Vagotomy and Acid Secretion

KOHTA YATSUKA, HIRONARU FUKUSHIMA, HIDEAKI YAMANA
HIROYOSHI MIZOTE AND TERUO KAKEGAWA

*Department of Surgery, Kurume University School of Medicine,
Kurume, 830 Japan*

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Vagotomy is widely considered to be effective in eradicating acid secretion by the stomach. In actuality, severing the vagus nerve only reduces the acid secretory responses to pentagastrin by approximately 50 %. The cephalic, gastric and intestinal phases of acid secretion probably have both nervous and hormonal components which work simultaneously. This means the Hollander test which is based on the theory that insulin stimulates acid secretion solely via the vagus nerve, is not a reliable method to determine vagal continuity.

Vagotomy is an important procedure for reducing acid output however its effectiveness has been exaggerated. The purpose of this discussion is to clarify the effect that vagotomy will have on gastric secretions.

First a brief review of the vagal nerve function is necessary. The vagus nerve provides the parasympathetic innervation to the stomach via anterior and posterior trunks. It has both excitatory and inhibitory influences on the motor and secretory functions of the stomach.

There are also extragastric branches of the vagus which innervate the liver, gall-bladder, pancreas and intestine and have minor effects on the stomach.

Gastric secretion is not entirely under vagal control, as Pavlov considered it to be. The discovery of gastrin by Dr. J.S. Edkins (1906) led to the idea that the cephalic phase of acid secretion was mediated

by the vagus nerve and the gastric phase was mediated by the release of gastrin from the stomach. Acid secretion is currently divided into cephalic, gastric and intestinal phases depending upon the site at which the secretion is initiated.

This should not imply that only one mechanism of acid secretion is involved in each phase. Rather, the three phases appear to have both nervous and hormonal components that are interrelated. In view of this discussion, what can be accomplished by vagotomy? Vagotomy does cause a marked and permanent decrease in basal acid secretion and a decrease in the responses to histamine and pentagastrin.

Acid secretion falls to its lowest level three months postsurgery and tends to increase slightly during the next two years. It remains constant after this period at a level of approximately 50 % of the maximum acid output in response to pentagastrin (Greenall et al. 1975).

It is also interesting that the various techniques of performing the vagotomy, including truncal, selective, and proximal gastric, all produce approximately the same reduction of acid secretion (Grossman, 1974).

When vagotomy is combined with antrectomy of the stomach, the maximal acid output response is reduced by 85 % (Sleisenger et al. 1978).

Even though severing the vagus suppresses acid secretion by approximately 50

%, it has been demonstrated that there is no change in the capacity of the cells to secrete acid. Prevagotomy levels of responsiveness to histamine and pentagastrin can be restored by giving a small dose of a cholinomimetic drug, such as carbachol. This is a pharmacological method of restoring the cholinergic influence that was removed by vagotomy (Roland, 1975). For recurring ulcer disease, after vagotomy, the physician must determine the degree of vagal innervation. At the present time there is no independent test for determining the completeness of the vagotomy.

The assumption that insulin acts solely through the vagus neurons is widely accepted. If acid secretion increases in response to insulin after vagotomy, it is considered to be an incomplete vagotomy. This assumption is unproven. It is based on Man's observation in dogs that vagotomy permanently abolishes the acid secretory effect of insulin and the assumption that the same is true in man. The following are some theoretical explanations for the persistent response to insulin after vagotomy.

I. Incomplete vagotomy

- A. Uncut uninjured fibers
- B. Uncut injured fibers that recover
- C. Cut fibers that regenerate
 - 1. End to end
 - 2. By sprouting

II. Nonvagal mechanisms

A. Neural nonvagal mechanisms: latent nerve paths (Splanchnics) become operative after the primary vagal paths are cut.

B. Non-neural, nonvagal mechanisms: for example, release of epinephrine by hypoglycemia may release gastrin which stimulates acid secretion (Grossman, 1974).

Soon after vagotomy, a small increase in acid production can be induced by insulin in 20 % of the patients. This increases to 70 % at two years postvagotomy. The

percentage remains fairly constant after two years (Lyndon et al. 1975). Large responses to insulin after vagotomy are usually due to the failure to cut major nerve branches. Most of the query relates to the small responses. It is possible that incomplete vagotomy and a positive response to insulin are not related (Grossman, 1974).

Since only 10 % of the vagotomized patients get recurrent ulcers, but 70 % respond to insulin, the insulin test has no practical value in identifying patients likely to have recurrences (Slisenger et al. 1978).

In summary, vagotomy is an important procedure for reducing acid output, although its effectiveness is often over-estimated. The cephalic, gastric and intestinal phases of acid secretion probably involve both nervous and hormonal components that work simultaneously. Thus it is difficult to accept the theory that insulin stimulates acid secretion solely via the vagus.

Finally, there is no reliable method available at present for assessing vagal continuity, although the Hollander test is widely acclaimed for this. When acid secretory testing is necessary, the information gained from safe tests, such as pentagastrin stimulation, is as valuable as that gained from insulin administration (Kronborg, 1973).

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