system was present in both fascicular and reticular zones. The comparatively low concentrations in the glomerulosa were due at least in part to a relative lack of specific dehydrogenase activity. One can not rule out by this method the possibility that TPN may also be low in the glomerulosa, because TPN is added in reaction I. If as has been suggested one of the roles played by this enzyme system in the cortex is to act as a TPNH generator, providing this reductant for important hydroxylations of the steroid molecule, it is reasonable that it be most active in the fascicular and reticular zones. These are the areas which seem to react most strikingly in situations of acute and chronic stress and following ACTH administration. Morphologic and histochemical criteria of response to stress including ascorbic acid depletion, lipid and cholesterol depletion and hypertrophy all appear to involve the reticular and fascicular zones(6). It has not been demonstrated that the glomerulosa participates actively in these adjustments, at least in the early stages. This zone appears to react demonstrably in a specific fashion to alteration in electrolyte concentration. In the light of our present knowledge it would seem that the fascicular and reticular zones are the areas of the cortex where increased synthesis of steroids occurs in response to acute and chronic stress, and it is here that one would suspect a requirement for increased TPNH and potential TPNH generator systems.

Summary. A histochemical technic was used to localize the activity of glucose-6-phosphate dehydrogenase system in zones of the adrenal cortex. It was very active in the fascicular and reticular zones. Activity in the glomerulosa was comparatively low. Since this enzyme system may be indirectly influenced by ACTH and is a potential provider of TPNH, a reductant important in steroid synthesis, demonstration of its predominance in a zone where active steroid synthesis is presumed to occur is reasonable and of considerable interest.

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Pathway of Enterogastric Reflex.* (24960)

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That acid in upper small intestine inhibits emptying of stomach was first described at end of 19th century by Serdjukov in Pavlov's laboratory(1). This phenomenon present in dogs exists also in human subjects (Barsony and Egan(2)). Thomas and co-workers(3,4, 5) pointed out that delay in gastric evacuation was not due to pylorospasm, but to reduction in motility of the whole stomach. They found the mechanism undisturbed by splanchnicectomy but reduced by division of the vagi. The name "enterogastric reflex" was coined for the phenomenon observed. Quigley *et al.*(6,7) reported similar findings in that vagotomy elevated the threshold for acid inhibition of the stomach. In human subjects we found the inhibitory effect of acid in the duodenum or upper jejunum, as measured by balloon-transducer-recorder system, persisted after complete supradiaphragmatic vagotomy, thoracolumbar sympathectomy, partial gastric resection, or transection of

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TABLE I. Inhibition of Gastric Motility by Acid in Jejunum.

Vol 0.1 N HCl (cc)	Duration of inhibition (min.)
10	1– 5
20	5–16
30	10–35
40	13–30
50	25–40
60	22–40
70	28–55
80	55–60
90	40
100	30–55

TABLE II. Effect of Vagotomy on Enterogastric reflex.

Vol 0.1 N HCl (cc)	Duration of inhibition (min.)
10	4– 6
20	5–12
30	6–18
40	8–15
50	10–21
60	10–20
70	35–40

spinal cord(8). The inhibitory effect of acid varied as a linear function of hydrogen ions introduced(9). Since vagus nerves did not seem of primary importance in the reflex arc responsible for acid inhibition of the stomach, the following experiment was performed on dogs.

Method. Sixteen dogs were prepared with Maydl fistula of proximal jejunum plus cannula gastrostomy and Heidenhain pouch. Motility was recorded from either or both vagally innervated main stomach and vagally denervated Heidenhain pouch, using a balloon introduced through cannula and connected to Statham pressure transducer and Sanborn twin-viso recorder. Acid in varying amounts was introduced through a catheter threaded into the jejunum through the Maydl fistula. Volumes of 0.1 N HCl varying from 10 to 100 cc were infused into the jejunum and duration of inhibition of gastric motility measured. In 2 dogs complete bilateral thoracolumbar sympathectomy was performed. In 8 dogs bilateral supradiaphragmatic vagotomy was done. In 3 dogs both vagotomy and thoracolumbar sympathectomy were completed. In 3 additional animals the celiac plexus was destroyed by dissecting clean the celiac and superior mesenteric arteries and the adjacent aorta, followed by application of 5% phenol solution to the area. Following these procedures in 16 dogs the same doses of 0.1 N HCl were infused into the jejunum while gastric motility was recorded.

Results. Control studies in 16 animals demonstrated that, as in the human, duration of gastric inhibition increased progressively with amount of HCl infused into the jejunum. Increases above 70 cc however, did not significantly increase period of inhibition. The results are summarized in Table I and an example presented in Fig. 1. In each of 32 studies performed, the same volume of 0.9% NaCl solution infused into the jejunum at the same rate failed to produce inhibition of gastric contractions. Duration of inhibition observed when recordings were made from the Heidenhain pouch was not significantly different from that observed in the main stomach, even where simultaneous recordings were made. The latent period between infusion of HCl and gastric inhibition varied from 10 to 30 seconds.

In 8 vagotomized dogs, 29 studies were performed following complete recovery. Inhibition of gastric motility was observed in all studies. The latent period between infusion and inhibition varied from 1 to 4 minutes, a slight increase over that observed in control studies. Duration of inhibition was, however, only slightly less than that seen with same volumes of infusion in the unvagotomized animals. The studies are summarized in Table II.

Eight studies were performed in 2 dogs following bilateral thoracolumbar sympathectomy. Latent period and duration of inhibition were unchanged over those observed in control studies on the same animals. Acid infusions in dogs with both vagotomy and lumbodorsal sympathectomy were entirely similar to those with vagotomy alone.

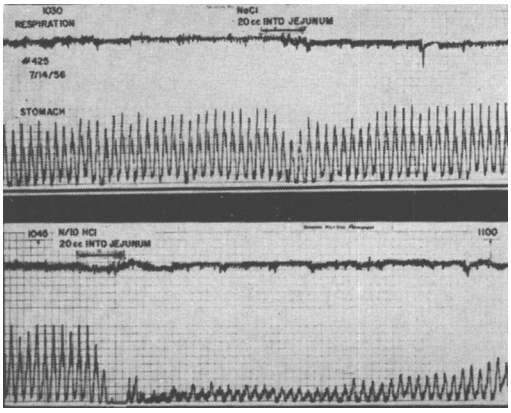

FIG. 1. Inhibition of gastric motility by acid in jejunum.

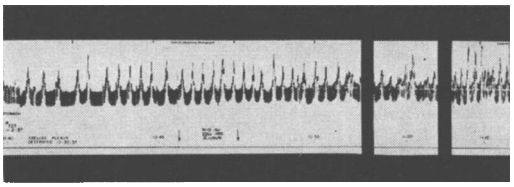

FIG. 2. Failure of acid in jejunum to inhibit gastric motility after destruction of celiac plexus.

Following celiac ganglionectomy, 8 studies were performed in 3 dogs with infusion of 10 to 40 cc of 0.1 N HCl into the jejunum. In no experiment was inhibition of gastric contractions observed. A typical example is presented in Fig. 2. When 10 cc of olive oil was infused into the jejunum, however, profound and prolonged inhibition of gastric motility was observed after latent period of 10 to 20 minutes.

Discussion. Inhibition of gastric motility as recorded from a Heidenhain pouch after infusion of dilute HCl into a jejunal fistula, was entirely comparable to that observed when contractions were recorded from the main stomach. Since the Heidenhain pouch is without vagal innervation, this observation indicates the vagi are not implicated in the enterogastric reflex. Following bilateral supradiaphragmatic vagotomy, the same inhibition from HCl in the small intestine was observed although latent peirod was longer and period of inhibition slightly less. This difference might be explained by general decrease in tonus and motility of stomach following complete vagal section. Complete bilateral thoracolumbar sympathectomy did not influence the enterogastric reflex and even when combined with bilateral vagotomy the phenomenon persisted. These data indicate that pre-ganglionic sympathetics and their spinal cord connections are not involved in the pathway of the enterogastric reflex.

Celiac ganglionectomy completely abolished inhibition of gastric motility resulting from 0.1 N HCl infusions into the proximal jejunum. These observations compare with those of Simba(10), who produced gastric inhibition by application of hypertonic saline to intestinal loops. This effect also was not influenced by vagotomy or sympathectomy, but was blocked by celiac ganglionectomy. This indicates that gastric inhibition resulting from intestinal hyperperistalsis, and perhaps intestinal distention, probably has the same nervous pathway through the post-ganglionic sympathetics as does acid inhibition.

Summary. Acid in upper small intestine causes immediate inhibition of gastric motility. Sixteen dogs were prepared with gastric fistula and/or Heidenhain pouch plus a Maydl fistula of upper jejunum. After control studies, the animals were (1) vagotomized; (2) totally sympathectomized; (3) both vagotomized and sympathectomized or (4) celiac ganglionectomized. Inhibitory effect of acid in the jejunum on gastric motility was still present after either or both vagotomy and sympathectomy. However, no inhibition was produced after celiac ganglionectomy. This study indicates the pathway for enterogastric reflex is through the post ganglionic sympathetics.

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