STOMACH TEARS IN THE NEWBORN


DISCUSSION

Dr. Clinton M. Cavett (Jackson, Mississippi): First, I need to just mention how indebted we are to Dr. Shaw, a member of this Association, who finally put to rest the idea that a congenital defect was responsible in these perforations.

I wish to point out another aspect of the considered cause in this type of perforation of the stomach. Dr. Touloukian and Dr. Lloyd have been most vocal in explaining the ischemic cause in this process; that is, the selective shunting of blood away from the splanchic vascular bed as a response to hypoxic stress. As was pointed out by Dr. Houck, the vast majority of these patients fall into a category of the premature infant who is already undergoing some type of hypoxic stress, such as delivery suite resuscitation, respiratory distress, or severe congenital heart disease with shunting.

This hypoxic stress, when occurring in the newborn, sets the stage for the ischemic damage. Adding the ischemic damage to the hyperacidity known to occur in the first few days of life offers another explanation as to this catastrophic event.

I must mention that this sequence of events is not seen in the older child or adult. In Dr. Touloukian’s lab—and we have begun to confirm this in our own laboratory—ischemic stress in the neonatal piglet leads to damage to the fundus and cardia of the stomach.

For completeness, I need to mention the pneumoperitoneum seen in this same group of patients who are on high-pressure ventilation for hyaline membrane disease. Ventilating these stiff lungs with high inspiratory pressures can produce pneumomediastinum, which will decompress by way of the peritoneal cavity. Therefore, a neonate on high-pressure mechanical ventilation can develop pneumomediastinum and/or pneumothorax, and, later, pneumoperitoneum. And these are patients which must be identified, and not rushed to the operating room. As Dr. Houck pointed out, aspiration of the large pneumoperitoneum and close observation are indicated here.

As must be pointed out, these patients are also in the same group who can develop the spontaneous gastric perforation.

Dr. Anthony Shaw (Charlottesville, Virginia): My interest in this uncommon problem was kindled by several cases of neonatal gastric rupture I encountered during my training in New York.

(slide) This is typical of seven cases we reported in 1965. Most of the infants we saw and whose case records we reviewed presented not with vomiting, but with respiratory distress, cyanosis, and abdominal distention. In this film you can actually see, quite well, the site of perforation on the greater curvature.

(slide) Grossly, these lesions look like tears, or blowouts, with ragged, thin edges. Histologically, the edges consist of mucosa and submucosa only, which you can detect in that infant.

On the basis of similar observations, Herbut, a pathologist at the University of Pennsylvania proposed a congenital gastric muscle defect as the cause of spontaneous gastric perforation in 1943. By 1962, when we saw this baby, this had become the prevalent theory of the cause of spontaneous gastric perforation. To us, these lesions look as if the stomach had blown out, or torn, with retraction of the seromuscular layer away from the mucosal perforation, which is a phenomenon familiar to all surgeons who have seen the results of distended, hollow viscera.

In the laboratory, we ran air into the stomachs of anesthetized newborn puppies, with cardia and pylorus occluded, until gastric rupture occurred. The lesions were not only identical grossly and histologically to those so-called spontaneous ruptures described by Herbut and others, but also to those seen by ourselves and others, in infants and adults where the cause of rupture was clearly pneumatic distention, such as situations where vigorous mask ventilation was carried out in babies with TE fistulas, babies with distal obstruction, and so on.

(slide) In the laboratory, this is a typical gastric perforation from overdistention. I might say that these perforations occurred in any part of the stomach. They were seen in the greater curvature, the lesser curvature, proximal, distal, anterior, posterior.

(slide) And note the curled edge of this defect. On the posterior surface of the same stomach is seen another area where there was a split in the seromuscular layer, but the mucosa was still intact.

(slide) Sections from the edge of the defect showed retraction of the muscle well back of the overhanging mucosal edge. Histologically, this looked exactly like the specimen in our infants, with retraction of the muscle back here, and the overhanging mucosal edge.

(slide) This is from an area of thinning, where the mucosa had not disrupted, but the seromuscular layer had.

(slide) Another area of the stomach of one of the same dogs, where the retractions had occurred and the mucosa was beginning to give.

(slide) And these are the findings in a case—this slide was provided to me by Dr. Peter Kottmeier, at the State University of New York Downstate—of an infant he operated on, where the split had occurred in the seromuscular layer, and the mucosa hung through as a huge pseudodiverticulum. He had actually caught the rupture before disruption of the mucosa took place.

The purpose of these remarks is to indicate that the observations my colleagues and I reported 15 years ago strongly suggest that, whatever the exact mechanism—and Dr. Houck has told us an in-
teresting one today—spontaneous gastric perforation results from pneumatic gastric rupture, and not from congenital absence of mus-
cles, congenital newborn disease, and, with apologies to Dr.
Touloukian and Dr. Cavett, I don't believe results from ischemic ne-
crosis—that is, the same disease that causes necrotizing enterocolitis
—either.

In our laboratory studies, we found that, as Dr. Houck has pointed
out, it takes great distending pressure to blow out a normal newborn's
stomach, although it can be done. I would therefore agree with Dr.
Houck that in many cases of spontaneous rupture it seems likely
that an additional force, beyond the pressure of ingested gas and
fluid, is necessary to achieve the pressure required to blow out the
normal newborn's stomach.

Dr. Houck's hypothesis that such great intra-gastric pressure
may result from a combination of distention and incoordinated vomit-
ing efforts, although highly speculative, follows nicely from what is
known about neonatal esophageal and gastric physiology, and is
probably as good an explanation of the events leading to at least some
cases of spontaneous rupture as we are likely to get for a while.

I am uncomfortable, however, with the fact that in many reported
cases, and in my own experience, vomiting or retching were never
observed prior to the onset of massive pneumoperitoneum. Kott-
meier proposed that some infants whose stomachs are distended
with amniotic fluid are compressed during the birth process, with
splitting at the vermiscolic line in the stomach, producing a mu-
cosal pseudodiverticulum such as the one you just saw in this slide.
Over the next few days, the mucosa gives way, while the infant
is being fed, producing the typical signs of gastric rupture on the
second, third, or fourth day of life.

Just a few final comments on the diagnosis and treatment. The risks
of gastric rupture are enhanced in several settings, and must be sus-
pected in infants who develop rapid abdominal distention and de-
teriorating respiratory function, who are being ventilated by face
mask, or who have lesions enhancing gastric distention, such as tracheoesophageal fistula, or interfering with gastric emptying, such as
diaphragmatic hernia or duodenal atresia. Many of these infants with
massive pneumoperitoneum will die a respiratory death. And as
Dr. Houck correctly points out, needle or catheter paracentesis may
be life-saving.

I have operated on another half-dozen of these cases in the past
ten years, and believe that tapping the air from the belly is the most
important first step in reversing the rapid deterioration Dr. Houck
described so well. At laparotomy, the ragged mucosal rim of the
defect is excised, and the blowout closed in one or two layers. Since
perforations may be multiple, the entire stomach, front and back,
should be carefully inspected. I usually ask the anesthesiologist to
flush a little methylene blue down the nasogastric tube after I've
closed the gastric defect, to look out for other possible perforations.

Dr. J. Alex Haller, Jr. (Baltimore, Maryland): Dr. Houck, I
very much enjoyed your comments, and particularly bringing to our
attention this idea of an emetogenic type of pneumatic rupture. My
associate, Dr. White, proposed several years ago (as you men-
tioned in your manuscript) the drain trap theory, a possible cause
in which the fluid level in the stomach covers up the gastric junction,
like a drain trap, and therefore allows air to become trapped in the
stomach. If there is an obstruction downstream, the stomach may
explode.

Your concept is appealing, and I would like to modify our posi-
tion and include the drain-trap component with your emetogenic one,
because I think it would require an attempt to vomit in order to rupture
the stomach. So I would like to jump on your bandwagon, if I may, because I think your explanation comes closest to the clinical facts.

I would like to ask you whether you combine gastrostomy with the
management of these patients? We have felt that that was important:
because, whatever the cause, the stomach might rupture again:
and with a gastrostomy present after the repair, we could decompress
and avoid that possibility.

Dr. W. S. Houck, Jr. (Closing discussion): I certainly agree with
Dr. Shaw that a number of these patients do not present with vomit-
ing. If I might, I'd like to give my explanation as to why this occurs.

We know if an individual vomits if we see the gastric contents or
see the act of vomiting. I theorize that the infants with spontaneous
gastric tear vomit and, with the first act of vomiting, rupture the
stomach: they, therefore, vomit inwardly rather than outwardly, and
we never really see the act of vomiting or see the gastric contents.
So I think more of these patients vomit than we really see and record.

So far as the gastrostomy tube in these patients, I certainly agree
with Dr. Haller. We have carried out a gastrostomy in one of our
patients and I think it added to the repair.

In reviewing the literature and reviewing the different ideas and
theories as to the cause for gastric ruptures in the newborn, I came
across the name of Dr. E. P. Pendergrass. He first proposed and
projected the idea of pneumatic rupture of the stomach in 1946.