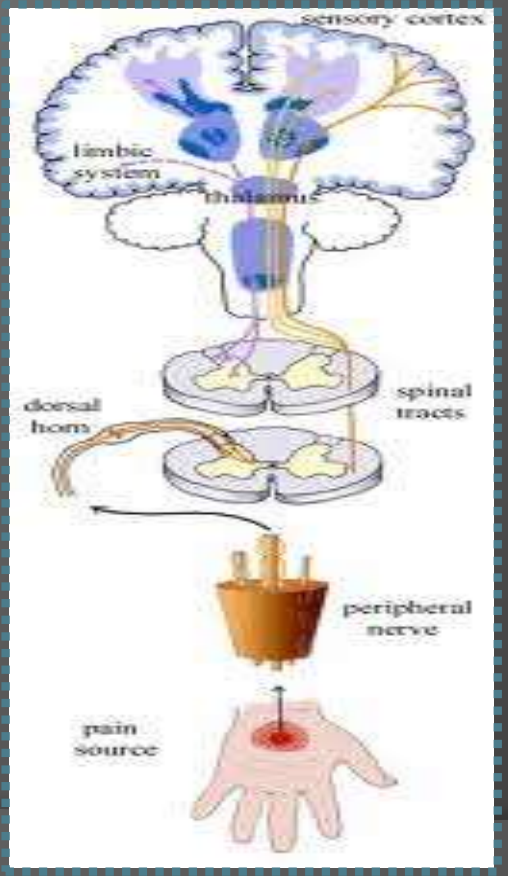
Pain Pathway & GATE Control Theory Of Pain



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UPUMS

What is Pain?

- Pain is not just a sensory modality but is an experience. The international association for the study of pain defines pain as
- “an unpleasant sensory and emotional experience associated with or resembling that association with actual or potential tissue damage, or described in terms of such damage.”
- This definition recognizes the interplay between the objective, physiological sensory aspects of pain and its subjective, emotional and psychological components.

More Definitions

- Hyperesthesia :

  An increase in sensitivity such that what would normally cause light pain causes more intense pain
- Allodynia : A painful response to a stimulus that doesnot normally cause pain
- Analgesia : absence of

  pain perception
- Anesthesia : absence of all sensation
- Anesthesia dolorasa : pain in an area that lacks sensation

- Hypalgesia (hypoalgesia) : diminished response to noxious stimulation (eg. Pinprick)
- Hyperalgesia : increased response to noxious stimulation
- Hypoesthesia : reduced cutaneous sensation (eg. Light touch, pressure, or temperature)
- Hyperesthesia : increased response to mild stimulation
- Neuralgia : pain in the distribution of a nerve or a group of nerve
- Paresthesia : abnormal sensation perceived without an apperant stimulous
- Radiculopathy : functional abnormality of one or more nerve roots

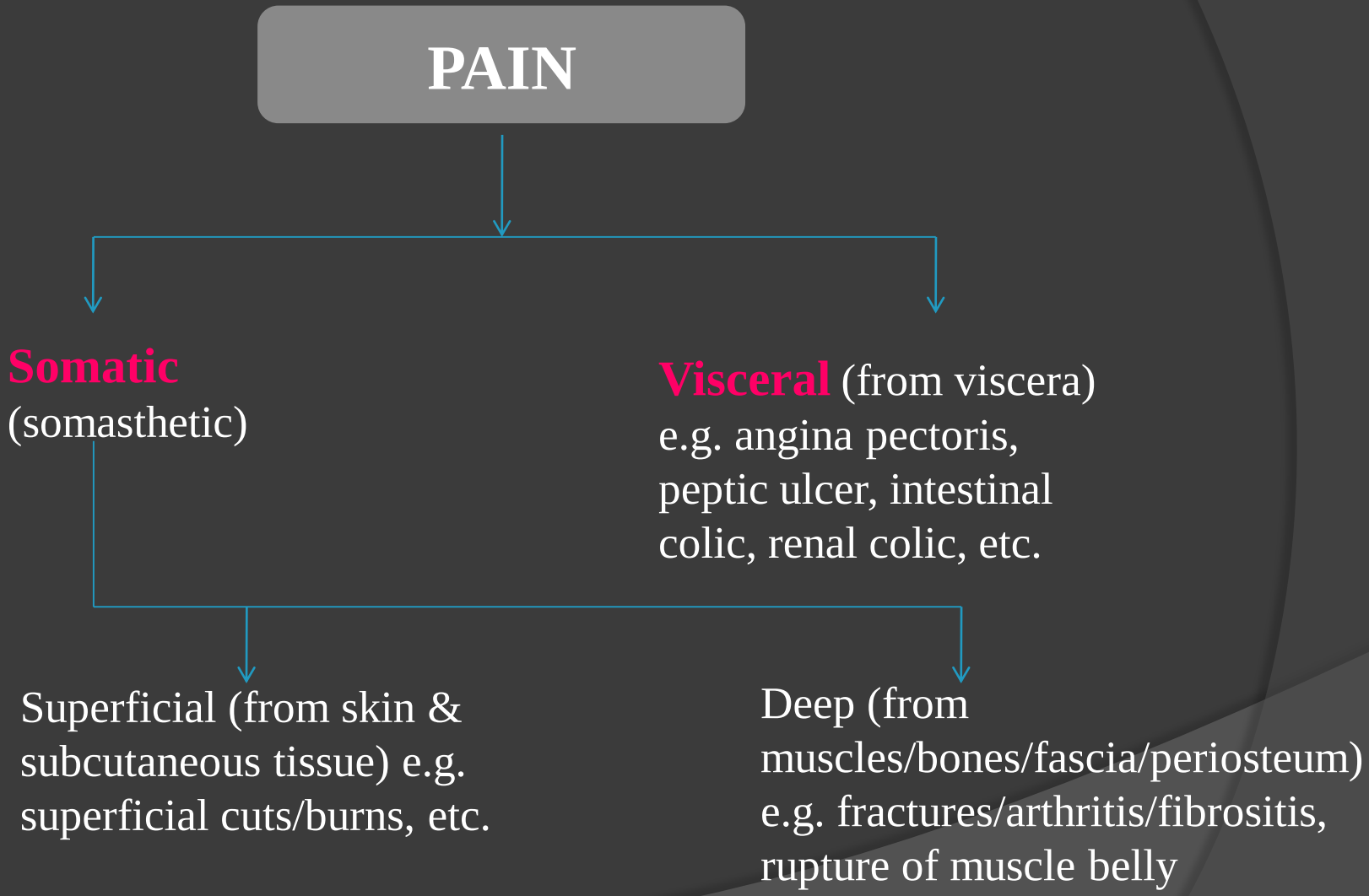
Physiological Effects of Pain

- Increased catabolic demands: poor wound healing, weakness, muscle breakdown
- Decreased limb movement: increased risk of DVT/PE
- Respiratory : shallow breathing, tachypnea, cough suppression
- Increased sodium and water retention (renal)
- Decreased gastrointestinal mobility
- Tachycardia and elevated blood pressure

Consequences of Untreated Pain

- Poor appetite and weight loss
- Withdrawal from social activities
- Physical and verbal aggression, wandering, acting-out behaviour, resists care
- Difficulty in walking or transferring; may become bed bound
- Skin ulcers
- Incontinence
- Impaired immune function

CLASSIFICATION OF PAIN



Major Categories of Pain

1. Nociceptive pain (stimuli from somatic and visceral structures)

2. Neuropathic pain (stimuli abnormally processed by the nervous system)



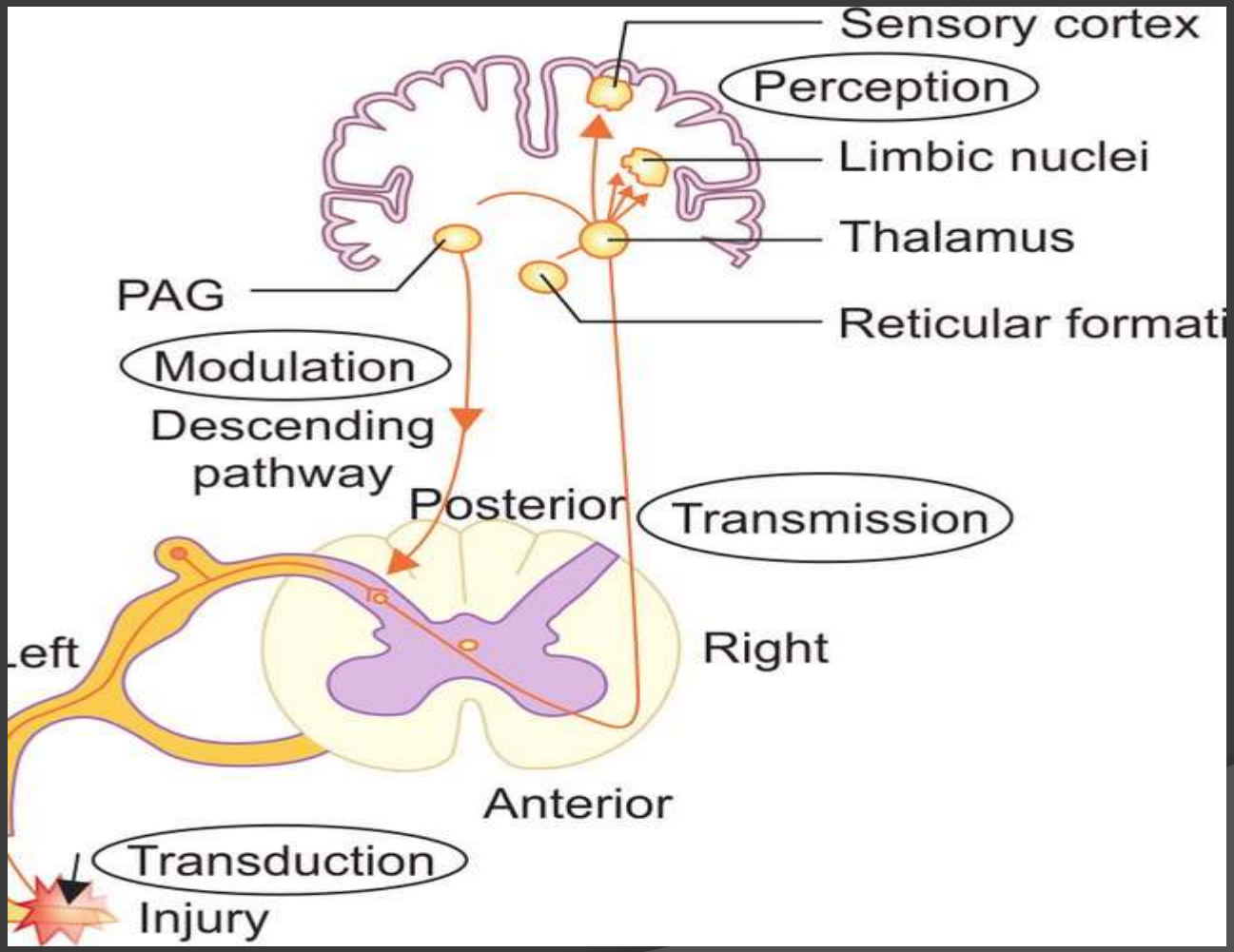
Nociception

- The term “ nociception,” which is derived from noci (Latin for harm or injury), is used to describe the neural response only to traumatic or noxious stimuli.
- All nociception produces pain, but not all pain results from nociception. Many patients experience pain in the absence of noxious stimuli.
- It is therefore clinically useful to divide pain into one of the two categories. 1.acute 2.chronic

- Acute pain: Acute pain can be defined as pain that is caused by noxious stimulation due to injury, a disease process or the abnormal function of muscle or viscera. It is usually nociceptive and subsides once the healing process is accomplished.

- Chronic pain: Chronic pain is defined as pain that persists beyond the usual course of an acute disease or after a reasonable time for healing to occur this period can vary from 1 to 6 months.
- Chronic pain may be nociceptive, neuropathic or mixed. A distinguishing feature is that psychological mechanisms or environmental factors frequently play a major role.

PAIN PATHWAYS



Pain Pathway

- Transduction
- Transmission
- Perception
- Modulation

Tracking the Path of Pain

- Peripheral receptors
- Neural pathways
- Spinal Cord mechanisms & long tracts
- Brainstem, thalamus, cortex & other areas.

Pain pathway is conducted along three-neurons pathway that transmit noxious stimuli from the periphery to cerebral cortex.

SENSORY NEURONS

- First Order
- Second Order
- Third Order



First Order Neuron

- Each sensory receptor is attached to a first order primary afferent neuron that carries the impulses to the CNS.
- The axons of these first-order neurons are found to have varying thickness. It has long been known that a relationship exists between the diameter of nerve fibers and their conduction velocities. The larger fibers conduct impulses more rapidly than smaller fibers.

Second Order Neuron

- The primary afferent neuron carries impulse into the CNS and synapses with the second-order neuron.
- This second-order neuron is sometimes called a transmission neuron since it transfers the impulse on to the higher centers.
- The synapse of the primary afferent and the second-order neuron occurs in the dorsal horn of the spinal cord.

Third Order Neuron

- Cell bodies of third order neurons of the nociception-relaying pathway are housed in: the ventral posterior lateral, the ventral posterior inferior, and the intralaminar thalamic nuclei.
- Third order neuron fibers from the thalamus relay thermal sensory information to the somesthetic cortex.

Peripheral receptor

- Nociceptors are the specialised sensory receptors responsible for the detection of noxious stimuli, transforming the stimuli into electrical signals, which are then conducted to the CNS.
- They are the free nerve endings of primary afferent Aδ and C fibres. Distributed throughout the body, they can be stimulated by mechanical, thermal or chemical stimuli.
- Inflammatory mediators (eg bradykinin, serotonin, prostaglandins, cytokines, and H+) are released from damaged tissue and can stimulate nociceptors directly.

Primary afferent fibres

In addition to the Aδ and C fibres that carry noxious sensory information, there are primary afferent Aβ fibres that carry non-noxious stimuli.

- Aβ fibres are highly myelinated and of large diameter, therefore allowing rapid signal conduction. They have a low activation threshold and usually respond to light touch and transmit nonnoxious stimuli.

- Aδ fibres are lightly myelinated and smaller diameter, and hence conduct more slowly than Aβ fibres. They respond to mechanical and thermal stimuli. They carry rapid, sharp pain and are responsible for the initial reflex response to acute pain.

- C fibres are unmyelinated and are also the smallest type of primary afferent fibre. Hence they demonstrate the slowest conduction. C fibres are responding to chemical, mechanical and thermal stimuli. C fibre activation leads to slow, burning pain.

Summary of receptor types

	Aß - fibres	Aδ - fibres	C- fibres
Threshold	Low	Medium	High
Axon diameter	6-14µm	1-6µm	0.2-1µm
Myelination	Yes	Thinly	No
Velocity	36-90	5-36	0.2-1
Receptor types	Mechanoreceptor	Mechano/Nociceptor	Nociceptor
Receptive field	Small	Small	Large
Quality	Touch	Sharp/first pain	Dull/second pain

Dorsal horn of the spinal cord

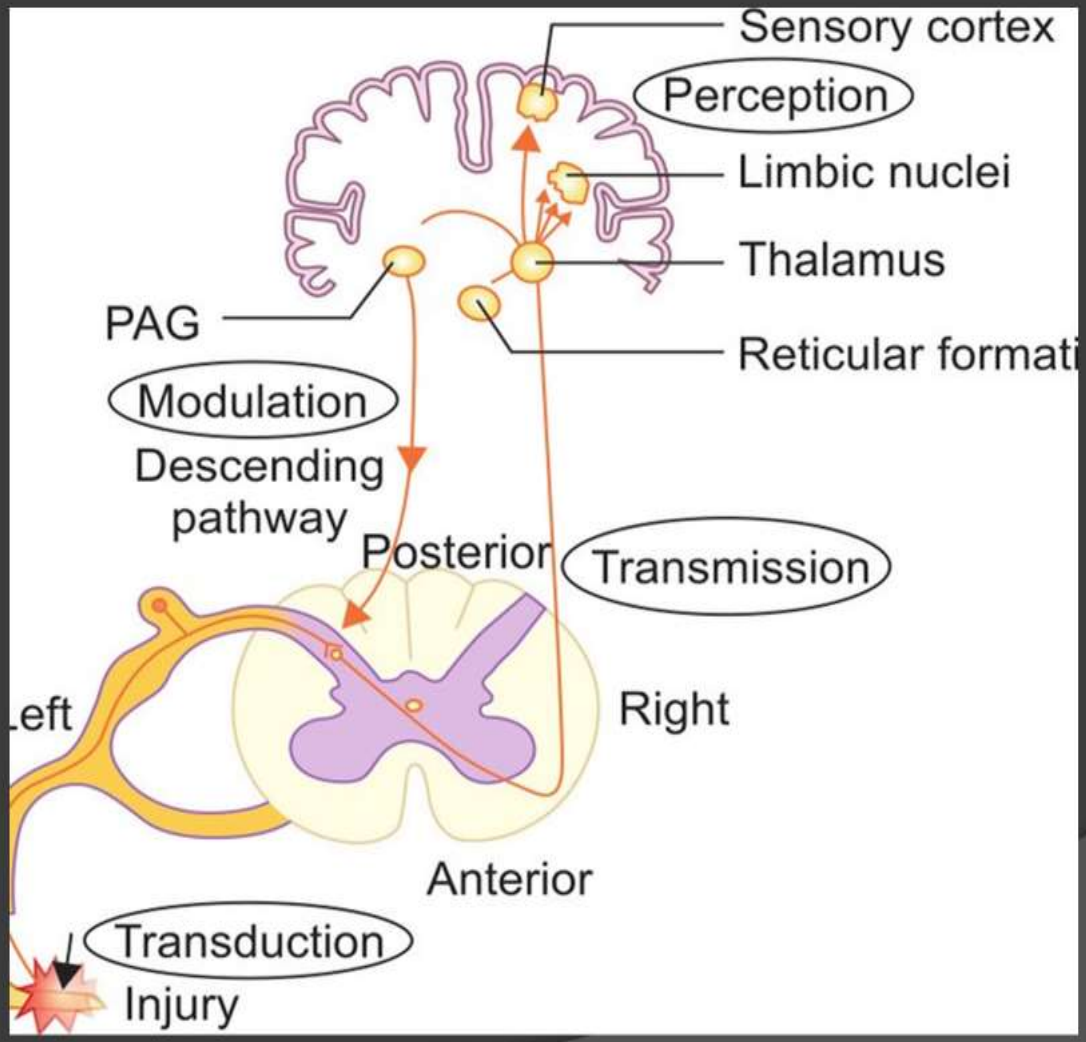
- Aδ and C fibres synapse with secondary afferent neurones in the dorsal horn of the spinal cord. The dorsal horn can be divided histologically into ten layers called laminae. Aδ and C fibres transmit information to nociceptive specific neurones in lamina I and II, in addition to projections to other laminae. Primary afferent terminals release a number of excitatory neurotransmitters including glutamate and substance P.
- Complex interactions occur in the dorsal horn between afferent neurones, interneurones and descending modulatory pathways.These interactions determine activity of the secondary afferent neurones. Glycine and gamma-aminobutyric acid (GABA) are important neurotransmitters acting at inhibitory interneurones.

Ascending tracts in the spinal cord

There are two main pathways that carry nociceptive signals to higher centres in the brain.

- The spinothalamic tract: secondary afferent neurones decussate within a few segments of the level of entry into the spinal cord and ascend in the contralateral spinothalamic tract to nuclei within the thalamus. Third order neurones then ascend to terminate in the somatosensory cortex. There are also projections to the periaqueductal grey matter (PAG). The spinothalamic tract transmits signals that are important for pain localisation.
- The spinoreticular tract: fibres also decussate and ascend the contralateral cord to reach the brainstem reticular formation, before projecting to the thalamus and hypothalamus. There are many further projections to the cortex. This pathway is involved in the emotional aspects of pain.

PAIN PATHWAYS



Gate Control Theory

- Implies a non-painful stimulus can block the transmission of a noxious stimulus.
- Is based on the premise that the gate, located in the dorsal horn of the spinal cord, modulates the afferent nerve impulses.

Gate opened or closed by 3 factors

1. Activity in the pain fibres - opens the gate
2. Activity in other sensory nerves - closes the gate
3. Messages from the brain - concentrating on the pain or trying not to think about it

Conditions that open or close the gate

	Conditions that open the gate	Conditions that close the gate
Physical conditions	Extent of the injury	Medication
	Inappropriate activity level	Counterstimulation, eg massage
Emotional Conditions	Anxiety or worry	Positive emotions
	Tension	Relaxation
	Depression	Rest
Mental conditions	Focusing on the pain	Intense concentration or distraction
	Boredom	Involvement and interest in life activities

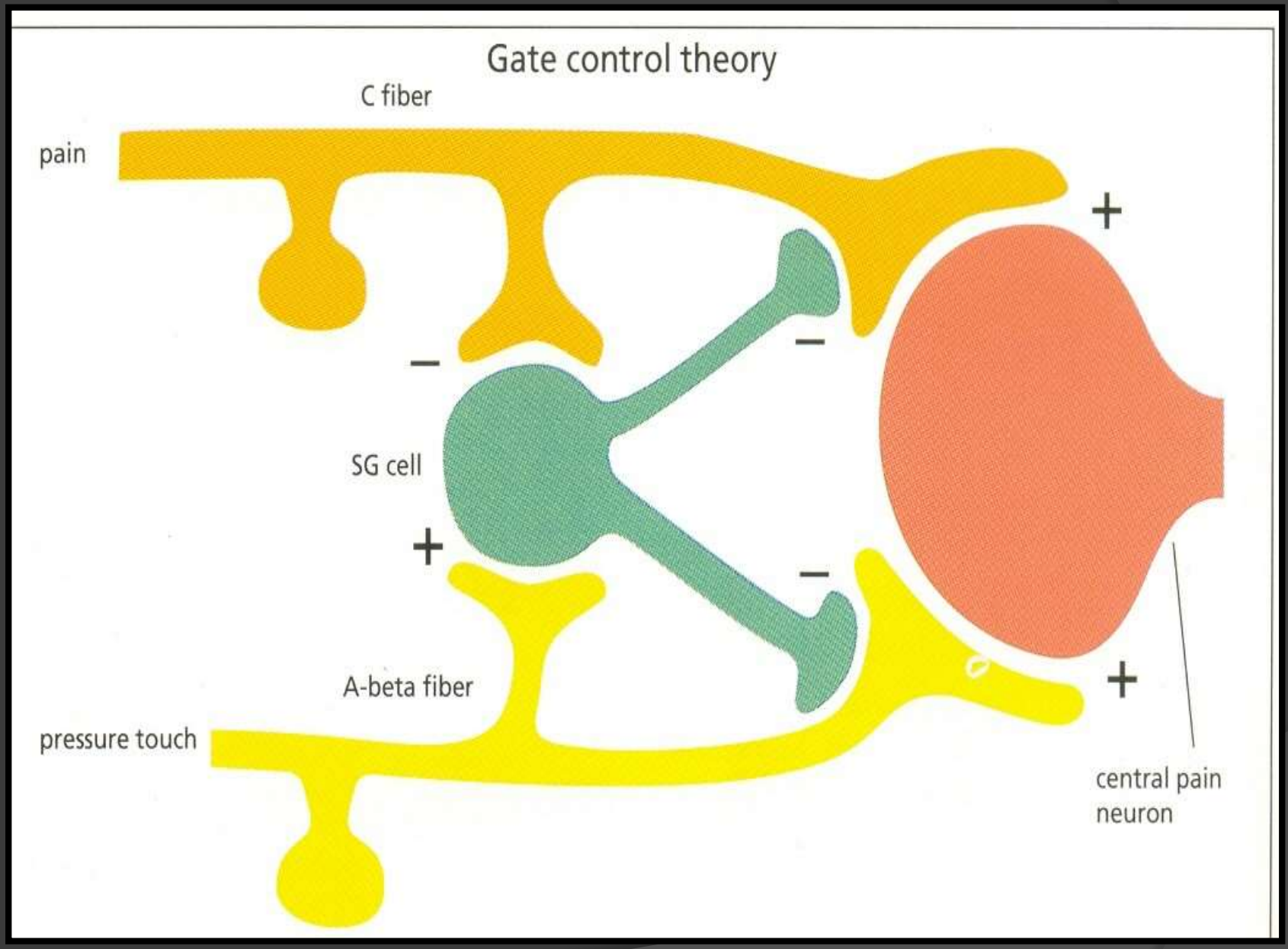
Three variables control this gate

1. A-Delta fibres (sharp pain)
2. C fibres (dull pain)
3. A-Beta fibres that carry messages of light touch

Gate Control Theory

- The gate control theory of pain, put forward by Ronald Melzack and Patrick David Wall in 1962, is the idea that the perception of physical pain is not a direct result of activation of nociceptors, but instead is modulated by interaction between different neurons, both pain-transmitting and non-pain-transmitting. The theory asserts that activation of nerves that do not transmit pain signals can interfere with signals from pain fibers and inhibit an individual's perception of pain.
- This theory of pain takes into account the relative in put of neural impulses along large and small fibers, the small nerve fibers reach the dorsal horn of spinal cord and relay impulses to further cells which transmit them to higher levels.
- The large nerve fibers have collateral branches, which carry impulses to substantia gelatinosa where they stimulate secondary neurons.

- The substantia gelatinosa cells terminate on the smaller nerve fibers just as the latter are about to synapse, thus reducing activity, the result is, ongoing activity is reduced or stopped –gate is closed.
- The theory also proposes that large diameter fiber input has ability to modulate synaptic transmission of small diameter fibers within the dorsal horn.
- Large diameter fibers transmit signals that are initiated by pressure, vibration and temperature; small diameter fibers transmit painful sensations.
- Activation of large fiber system inhibits small fiber synaptic transmission, which closes the gate to central progression of impulse carried by small fibers.


Gate control theory
C fiber
pain
+
–
–
SG cell
+
–
A-beta fiber
pressure touch
+
central pain
neuron

Gate control Theory

- Special neurons located in the grey matter of the spinal cord make up the gate. This gate has the ability to block the signals from the a-delta and c-delta fibres preventing them from reaching the brain.

Gate Control Theory

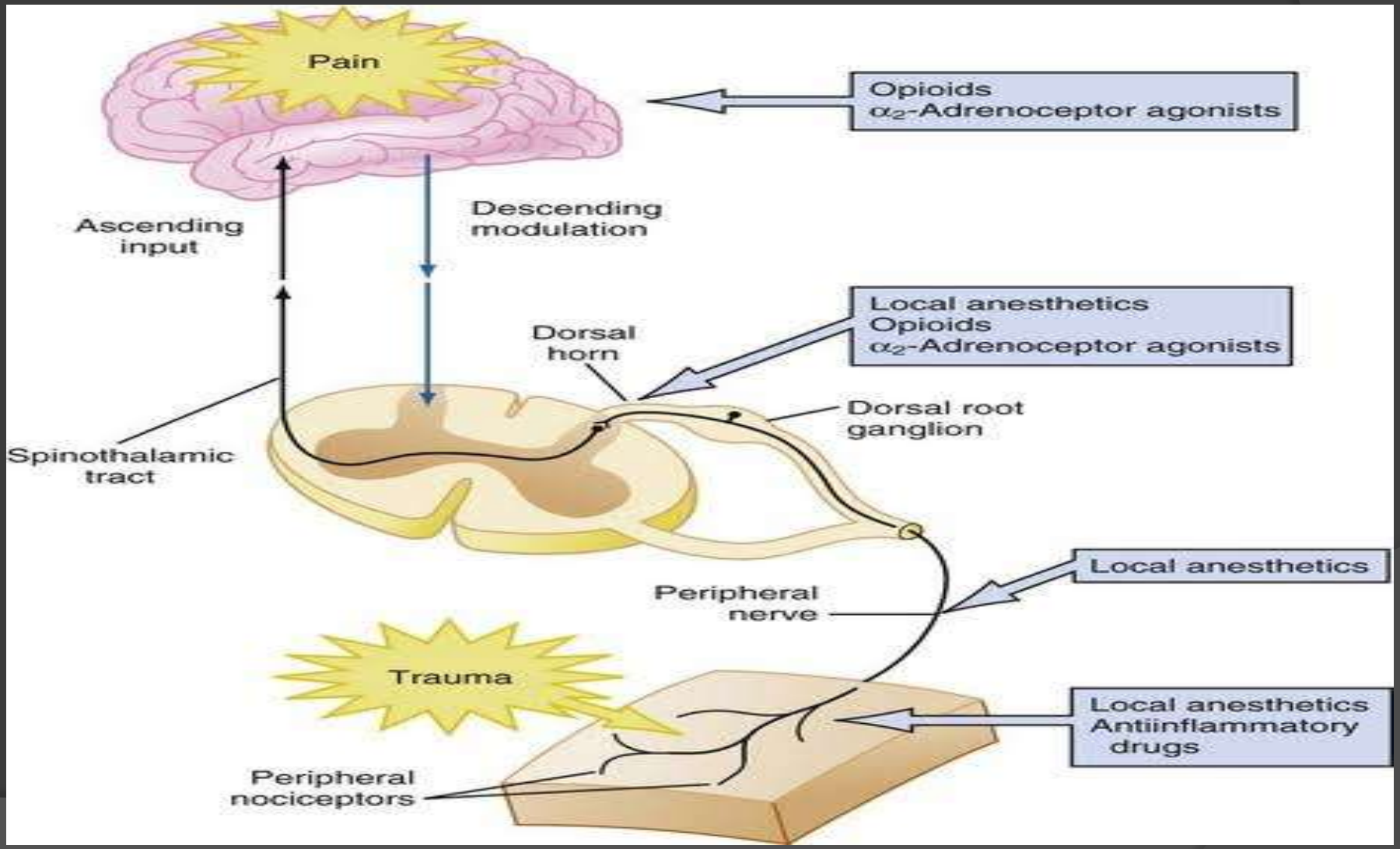
- The special neurons in the spinal cord are inhibitory ie they keep the gate closed. These special neurons make a pain blocking agent called enkephalin. This is an opiate substance similar to heroin which can block Substance P the neurotransmitter from the C fibres and the A-delta fibres and this keeps the gate closed.

Gate control theory

- Gate control theory thus explains how stimulus that activates only nonnociceptive nerves can inhibit pain. The pain seems to be lessened when the area is rubbed because activation of nonnociceptive fibers inhibits the firing of nociceptive ones in the laminae.

- In transcutaneous electrical stimulation (TENS), nonnociceptive fibers are selectively stimulated with electrodes in order to produce this effect and thereby lessen pain.

Durgs site of action



WHO Analgesic ladder

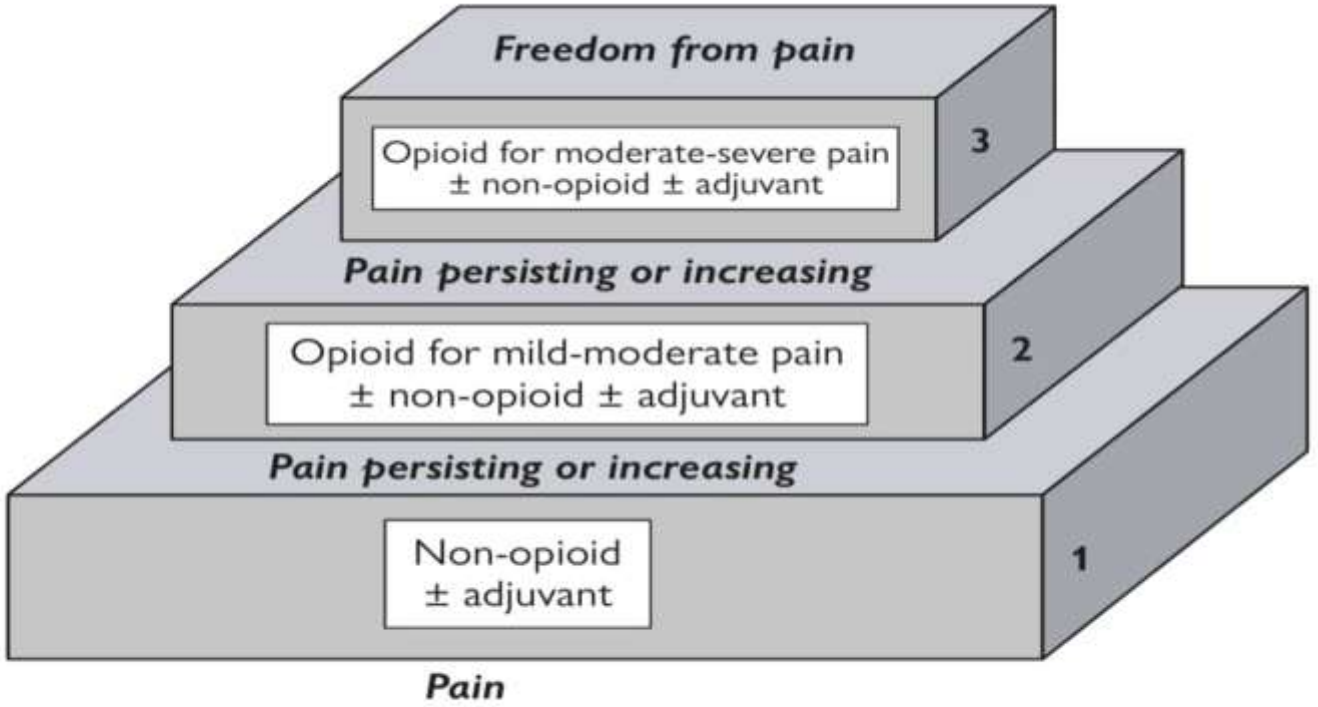


Fig. 2.1 The WHO analgesic ladder.

Level 1:• NSAIDs (aspirin, ibuprofen) •Paracetamol.

Level 2: weak opiates for mild to moderate pain (codeine, tramadol).

Level 3: opiates for moderate to severe pain (morphine, oxycodone, fentanyl and methadone).

The WHO coined 5 phrases to emphasize the correct use of analgesics:

• ‘By mouth’: oral is the preferred route of administration.

• ‘By the clock’: regular administration of analgesics as determined by the pharmacokinetics rather than ‘on demand’.

• ‘By the ladder’: such that the drugs employed should be escalated in line with the pain reported such that for each patient the pain should be treated with level 1 drugs before escalating to add drugs from level 2 or 3.

- ‘For the individual’: the dose of drug, particularly opiates, should be tailored to the individual.

• ‘Attention to detail’: the effectiveness of the analgesic regimen depends on accurate prescription, administration, and monitoring of analgesic drugs in relation to both effect and side effects.


Thanks!