Gastroenterology

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Within the ANS, the gastrointestinal (GI) tract is uniquely innervated by the PSNS. Upper and lower GI disturbances are marked by excess or insufficient parasympathetic activity. PE in the upper GI tract causes an overactive stomach leading, for example, to gastroesophageal reflux disorder (GERD). Parasympathetic insufficiency is associated with gastroparesis which may also lead to GERD. An effect of PE in the lower GI tract is diarrhea, and parasympathetic insufficiency may be associated with constipation.

GERD, Gastroparesis, and Irritable Bowel Syndrome

Given that GI track motility is uniquely controlled by the PSNS, it should not be surprising that parasympathetic dysfunction should be implicated in GI upset. However, an important question would be whether parasympathetic dysfunction is primary or secondary. This dichotomy affects therapy selection and dosing and titration for the individual patient. Autonomic assessment by symptoms may also be misleading. Acid reflux, as a symptom, may be due to an overactive stomach forcing acid into the esophagus or due to an underactive stomach resulting in food displacing acid into the esophagus. The former is the result of overactive parasympathetic control and the latter (gastroparesis) the result of underactive parasympathetic control.

Irritable bowel syndrome (IBS), once demonstrated, is associated with sympathetic excess (SE), arguably due to the associated pain. IBS includes two subtypes: with diarrhea and with constipation. The subtype associated with diarrhea seems to be associated with a concurrent PE. The subtype associated with constipation seems to be associated with a concurrent parasympathetic insufficiency. P&S monitoring helps to guide therapy by differentiating underlying P and S dysfunction.

Note the pathophysiology leading to IBS may be a parasympathetic insufficiency, perhaps secondary to SE. Parasympathetic insufficiency would have caused slowed bowel motility, enabling the toxins to build up and persist for too long, possibly causing the irritation that may lead to the syndrome. Further, the pain due to IBS is a secondary effect, suggesting that SE may not be part of the cause. It is likely that either P or S dysfunction is the cause and which depends on the individual patient's history.

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