Evaluation of the Grassi Test for the Completeness of Vagotomy

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SUMMARY

This article describes our experience with the Grassi test in the experimental laboratory, using dogs. We found the reliability of this test questionable for completeness of vagotomy.

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Since Dragstedt popularised vagotomy in 1943, this procedure has become a cornerstone in the surgical treatment of peptic ulcers. However, the high rate of incompleteness of vagotomy as shown by postoperative Hollander test (20 - 30%) poses a major drawback. The need for a reliable intra-operative test for complete vagal section is self-evident.

The requisites for such a test include the following: it must be safe and inexpensive; it must not unduly prolong the operative procedure; and it must be absolutely reliable.

Tests Presently Available

1. Electrical stimulation test as described by Burge and Vane.[1]
2. Leucomethylene blue staining of the lower oesophageal and proximal gastric walls, for defining of nerve filaments as described by Lee.[2]
3. Grassi test[3]—based on evaluation of pH variations in the stomach and the lack of response of the acid-secreting gastric mucosa to histamine in the presence of complete vagal section.
4. Congo red test recently described by Nyhus, where staining of the gastric mucosa is gastroscopically assessed before and after vagotomy.
5. The 2-deoxy-d. glucose neutral red test described by Cole.[4]
6. The 2-deoxy-d. glucose motility test as proposed by Franks and Griffen.[5]

The Grassi Test

This test relies on the fact that histamine will stimulate gastric secretion under anaesthesia. There is a marked difference in this secretory response between innervated and denervated areas of gastric mucosa. (Background vagal tonus enhances the response to humoral stimulation.)

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METHODS AND RESULTS

The dogs are anaesthetised by intraperitoneal Sagital, intubated and ventilated with oxygen. At operation the abdominal vagi are identified and isolated on tapes. After gastrostomy and thorough cleansing of intragastric contents, a small cone-shaped metal frame is inserted through the gastrostomy incision, keeping the surfaces of the stomach apart. The oesophageal and pyloric openings are tamponaded to prevent soiling of the stomach. The resting stomach juice is cleared by careful swabbing until all surfaces of the stomach show a pH reading (Beckman pH meter) greater than 5.

Project 1

Repeated pH measurements for up to 35 minutes showed no change in pH in 5 dogs who had shown an initial pH greater than 5, thereby proving the absence of basal secretion under anaesthesia.

Project 2

Using these same 5 dogs, histamine (0,4 mg/kg) was administered intravenously. Within 5 - 7 minutes there was a marked acidic response, lowering the pH to 1 - 2. This response lasted for 15 - 20 minutes. This proves Grassi's claim that histamine stimulates gastric secretion under anaesthesia. This secretory effect was most marked on readings taken on the posterior gastric mucosae.

Project 3

Next we tried to verify Grassi's claims that this response to histamine is abolished by total vagotomy.

The vagi in 5 dogs were isolated and bilaterally sectioned in the neck. Histamine was again given and to our dismay we found that all 5 stomachs still secreted actively.

In the next 5 dogs, total vagotomy was performed subdiaphragmatically. (This was supplemented by complete section of the oesophagus for the possible intramural vagal fibres.) This procedure abolished the acid response to histamine in 3 of these dogs but, alas, vagotomy again failed to abolish secretion in the other 2.

Project 4

We then, like Grassi, did intentional incomplete vagotomies (leaving one major trunk) to see if this could be detected by this test.

In 5 dogs, only an anterior vagotomy was performed. This resulted in a drop of pH on the posterior (innervated) surfaces of these stomachs only.

In 5 dogs, only a posterior vagotomy was performed. After histamine, we found a very poor and insignificant response on these anterior (innervated) surfaces compared with the posterior (denervated) surfaces.

DISCUSSION

From our limited experience with this test, we agree with Grassi that there is no basal secretion of acid in the anaesthetised dog.

We further agree that histamine administered intravenously in the maximal dosage, quickly and effectively stimulates gastric secretion in the anaesthetised dog. An unexplained finding in this regard was that the anterior surface of the stomach in this animal responds poorly to histamine stimulation when compared with the posterior surface. We can offer no explanation for this finding.

We had more success with subdiaphragmatic vagotomy than with cervical vagotomy for total denervation of the stomach in dogs.

We have found this test unreliable for detecting the completeness of vagotomy intra-operatively in the dog. This especially applies to detection by this method of an intact anterior trunk. This ties up with our finding of a diminished responsiveness to histamine stimulation of the anterior surface in the non-vagotomised dog stomach. We were also unable to detect smaller residual fibres with this test, as claimed by Grassi, and therefore cannot propose the test for this purpose. This test adheres admirably to the stated requisites in all other respects.

This work will now be repeated in primates to see if the reliability as claimed by Grassi's work can be reproduced in these animals.

REFERENCES

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