	GastroPanel and GastroSoft	^{13}C urea breath test or Stool antigen test
In the diagnosis of		
Functional dyspepsia	YES When GastroPanel results are normal, symptoms are often caused by functional dyspepsia or other disease not involving the gastric mucosa	NO
***H. pylori* infection** (chronic gastritis)	YES	Limited Sensitivity[1]
Atrophic gastritis (damaged and severely dysfunctional gastric mucosa of the corpus or antrum or both)	YES	NO
The risks (due to atrophic gastritis) of		
Gastric cancer (in antrum and / or corpus)	YES [2]	NO
Vitamin B12 deficiency (corpus)	YES	NO
Calcium, iron, magnesium and zinc deficiency (corpus)	YES [3]	NO
Peptic ulcer disease (antrum)	YES [4]	NO
The risks of the complications of GORD		
Oesophagitis, Barrett's oesophagus, oesophageal cancer	YES [5]	NO
If necessary, a recommendation for		
Gastroscopy and biopsy examination	YES	NO
Eradication of *H. pylori* infection	YES [6]	Limited sensitivity[1]
Determination of vitamin B12 and homocysteine	YES	NO
Determination of calcium and iron	YES	NO
Follow-up examination to monitor		
Incidence of atrophic gastritis	YES [7]	NO
Eradication of *H. pylori* infection success	YES	NOT RELIABLE[1]
Healing of atrophic gastritis	YES	NO
The functional status of the stomach		
Acid output normal – normal intragastric acidity	YES	NO
Acid output increased – high intragastric acidity	YES	NO
Acid output decreased – low intragastric acidity or achlorhydria	YES	NO
Non-Helicobacter associated inflammation		
Biomarkers upregulated by acute, active inflammatory reactions	YES	NO

1. The 13C urea breath test and stool antigen test may give up to 40 % false negative results if the patient has a) atrophic gastritis and related risks, b) MALT lymphoma or c) bleeding peptic ulcer disease or d) if the patient is currently receiving antibiotics or PPIs (proton pump inhibitors). The GastroPanel *H. pylori* IgG antibody test is devoid of such interferences.
2. The risk of gastric cancer is low in the absence of atrophic gastritis. In some cases, a *H. pylori* infection without atrophic gastritis may be associated with gastric cancer and peptic ulcer disease.
3. Proper absorption of dietary calcium, iron, magnesium and vitamin B12 as well some drugs requires normal acid secretion. Acid dysregulation (impaired acid secretion) in atrophic gastritis and in long term PPI therapy leads to calcium and other micronutrient malabsorption, leaving individuals at risk of osteoporosis and fractures. Hypochlorhydric states such as atrophic gastritis and partial gastrectomy have long been a known cause of iron and vitamin B12 deficiency as well.
4. Peptic ulcer disease does not develop in patients with corpus atrophy (no acid, no ulcer).
5. Normal or high Pepsinogen I and / or Pepsinogen I to Pepsinogen II ratio in conjunction with low Gastrin-17 (below 1.0 pmol /l) may indicate high acid (HCl) output and high intragastric acidity. Risks of gastro-oesophageal reflux disease (GORD) increase.
6. Pepsinogen II level below 10 µg /l two months after the treatment indicates that the *H. pylori* eradication is successful. Increased level of Pepsinogen II (over 10 µg /l) indicates active *H. pylori* gastritis or inflammation due to the use of non-steroidal anti-inflammatory drugs (e.g. aspirin) or strong alcohol.
7. When the incidence of *H. pylori*-related atrophic gastritis is monitored, the patient can be offered targeted, safe treatment at the right time. The need for medication and the costs and adverse effects of medication can thus be reduced. If the patient has been diagnosed with peptic ulcer disease (gastric or duodenal ulcer), *H. pylori* should be eradicated. If the patient has atrophic gastritis, *H. pylori* eradication is mandatory. The patient and the physician may agree on eradication treatment for other reasons, for example, when patients' relatives have been diagnosed with gastric cancer.