

## APPENDIX 2

### RAISED GASTRIN AND CGA

Gastric achlorhydria, as a result of atrophic gastritis, is common. When chromogranin A (CgA) and gastrin are raised, antibodies to intrinsic factor and to parietal cells should be assessed in order to exclude autoimmune disease, the usual cause of atrophic gastritis. Another cause of atrophic gastritis, peptic ulcer disease (PUD), is common; therefore, suspicion of gastrinoma is not first-line. *Helicobacter pylori* infection, the usual cause of PUD, raises circulating gastrin.[1, 2] Eradication of *H. pylori* must be complete in order for PUD to be treated successfully. Recurrent PUD in the absence of *H. pylori* should lead to suspicion of gastrinoma. Acid suppression resulting from proton pump inhibitor (PPI) or H<sub>2</sub> antagonist drugs remains problematic.[3-5] PPIs render the stomach acid-free, resulting in raised CgA and gastrin; the diagnosis of gastrinoma therefore remains difficult, as almost all patients who are suspected of having a gastrinoma are already receiving PPI therapy. Withdrawal of PPI in such patients should be undertaken with much care and preferably under hospital conditions, as upper gastrointestinal bleeding or perforation may rapidly occur. Withdrawal of PPI is necessary for 10 days to two weeks to confirm the diagnosis. H<sub>2</sub> antagonists may be used to reduce gastric acid secretion during this time, but it is advisable for these to also be interrupted 48 hours prior to the test.

### REFERENCES

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