

Chronic, non-visceral abdominal pain

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When patients present with persistent or recurrent abdominal pain it is all too easy to consider a visceral source and overlook other origins for their symptoms. Patients with chronic abdominal pain are often subjected to a variety of procedures in an attempt to find a cause: simple investigations may give way to more complex and invasive ones in the pursuit of ever more obscure diagnoses. Then failure to find a visceral cause for the pain may prompt the physician to apply a functional or psychosomatic label to the patient, with any treatment directed along those lines. An awareness, however, that abdominal pain may have a non-visceral origin^{1,2} can forestall a fruitless search for intra-abdominal pathology. A careful history and examination, and being alert to the possibility of the symptoms arising from outside the abdominal cavity, should permit an accurate diagnosis to be made, appropriate treatment given, and an ever downward spiral of yet more negative investigations avoided.

Abdominal wall

The abdominal wall comprises the parietal peritoneum, fat, aponeurosis, musculature, and skin, and derives its somatic nerve supply from the intercostal nerves T7 to T12. Patients have difficulty describing their pain clearly because the accuracy of cutaneous sensation diminishes considerably through the deeper tissues to the viscera. Referred pain is not yet fully understood, but the autonomic system provides visceral pain afferents through the sympathetic system, which uses the same dorsal horn as the intercostal somatic nerves. Because of previous experience that most sensory impulses come from the skin the brain is thought to interpret the signal as originating superficially. The clinician needs to be aware not only of possible visceral sources of the pain, but also the more specific symptoms and tests that point to an abdominal wall origin (Tables I and II).

CARNETT'S SIGN

In 1926 Carnett described³ a clinical test to aid in the diagnosis of abdominal wall pain. The patient is examined supine and the site of maximum tenderness identified with the examining finger. He is then asked to fold his arms across the chest and sit halfway up. If continued palpation at the same point elicits similar or increased pain the test is said to be positive. Carnett's hypothesis was that if the pain arose from visceral disease the tensed muscles protected the underlying organs so that the tenderness was reduced; while continued pain implied abdominal wall pathology. Sitting up as Carnett describes requires a level of fitness not always attained by our patients and suggested

TABLE I *General features of musculoskeletal abdominal wall pain*

<i>Onset</i> – often insidious but a history of injury – both minor and major – or repetitive trauma or unaccustomed physical activity.
<i>Pain</i> – sharp component, often followed by a dull, persisting ache on the affected side, which may radiate widely both anteriorly and posteriorly in the distribution of a dermatome.
<i>Positional component</i> – the pain may be aggravated or relieved by certain positions, changes in posture, lifting, coughing or sneezing.
<i>Modulating factors</i> – relief by local application of heat, certain positions, gentle pressure to support the area.

modifications to Carnett's original test include tensing of the abdominal musculature by raising just the head and shoulders from the bed, or lifting both heels off the couch. In our experience just enough movement of the head and shoulders to tense the muscles without flexing the trunk gives the optimum opportunity for keeping one's finger on the tender spot and eliciting any change or persistence in abdominal wall tenderness. This test has been found to be sensitive and specific,^{4,5} in one study saving on average \$900 per case on unnecessary investigations.⁴ Injection of local anaesthetic into the tender area⁶ gives relief of pain and is of therapeutic as well as diagnostic value. It affords excellent reassurance to the patient when effective.

MYOFASCIAL TRIGGER POINTS

A common but often overlooked cause of abdominal wall pain is a myofascial trigger point,⁷ previously called fibrositis, myalgia or muscular rheumatism. It is defined as a locus of hyperirritability in a muscle or its connective tissue, which is tender when compressed and if sufficiently hypersensitive gives rise to referred pain and a muscular twitch. The precise histological nature of these trigger zones has never been characterised although there are reports of fibrous tissue reactions and bands, with fatty infiltration, and aggregates of nerve fibres. They may result from mechanical overload or as sequelae of local inflammation. Myofascial trigger points are found in many areas of the body, especially around the scapulae and down the back,⁸ but can occur too in the anterior abdominal wall. An active trigger point is always tender and found as a palpable band of muscle fibres, which seems to prevent full lengthening of the muscle fibres caused by associated spasm. Sharp

TABLE II *Examination for musculoskeletal abdominal wall pain*

Inspection for scars – a common source of such pain
Mild scoliosis from muscle spasm
Tenderness
over site of pain
persists on tensing abdominal wall (Carnett's sign)
over vertebral body
over the lateral spinous processes
Hyperaesthesia on light touch (hover sign)
Pain aggravated by lightly pinching of the skin of the affected area

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pressure may elicit a twitch response of the muscle band, accompanied by transient pain making the patient flinch and even jump.

Myofascial trigger points can be found in any part of the abdominal musculature, especially in the rectus abdominis, from where pain may be referred throughout the abdomen and even round into the back. Treatment is again by injection of local anaesthetic, which usually gives rise to swift improvement, but this is not always sustained and longer lasting treatment may be required with a local injection of phenol, TENS machine, or hot or cold applications over the affected area.

NERVE ENTRAPMENT

Nerve entrapment may occur anywhere along the path of the nerve fibre affected, from its origin at the spinal cord, through its passage out through the tissues, to its termination over the anterior abdominal wall. Several specific nerve entrapment syndromes have been described.

Rectus abdominis nerve entrapment syndrome

Several authors have described a point of tenderness just over the linea semilunaris of the rectus sheath.^{9,10} Tensing the recti (as in Carnett's sign) makes the pain worse, and sometimes in thin patients a dimple may be seen at the site of tenderness. An anatomical explanation was offered by Applegate,¹¹ in which he explained how the neurovascular bundle containing the anterior branches of the intercostal nerves turn rather sharply at the lateral border of the recti to run through fibrous tunnels in the muscle. He suggested that the trigger point is produced by herniation of the bundle with its protective fat pad through gaps in the anterior rectus sheath. Clinically, however, it is often uncertain as to whether this is a true nerve entrapment or a myofascial trigger point; distinguishing between the two is not always possible. Again, an injection of local anaesthetic at the tender point is a useful diagnostic and therapeutic procedure.

Ilioinguinal and iliohypogastric nerve entrapments

The ilioinguinal nerve arises from T12/L1 nerve roots. It runs across the posterior abdominal wall under the peritoneum and then becomes more superficial as it courses round the oblique muscle layers. At a point 2–3 cm medial to the anterior superior iliac spine, the nerve emerges superficially and runs down to supply the skin on the upper medial aspect of the thigh, the root of the penis and anterior scrotum, or the mons pubis and labium majus. The iliohypogastric nerve also arises from T12/L1 and runs a similar course to the ilioinguinal, overlapping its area of innervation, but including the hypogastrium.¹² These are nerves that are particularly likely to be injured by surgery, such as appendicectomy, hernia repair, and a Pfannensteil incision.¹³ Nerve entrapment is associated with a persistent burning sensation in the iliac fossa, inguinal region radiating into the groin, the top of the thigh, and the lower abdomen. It is made worse by movement, including walking and straining;

while curling up to flex the hip often gives some relief. There is hyperaesthesia and sometimes paraesthesiae felt in the distribution of the nerve.¹⁴ Hyperextension of the hip may aggravate the pain. Tender spots should be sought and the area injected for diagnostic and therapeutic purposes. Surgery is sometimes required to remove the nerve and give relief.¹⁵ The hover sign may be helpful. Because of the hyperaesthesia, if the hand hovers over the affected area the patient will tend to tense up and the lightest of touches will make them wince and try to move the hand away.¹⁶ Formal testing for light touch may also be helpful in identifying an area of hyperaesthesia.

POSTOPERATIVE, INCISIONAL PAIN

Any abdominal incision, especially if lateral, can cause local injury to nerves, with the subsequent formation of a sensitive neuroma. When examining the patient it is vital to tense the abdominal wall, not only looking for a tender spot (Carnett's sign) but also for any evidence of weakness of the wound, or incisional hernia, which may be amenable to surgical repair. If no incisional hernia is found and a localised tender area found, injection of local anaesthetic is helpful.¹⁷ As already mentioned the ilioinguinal and hypogastric nerves are especially at risk from scars of appendicectomy, hernia repair, or hysterectomy.

SKELETAL PAIN

Thoracic cause for abdominal pain

The anterior abdominal wall is innervated by the intercostal nerves (T7–12), so the chest and thoracic spine may be a source of abdominal symptoms.

*Painful rib syndrome*¹⁸ (*slipping rib syndrome, rib tip syndrome*) – This disorder is characterised by pain in the lower costal margin⁸ associated with increased mobility of the anterior costal cartilages of the 8th, 9th, 10, and 11th ribs. There are fibrous attachments binding the lower costal cartilages to each other and it is thought that these bindings may be loosened by injury, permitting greater mobility so that one rib can move on another, producing a sharp pain followed by a dull ache in that area, possibly from a brief nerve entrapment of the intercostal nerve as it crosses over that costal cartilage.¹⁹ Some patients can associate the pain with particular movements. There is diffuse tenderness over the lower costal margin but one spot, usually the rib end, is particularly tender.¹⁸ Hooking the fingers under the loose rib and moving it up can reproduce the pain in many patients.²⁰ An explanation, and infiltration with local anaesthetic, are usually helpful although the tenderness and pain often persist.¹⁸

Precordial catch syndrome – This not uncommon complaint particularly affects younger people who experience an abrupt sharp, stabbing, chest pain, causing them to catch their breath.¹⁹ The sharpness of it may make the patient press on the affected part of the chest wall and lean towards the affected side, wincing with pain. The pain lasts seconds and then the subject can gradually

straighten up and breathe normally again: a dull ache may follow for a time. It may affect either side of the chest, but when it occurs low down on the right side it may mimic biliary pain. A careful history is needed – the sudden, sharp pain, catching of breath, and lasting such a short time is characteristic. There is rarely chest wall tenderness, and there is no diagnostic test. The cause is unknown, but reassurance is usually sufficient to relieve the patient's worries.

Costochondritis (anterior chest wall syndrome) – Chest pain from tenderness of the costal cartilages is well recorded, particularly as Tietze's syndrome (characterised by swelling and tenderness of the second and third left costosternal cartilages). Tenderness of the costal cartilages including the rib ends can be found anywhere down the sternum, including the costal margin and the xiphisternum.^{22,23} The patient complains of a dull ache and there is tenderness over the affected cartilages. The cause is unknown and treatment difficult. Reassurance is important. Clinically there is possibly an overlap with the slipping rib syndrome.

Spinal

Both visceral sympathetic and somatic nociceptive afferents converge in the same dorsal horn. Also visceral and somatic noxious stimuli may be conveyed in the same spinothalamic tract. Thus pain from the spinal muscles or vertebral bodies may be interpreted as visceral in origin. Jorgensen and Fossgreen²⁴ compared 39 patients with upper abdominal pain without a demonstrable organic intra-abdominal cause and 28 healthy controls. They found that 72% of their patients also had back pain, compared with 17% of controls. The examinations were blinded with regard to the symptoms, 75% of the patients with back pain had vertebral abnormalities shown at the physical examination, pointing to some organic mechanism affecting the spine. Most of the findings were localised to the lower thoracic and thoracolumbar segments, the same ones that innervate the upper gastrointestinal tract. Nearly half of their patients had symptoms of the irritable bowel syndrome or heartburn, which they felt were significantly related to the back pain. Ashby²⁵ studied 73 patients in one year in his clinical surgical practice whom he considered to have a spinal origin for their abdominal symptoms. They complained of a burning or sore pain, nagging like toothache. Nearly half had noticed aggravation by posture. Examination often showed tenderness near the tip of the vertebral transverse process, which was a particularly valuable sign, interpreted as indicating sensitivity of the adjacent intercostal nerve. Spinal movements were also assessed and were often limited in these patients. Tensing the muscles aggravated the pain in almost all cases. An intercostal nerve block gave relief in two thirds of his cases and Ashby felt that this broke a spiral of pain, which he described as the 'spinal reflex pain syndrome'. This was based on the idea that transient painful trigger stimuli can initiate a prolonged cycle of pain and spasm of the muscle through spinal reflexes, which may be interrupted by a local anaesthetic breaking the

cycle. The importance of individual vertebral tenderness has also been highlighted by other workers.²⁶

HERNIAS

Most abdominal wall hernias are either visible or palpable on clinical examination. The presence of a lump with an expansile cough impulse is sufficient to make the diagnosis. In a few patients, however, particularly the obese, the diagnosis may be difficult. If symptoms suggest a hernia and there is a persisting discomfort in one area, which is tender to the examining finger, additional investigations such as ultrasound²⁷ and herniography²⁸ are probably useful. The technique of herniography entails injecting contrast into the peritoneal cavity and the patient postured to see if the contrast fills a hernial sac. This is useful both for inguinal and unusual hernias such as Spigelian hernia.

HYPERVENTILATION

Hyperventilation is typically associated with panic attacks in which the patient feels faint, lightheaded, sweaty and trembly; the vision may fade and the patient feel remote from his/her surroundings. Numbness or paraesthesiae of the limbs may be present and there is often a feeling of heat or cold with profound tiredness. Chest pain is common with hyperventilation, but abdominal pain features less often and is therefore less well recognised.²⁹ In the chronic type of hyperventilation only a slight increase in respiratory drive over a prolonged period can produce the biochemical changes of hyperventilation,³⁰ so these patients rarely recognise that they are overbreathing. Patients with chronic hyperventilation have been divided into two groups – in the first, symptoms are associated with exercise (when they are often referred to a cardiologist), while in the second symptoms are more persistent and unrelated to exercise. Curiously, hypocapnoea may persist even into sleep, the PCO₂ not returning to normal until well into the night.³¹ Hyperventilation has been thought to cause oesophageal motor disorders³² but has not been found to affect colonic motility.³³ It is useful to ask the patient to hyperventilate deliberately; this may reproduce their symptoms. The syndrome has been described as being responsible for pseudoangina (possible oesophageal dysmotility), flatulence, and abdominal distension.³⁴

METABOLIC

Metabolic causes of chronic non-visceral abdominal pain, such as chronic renal failure, Addison's disease, porphyria, etc, are well recognised and will not be dealt with further here.

PSYCHOGENIC

Psychological factors are inextricably linked to the genesis of pain.³⁵ The pain is often described as severe and persistent, having been experienced for several years and being present without

let up. The pain is inconsistent with the anatomy of the nervous system, and no organic pathological or pathophysiological mechanism can be found responsible. Symptoms are often grossly out of proportion to the physical findings, and the patient usually looks well. It has been suggested that anxiety and depression may perpetuate the pain by enhancing muscle spasm, facilitating spinal reflexes, and then leading to a preoccupation with the site of the pain. A wide variety of mental disorders affect the perception of pain, and range from a depressive illness (which often responds to treatment), to hypochondriasis and somatoform disorder, which are much more difficult to manage.

In conclusion, the clinician always needs to be aware of non-visceral causes for a patient's symptoms. A careful history is crucial, and examination should always include an assessment of tender spots, with Carnett's sign, hyperaesthesia, and tenderness over the vertebral bodies. Several authors have found particular value in Carnett's sign,^{1,2} which has been shown to be both specific and sensitive.^{4,6} It not only relieves the pain and reassures the patient, but also saves a large number of unnecessary and often unpleasant investigations. Carnett's sign is not infallible; a positive test must always be put into the whole clinical picture. None the less, following up a positive Carnett's sign with a successful injection of local anaesthetic must be one of the most cost effective procedures in gastroenterology.⁴

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