

For the Primer, visit doi:10.1038/s41572-019-0135-7

➔ **Gastrointestinal (GI) pain** — a form of visceral pain — is a common symptom of some GI disorders, such as Crohn's disease, chronic pancreatitis and irritable bowel syndrome (IBS).

## MECHANISMS

Acute pain is predominantly conveyed to the spinal cord via visceral afferent fibres, specifically via c-fibres and A $\delta$  fibres, following which, the pain signal is transmitted to the brain

## DIAGNOSIS

GI pain is often initially poorly localized, although the pain presentation can change over time. For example, pain in acute appendicitis is initially diffuse, but localizes to a specific region of the abdomen (McBurney's point) during later stages of disease. In addition, GI pain can be referred to somatic structures, such as muscle or skin, and can be accompanied by autonomic symptoms, such as diarrhoea, sweating and heart palpitations. The main aim of diagnosis of GI pain is to identify and initiate treatment for the causative disorder. Diagnostic work-up includes abdominal imaging (typically MRI or CT), laboratory tests (for example, assessing liver enzymes and amylase levels) and assessment of pain intensity using general pain questionnaires. Pain can be classified as primary pain (also known as functional pain, whereby no disease process can be identified) or secondary pain (caused by a specific disease), and as acute or chronic depending on its duration.



## EPIDEMIOLOGY

Abdominal pain is common and has been estimated to occur in up to 25% of adults in community-based, cross-sectional studies, although other studies have reported different rates. Pain is associated with many different GI

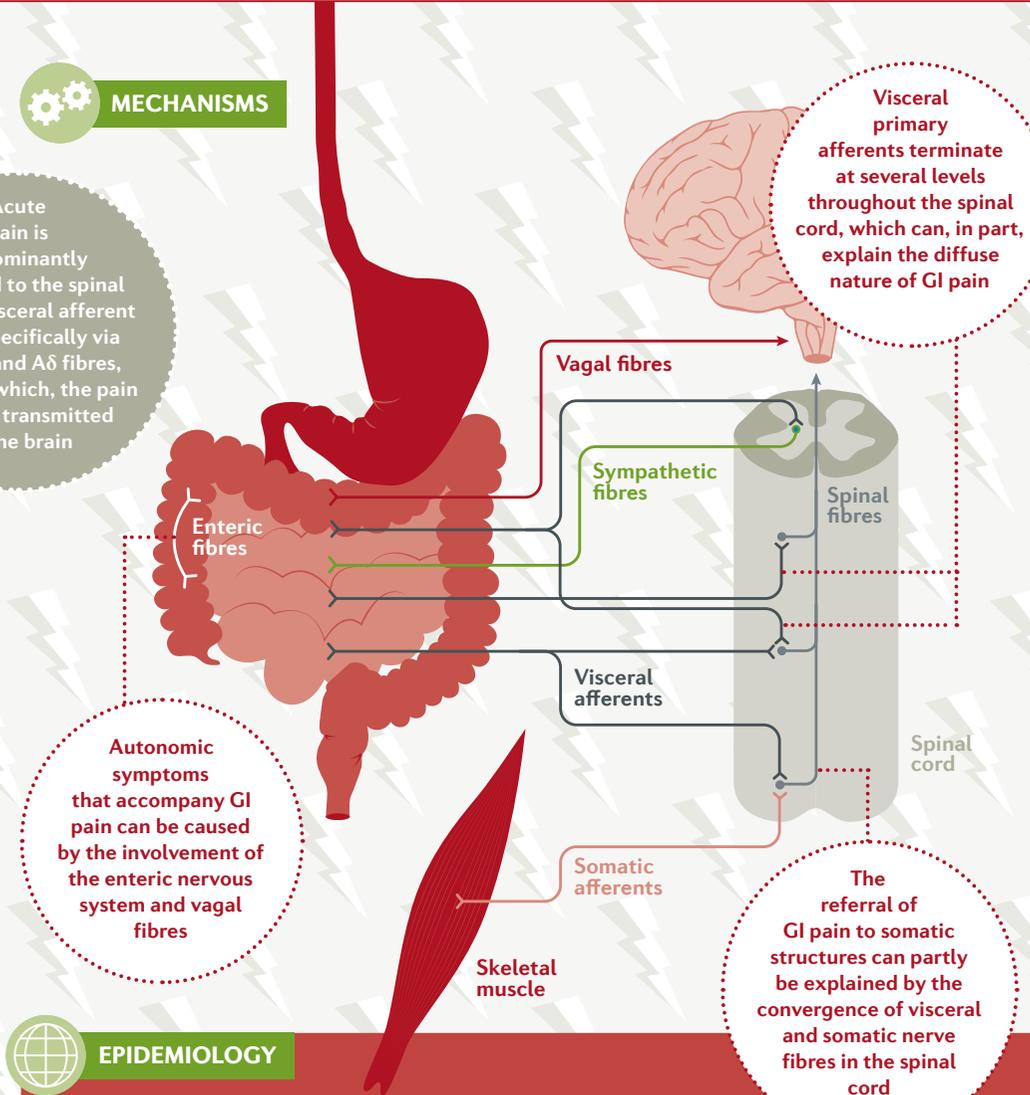
disorders, such as inflammatory bowel disorders (in which 50–70% of patients have abdominal pain as a presenting symptom), chronic pancreatitis (in which most patients have pain during the course of disease) and

malignancies (such as pancreatic cancer, in which abdominal pain was reported as a primary symptom in 44% of patients in one study).

Autonomic symptoms that accompany GI pain can be caused by the involvement of the enteric nervous system and vagal fibres

The referral of GI pain to somatic structures can partly be explained by the convergence of visceral and somatic nerve fibres in the spinal cord

Visceral primary afferents terminate at several levels throughout the spinal cord, which can, in part, explain the diffuse nature of GI pain



## Rx MANAGEMENT

In some cases, treating the underlying cause of GI pain is sufficient for pain reduction. However, pain-specific management can be required for some disorders, such as acute pancreatitis. Although no pharmacological therapies have been approved specifically for GI pain, clinicians often use therapies that are approved for musculoskeletal or neuropathic pain, such as NSAIDs, acetaminophen, gabapentin, tricyclic antidepressants and serotonin–noradrenaline reuptake inhibitors. Of note, NSAIDs should be administered with proton pump inhibitors in patients with GI disorders, owing to their effects on the gut. Opioids can be used for severe GI pain but should be used with extreme caution owing to the high risk of dependence. Non-pharmacological treatments can be used in some patients, such as endoscopy for those with obstruction of the biliary tree or main pancreatic duct, and surgery in some patients with chronic pancreatitis.

## QUALITY OF LIFE

Quality of life worsens with GI pain severity. Chronic GI pain can be associated with several factors that affect quality of life, such as functional impairment, anxiety, depression, disturbed sleep patterns and impaired cognition.

! Several questionnaires can be used to assess quality of life in people with GI pain, including general questionnaires (such as the Short-Form Health Survey 36) and those for specific GI disorders (such as the Pancreatitis Quality of Life Instrument and the Irritable Bowel Syndrome-Quality of Life Measure).