Visceral pain

Anu Kansal

John Hughes

Abstract

Pain is one of the commonest symptoms the patient presents with. Visceral organs were thought to be insensitive to pain in the past but now we know this not to be true. It is more common than somatic pain and originates from the internal organs in the thorax, abdomen or pelvis. These organs are innervated by parasympathetic (vagus and sacral parasympathetic fibres) and sympathetic (thoracolumbar sympathetic chain – T1–L2) nervous systems. The afferent and efferent fibres to the organs accompany the sympathetic nervous system. The sensory system to the gut is specialised and divided into enteric and extrinsic nervous system.

The physiology of visceral pain is poorly understood compared to somatic pain but it is well established that peripheral and central sensitisation along with dysregulation of the descending pathway plays a significant role. Pain originating from visceral organs is usually diffuse, dull aching, poorly localised and can be associated with phenomenon such as, referred somatic pain, referred hyperalgesia, visceral hyperalgesia and viscero-visceral hyperalgesia.

Treatment of visceral pain involves identifying and treating the cause, if identified and the management of pain. Patient education and information plays an important part in management along with pharmacological and non-pharmacological treatments.

Keywords Neuro-anatomy; visceral hyperalgesia; visceral pain; viscero-somatic convergence; viscero-visceral hyperalgesia

Royal College of Anaesthetists CPD Matrix: 1A01, 1A02, 2EO3, 3E00

Introduction

Vertebrates have been considered 'dual entities', composed of a 'somatic' and a 'visceral' component responding to different environments: an external environment in which the organism is situated, and an internal environment in which the tissue elements live.[1]

Pain is one of the most common presentations to seek medical help. Visceral pain is pain that results from the activation of nociceptors of the thoracic, pelvic, or abdominal visceral organs. In the past visceral organs were considered insensitive to pain but now it is clear that the social burden of visceral pain is much greater than somatic pain. Pain in the viscera usually comes from distension, inflammation or ischaemia.

Anu Kansal *MBBS MD Anaesthesia FRCA FFPMRCA is a Pain fellow in the Department of Pain Medicine at James Cook University Hospital, Middlesborough, UK. Conflict of interest: none declared.*

John Hughes *MBBS FRCA FFPMRCA is a Consultant in Pain Medicine at James Cook University Hospital, Middlesborough, UK. Conflict of interest: none declared.*

Learning objectives

After reading this article you should be able to:

- explain the neuroanatomy of visceral pain
- describe the pathophysiology of visceral pain
- identify the clinical presentation of visceral pain
- formulate a management plan

Neuro-anatomy of visceral pain

Visceral efferents

Most of the thoracic and abdominal visceral organs, except the pancreas, are innervated by both the parasympathetic (craniosacral) and sympathetic (thoracolumbar) nervous system (Figure 1). The vagus is the main parasympathetic nerve for thoracic and upper abdominal viscera while the sympathetic fibres come from thoraco-lumber sympathetic trunk (T1 to L2 or may extend to L3). The lower abdomen and pelvis are mainly supplied by thoraco-lumber sympathetic fibres and sacral parasympathetic fibres.

The sympathetic preganglionic fibres originate from the respective ventral root and pass to the sympathetic trunk via the grey rami communicans. Most sympathetic fibres synapse in the sympathetic trunk and post-ganglionic myelinated fibres pass via the splanchnic nerves to the ganglions (for example coeliac ganglia, superior mesenteric ganglia and inferior mesenteric ganglia) and onward to the end organs. The fibres, that don't synapse at the sympathetic chain, synapse in the ganglion near the organs. The parasympathetic fibres travel via either the vagus nerve or the pelvic parasympathetic fibres and synapse in the ganglion near the organs. Some parasympathetic fibres pass with the sympathetic fibres.

Visceral afferents

The general visceral afferent (GVA) fibres conduct sensory impulses from the viscera, glands, and blood vessels to the central nervous system. They are considered to be part of the visceral nervous system, not the autonomic nervous system. In the abdomen the afferent fibres accompany the sympathetic efferent fibres.

The primary sensory afferents are pseudo-unipolar cells having central and peripheral axonal processes. The peripheral process innervating the visceral organ may have specialised end-organ-like Pacinian corpuscles or free nerve endings. The afferent fibres (from abdomen and most of the pelvis) usually accompany sympathetic efferent fibres and pass from the receptors in the end organ to the respective ganglion. From here, they go via the splanchnic nerves to the sympathetic trunk without synapsing in the ganglia or sympathetic trunk, pass into the ventral ramus via the white rami communicans and finally synapse with the mixed spinal nerves (Lamina V). The path of the afferent fibres diverges from the efferent sympathetic fibres as efferents come via the ventral root of the spinal column. Afferents follows the dorsal root into the dorsal root ganglion, where the cell body of the visceral afferents is located. They converge with the somatic afferent nerve fibres in the dorsal horn of spinal cord. This convergence of the somatic and visceral fibres at the level of the dorsal root explains the "referred pain" seen with visceral pathology. There is also convergence of the

Please cite this article in press as: Kansal A, Hughes J, Visceral pain, Anaesthesia and intensive care medicine (2016), http://dx.doi.org/10.1016/j.mpaic.2016.08.013

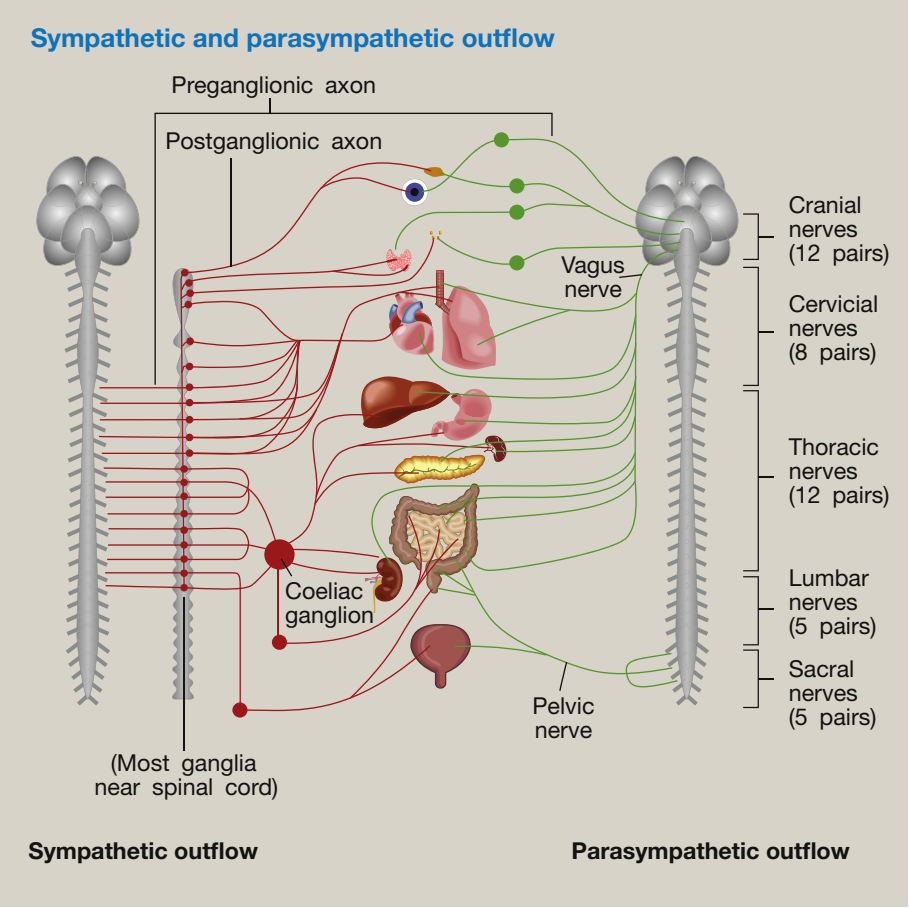

Figure 1

afferent fibres from different organs allowing for viscero-visceral referral patterns.

Some of the afferents from the lower pelvis follow the parasympathetic efferent system.

Enteric nervous system

The sensory system of the gastrointestinal tract consists of intrinsic (enteric) sensory system and extrinsic (valgus, spinal, and pelvic) afferents. The intrinsic system functions independently of the CNS. Neurons are directly exposed to the mechanical forces and the chemical environment which is unlike somatic afferents neurons. Entero-chromaffin and entero-endocrine cells within the mucosa release serotonin, cholecystokinin, orexin, and leptin which modulate and regulate motor activity. The submucosal enteric plexus and myenteric plexus have a high degree of synaptic interactions which can be either inhibitory or stimulatory for the purpose of regulating gastrointestinal motility and peristalsis. Both plexuses received input from parasympathetic and sympathetic efferents. There is a crosstalk between intrinsic and extrinsic systems.

Neuro-physiology of visceral pain

The physiology of visceral pain remains less well understood compared with that of somatic pain.[2] This may be in part due to the diverse nature of the visceral organs and their function. In spite of this, it is well established that visceral pain can occur due to:

- Sensitisation of primary sensory afferents innervating the viscera
- Hyper-excitability of spinal ascending neurons (central sensitisation) receiving synaptic input from the viscera
- Dysregulation of descending pathways that modulate spinal nociceptive transmission.

As with somatic pain, peripheral and central mechanisms both play a significant role in the pathophysiology of visceral pain. Inflammation or excessive stimulation of the visceral organ (e.g. over distension) will cause the lowering of the threshold of the "High threshold" receptors and stimulation of the "Silent receptors" which are found in abundance in all visceral organs. This results in peripheral sensitisation and an increased barrage of impulses into the central nervous system.[2]

Increased input to the spinal cord leads to neuro-plastic changes in the central nervous system such as increased sensitivity and excitability leading to central sensitisation. *N*-methyl-D-aspartate (NMDA) receptor plays a significant role in mediating increase in central excitability.

The peripheral and central sensitisation plays a significant role in visceral and viscero-visceral hyperalgesia. This involves sensitisation of the central neurons receiving convergent input from multiple visceral organs.[3]

Please cite this article in press as: Kansal A, Hughes J, Visceral pain, Anaesthesia and intensive care medicine (2016), http://dx.doi.org/10.1016/j.mpaic.2016.08.013