sequent of events, therefore, in acute ulceration of the stomach is hæmorrhage, followed by gastric digestion. Because of the importance which digestion plays in the production of these necrotic lesions, Virchow [1] proposed to call them "corrosive ulcers." Simple and uncomplicated autolysis of the stomach wall is not to be considered.

The causes of the vascular disturbance are another and more obscure matter. They will be discussed separately under the six headings of con-

gestion, embolism, thrombosis, direct vascular injury, vascular diseases and diseases of the nervous system.

I. *Congestion.*—Congestion of the arterioles and venules which is so extreme as to cause rupture followed by hæmorrhage into the tissues is probably the most important single cause of erosions and acute ulcerations of the stomach. The high incidence of such lesions in new-born infants points to the fact that the extreme congestion of all of the abdominal vessels associated with the process of birth is shared by the smaller vessels of the stomach and duodenum and in some of these the pressure is sufficient to cause rupture. In Theile's [2] series of 248 cases of ulcers of the stomach and duodenum in children, 138 occurred in the first year and of these, 83 or one-third of the entire series were found in the first month of life. Butka [3] in 1927 was able to find only three cases of ruptured gastric ulcers in infants under two months of age reported in the literature, although Theile,[2] as early as 1919, was able to collect thirteen such cases. The average age of twelve of these patients was five days, while the thirteenth was seven weeks old. Later in life congestion can be caused by numerous other factors, notably heart disease and cirrhosis of the liver, but ulcerations of the stomach in children with these diseases is not a common or noteworthy finding at autopsy. Talma [4] was able to produce hæmorrhages in the gastric mucosa of rabbits by simple over-distention of the stomach. Following this, there was softening due to digestion with subsequent necrosis and perforation. In an admirable paper by Busch [5] on so-called spontaneous rupture of the stomach, published in 1924, it is pointed out that over-distention or acute dilatation is the commonest predisposing cause in these cases and that the majority have occurred in harvest time due to over-eating of easily fermented fruits and undue consumption of beer. Busch at the same time stresses the fact that other possible causes of rupture, such as carcinoma, chronic ulcers, inflammation of the vessels and thrombosis, must be eliminated before spontaneous rupture of an otherwise normal stomach can be considered. In the patient here reported, thrombosis of the vessels does exist, although there is every reason to believe that over-distention was also present.

II. *Embolism.*—Embolic occlusion of a vessel in any part of the body is possible from any given source. That such lesions in the stomach can occur cannot be denied, or even doubted, although their incidence, judging from the recorded cases, is probably low. Theile [2] collected five cases of ulcers of the stomach in children which appeared to be related to infectious processes in other parts of the body. Two of these were in patients with pneumonia and one each in patients with typhoid fever, measles and scarlet fever. In a series of 165 cases of hæmorrhagic erosions of the stomach which Beneke [6] reported, 66 per cent. were found in non-septic individuals. Schwartz,[7] in 1929, reported three cases in which punched-out or indefinite openings were found in the stomach. The edges of all three of these were exactly as if a hole had been punched out of an area of indefinite inflammatory tissue. In discussing these, he says that it is difficult to speak of an early ulcerative

process and it is better to consider it at a circumscribed necrotic change, the result of rapid and increasing disturbance of nutrition or blood supply to a localized area, the cause most likely being an embolus. No proof for this assumption is given.

Ulceration of the stomach secondary to emboli from an active endocarditis has not been observed in children.

Emboli from the rapidly thrombosing umbilical vein have been considered by Landau [8] to be the cause of the vascular lesions of the stomach and duodenum which have already been mentioned as being frequent in very young infants. He traces these emboli from the venous circulation through a patent ductus botali into the systematic arterial circulation. Such an hypothesis is tenable, although highly speculative.

III. *Thrombosis.*—At the present time, little is understood of thrombosis in any part of the body and a discussion of the various theories of its causes and predisposing factors need not be entered into here. Retrograde thrombosis from other lesions in the peritoneal cavity is perfectly possible and is rather frequently observed following acute suppurative appendicitis. In these cases it need not necessarily be a suppurative process.

Theile [2] devotes considerable space to chronic gastric ulcers occurring in marasmic and weak infants, although no definite exciting cause is mentioned. In four of his collected cases there was perforation of the ulcer. In the 65 cases of duodenal ulcer in Holt's [9] series in which the age was given, 70 per cent. of the ulcers occurred between the ages of six weeks and five months, the greatest incidence being between the sixth and tenth week. Holt says that "the age incidence is very striking. It corresponds very closely with the age incidence of deaths from marasmus." Can not these be due to thrombosis in veins filled with blood which is stagnated as the result of a weakened and inadequate circulation?

Thrombi are present in the vessels of the stomach wall in the case here reported. Whether they are primary or were formed secondary to some underlying and more fundamental process is a matter of opinion only. Definite causes of thrombosis, such as we know them to be, are, in this patient, lacking. We know of no altered chemical reaction of the blood, altered metabolism, slowing of the circulation or injury to the intima of the vessels, although it is futile to deny any or all of these. Such a discussion leads to no end. It is, however, the fact that thrombi were demonstrated in this patient in the vessels in the region of the destructive process in the wall of the stomach which I wish to record.

IV. *Vascular Disease.*—Diseases of the vessel walls can be dismissed without discussion. In adults, changes due to endarteritis and endophlebitis or to hyaline, amyloid and fatty degeneration of the vessel walls might possibly play a rôle in the etiology of ulcerative lesions in the stomach, but in children these changes are of the utmost rarity and the possibility of their occurrence is mentioned only for the sake of completeness.

V. *Direct Injury to the Mucosa.*—Direct injury to the gastric mucosa and underlying blood vessels by caustic and acid solutions, foreign bodies and coarse particles of food must always be kept in mind. These are things which are easier to prove than they are to disprove. In the patient reported, there is a definite history of her having had access to broken pieces of glass three or four days before her death, but at operation and autopsy no foreign bodies could be found, although a careful search for these was made. Chemical analyses of the free fluid in the peritoneal cavity failed to disclose the presence of poisons and there was nothing else in the findings to suggest the ingestion of such substances.

Under the heading of local injuries of the mucosa of the stomach must be noted the tuberculous ulcers which are probably due to the ingestion of tubercle bacilli. Theile [2] collected twenty-one such cases of tuberculous ulcers in the stomach and duodenum of children. One of these in the stomach had progressed to perforation.

VI. *Nervous Influences.*—The neurogenic theory of the origin of vascular occlusion is the one which is included as the final "catch-all" for any case which will not with ease fit into the other groups. It need not detain us long. Spasm in health or disease may occur and this may presumably be limited to certain local areas in the stomach wall. Hauser [10] mentions brain diseases under the causes of hæmorrhagic erosions, but the exact proof of their relation to these changes is not given.

Course.—Hæmorrhagic erosions of the mucosa of the stomach in infants is of rather frequent occurrence and the majority of these progress to healing. Acute ulcerations, on the other hand, are more violent in onset, involve a greater area and depth of tissue and, as a rule, run an acute course, leading in a short time to perforation (Hauser [10]). This tendency to perforation led Rokitansky [11] in 1842 to suggest the name of "perforating stomach ulcers" for these acute lesions. Theile [2] and Hauser [10] both think that in some instances the reparative process may be sufficient to turn these sudden and destructive lesions into ordinary ulcers of the chronic type.

SUMMARY

An acute, fatal, perforated ulceration of the stomach wall is reported in a female infant of twenty-two months of age. The history of ill-health for only two days, the site of the ulcer on the posterior wall near the fundus of the stomach and the lack of all signs of a chronic ulcer lead one to assume that the changes noted are of an acute and rapidly destructive nature. The markedly hæmorrhagic edges of the necrotic area suggest that the primary lesion was one of hæmorrhage, either from local injury, overdistention, or vascular thrombosis, and that gastric digestion of this area caused the perforation. This case is of especial interest in that the perforation was found and measured at operation before post-mortem digestion became manifest.

BIBLIOGRAPHY

[1] Virchow: Quoted by Hauser.
[2] Theile, P.: Ergebnisse der inn. Med. und Kindheilk, vol. xvi, p. 303, 1926.
[3] Butka, H. E.: J.A.M.A., vol. lxxxix, p. 198, 1927.
[4] Talma, S.: Zeitsch. f. klin. Med., vol. xvii, p. 10, 1890.
[5] Busch, M.: Frankfurter Zeitsch. f. Path., vol. xxx, p. 30, 1924.
[6] Beneke, R.: Verhandl. d. Deutsch. Path. Ges., 12th Day, p. 284, 1908.
[7] Schwartz, I. S.: Zent. f. Chir., vol. vii, p. 1057, 1929.
[8] Landau, L.: Ueber Melaena der Neugeborenen nebst Bermerkungen ueber obliteration der foetalen Weg. Maruschke u. Berendt, Breslau, 1874.
[9] Holt, E.: Am. Jour. Dis Child., vol. vi, p. 381, 1913.
[10] Hauser, G.: Handb. d. Spec. Path. Anat. u. Hist., Henke u. Lubarsch, vol. iv, part 1, p. 339, Berlin, 1926.
[11] Rokitansky: Quoted by Hauser.