29 Pain modulation

CNS

Sources:

Females slides

OBJECTIVES

- **Describe the control of pain perception including:**
- Spinal modulation (Gate theory of pain control).
- Supra spinal modulation (Special pain control analgesic system).
- Pain modulation by opioid neurotransmitters.
- **Sites & mechanism of pain relief**

NEW TERMS :

- SGR : substantia gelatinosa of rolandi
- PAG : periaqueductal grey matter
- NRM : Nucleus Raphe Magnus
- TENS : Trans Cutaneous Electrical Nerve Stimulation
- PIC : pain inhibitory complex

WHAT IS PAIN MODULATION AND BY WHAT IS IT INFLUENCED ? :

Pain modulation means pain perception variability which influenced by :

- ✓Endogenous mechanism
- ✓Exogenous mechanism

PAIN MODULATION CONTROLLED BY :

1.Spinal modulation of pain input :

Gate theory

2.Supraspinal modulation **(Special pain control analgesic system) :**

- ❖**Role of periaqueductal grey (PAG) matter**
- ❖**Role of Nucleus Raphe Magnus (NRM)**

3.Pain modulation by opioid neurotransmettir as :

endorphin, enkaphalin, dynorphin

1.GATE THEORY OF PAIN CONTROL :

Mechanism

Special neurons in the dorsal horn of spinal cord (SGR) form the gate through which pain impulses must pass to reach brain , this gate has the ability to block the signals from the A-delta and C fibers preventing them from reaching the brain.

Roles of opining & closing

- Activity in the pain fibers - opens the gate. → Impulses coming long type C fibers cause releasing of substance P from those fibers and which lead to opining of the gate
- Activity in other sensory nerves - closes the gate. → While impulses coming along Aβ fibers tend to keep the gate closed by process of presynaptic inhibition of C fibers and postsynaptic inhibition of secondary neurons in dorsal horn.
- Messages from the brain. → Specialised nerve impulses arise in the brain itself and travel down the spinal cord to influence the gate,his is called the central control trigger and it can send both inhibitory and excitatory messages to the gate sensitising it to either C or A-β fibres.

Controlled By

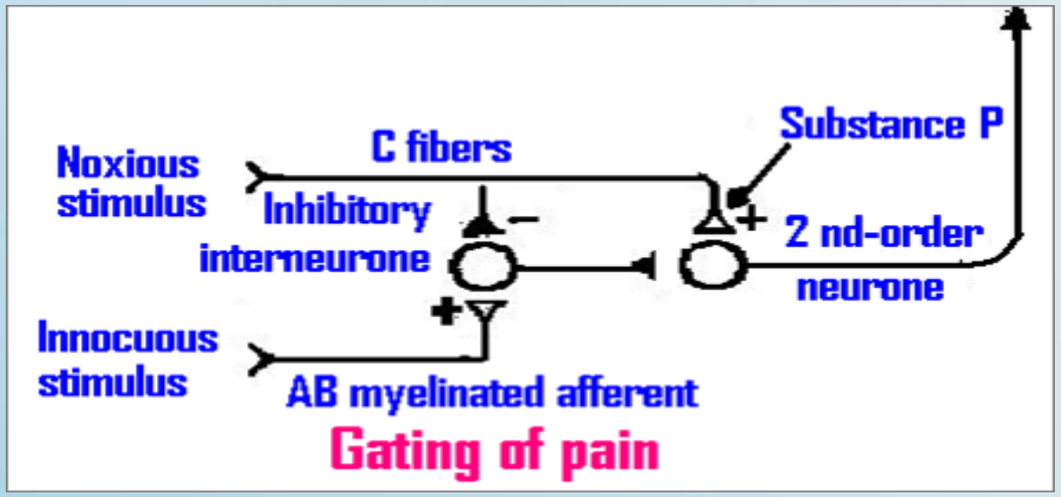
- ❖A-Delta fibres (fast pain).
- ❖C- fibres (slow pain).
- ❖A-Beta fibres (light touch) – fibers of the dorsal column system.

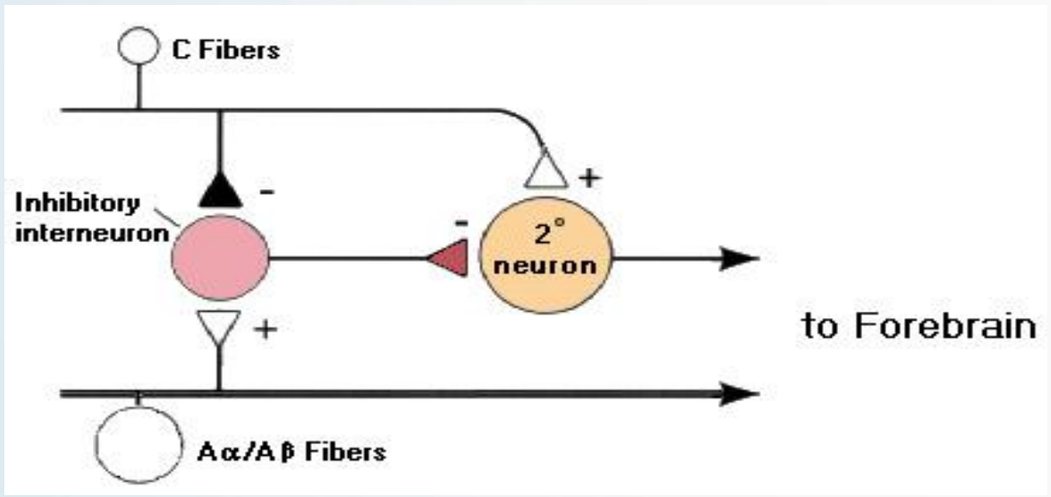
The inhibitory neurons make a pain blocking agent called enkephalin , Encephalin is an opiate substance similar to heroin which can block Substance P, the neurotransmitter from the C fibres and this keeps the gate closed.

1.GATE THEORY OF PAIN CONTROL : CONT.

The gate will open if → the impulses in C & A-delta fibers stronger than the A-beta fibers .
The gate will close if → the impulses in A-beta fibers stronger than C & A-delta fibers .

skin rubbing, shaking the painful part, acupuncture & trans Cutaneous electrical Nerve stimulation (TENS) → will stimulate mechanoreceptors that activate neurons of dorsal column, the collaterals relieve pain → and that what gate theory explain it





2.SUPRASPINA MODULATION

(SPECIAL PAIN CONTROL ANALGESIC SYSTEM) :

This is a specific system that blocks pain transmission in CNS. Its major constituents are:				
Site :	**hypothalamus :**	**Mid brain :**	**Upper medulla :**	**DH of Spinal cord :**
Components :	**Periventricular N neat to 3rd ventricle**	**Periaqueductal grey area (PAG)**	**Nucleus Raphe Magnus (NRM)**	***Pain inhibitory complex (PIC)**

MECHANISM :

Periaqueductal grey area receives neuronal inputs from thalamus, hypothalamus, cerebral cortex.

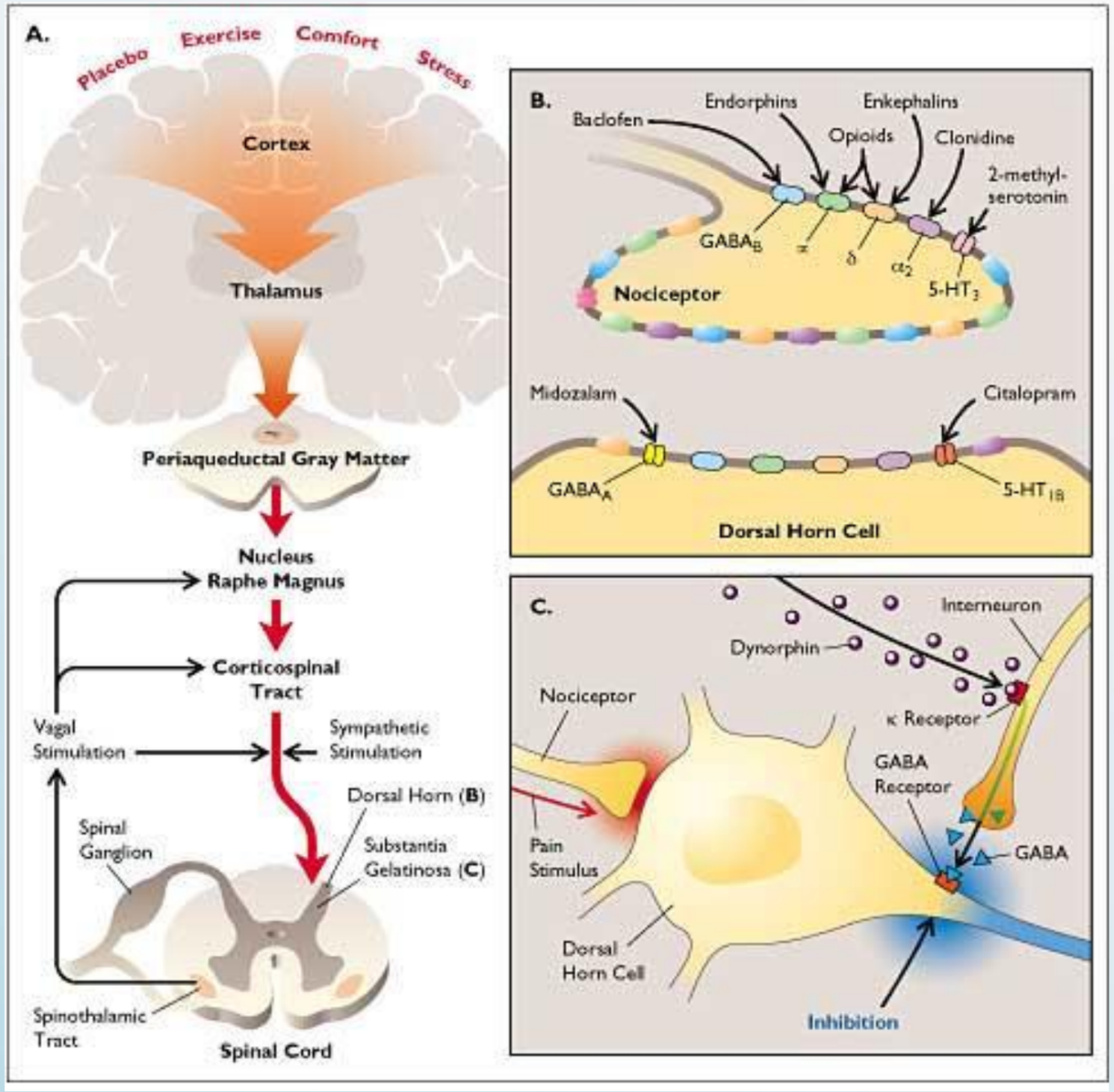
PAG projects neurons containing aspartate & glutamate that stimulate raph magnus nucleus (RMN)

RMN projects serotoninergic neurons, this in addition to noradrenergic neurons project from adjacent medulla to dorsal horn. They block pain signals by activating PIC

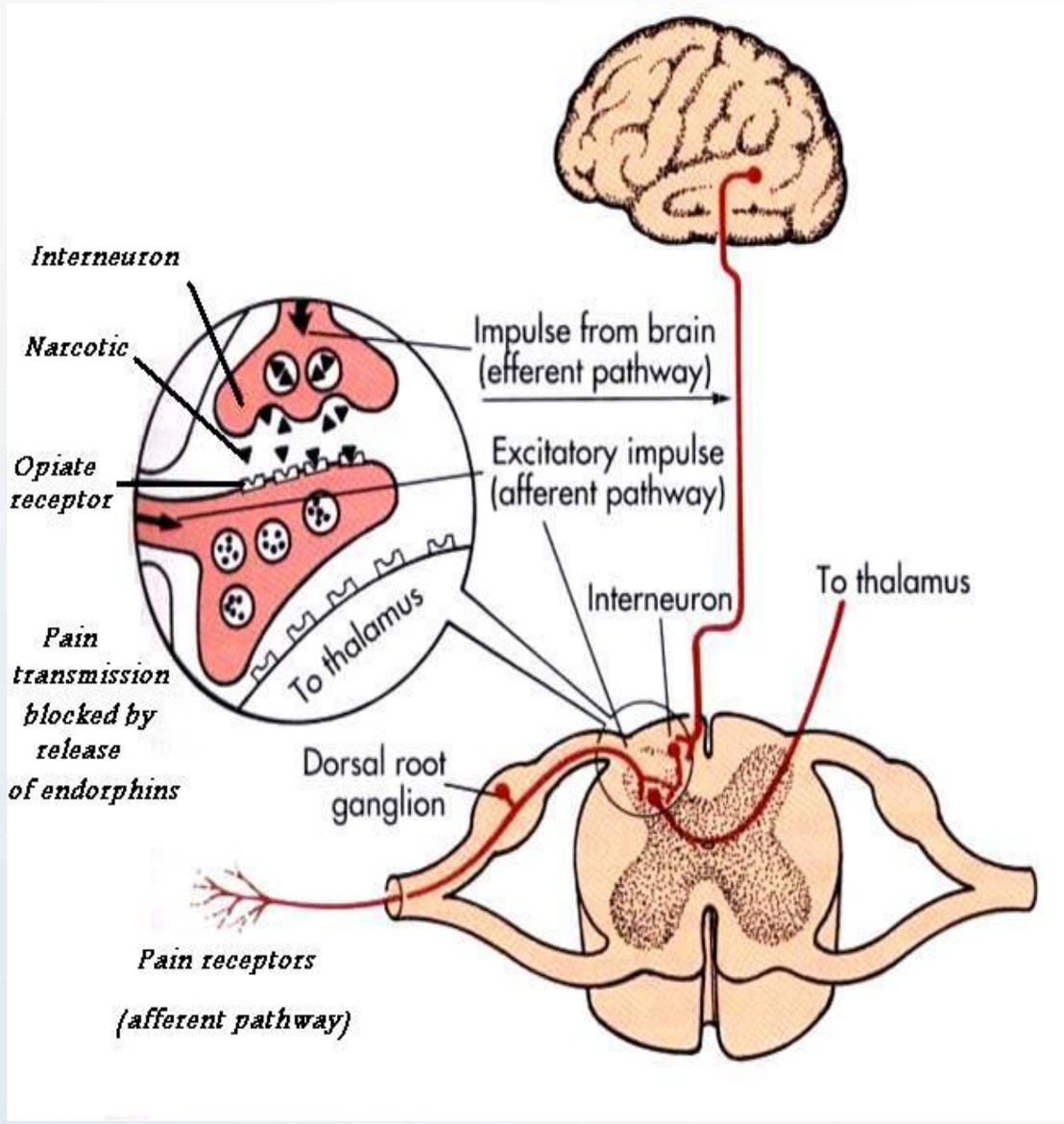
PIC will release encephalin which cause pre & post synaptic inhibition of pain transmission

Pain inhibitory complex (PIC) :

It consists of multiple short encephalinergic neurons that terminate on central endings of pain conducting afferent fibers.



SUPRA SPINAL MODULATION



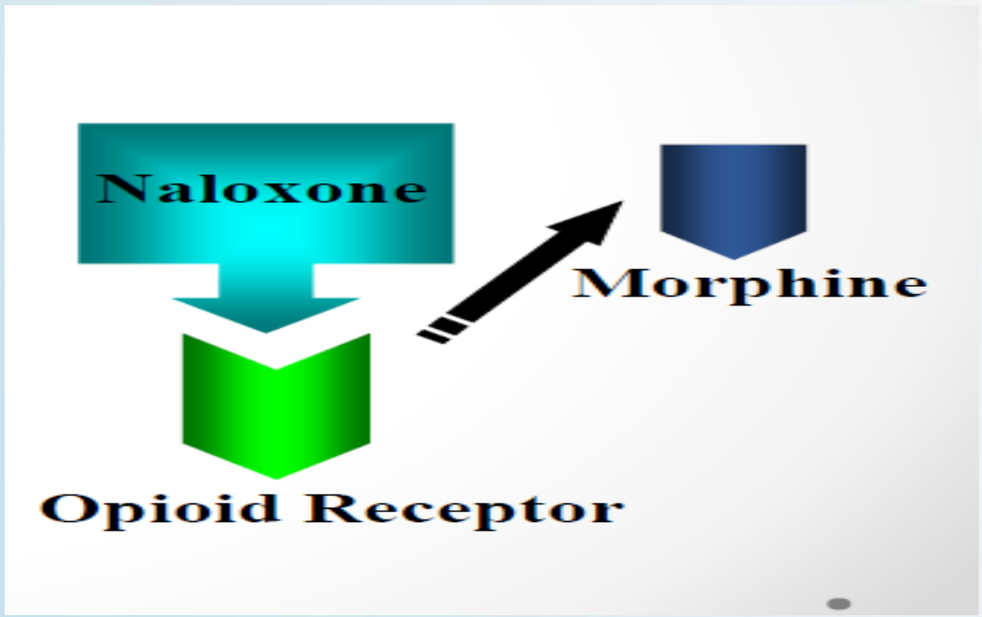
PAIN INHIBITORY COM

3.OPIOID RECEPTOR MODULATION :

- Opioid peptides are morphine-like substances present in body.
- They are natural analgesic substances that act by binding to opiate receptors in analgesic system and dorsal horn of SC on central ending of pain conducting pain fibers.

OPIOID ANTAGONIST: NALOXONE

Uses :	**reverse opioid overdose**
Mechanism :	**Displaces receptor-bound opioids (stop its action)**
Good option for :	**overcoming respiratory and CV depression**



MECHANISM OF OPIOID NEUROTRANSMITTER ACTION:

Endorphin:	**Neurons using endorphin or enkaphalin are found in PAG where they inhibit GABAnergic interneurons that normally suppress the anti-nociceptor neurons**
Enkephalin:	**It is used by interneurons in lamina II responsible for inhibiting the lamina – I nocioceptor-specific spinothalamic neurons**
Dynorphin:	**In hypothalamus, PAG, reticular formation, and dorsal horn.**
Endogenous morphin	**In terminals forming synapses with neuron having μ-opioid receptors in pain modulating pathways.**

TERMS FREQUENTLY USE :

Hyperalgesia	Excessive pain like **pain due to sun burn**
Allodynia	Pain caused by any other sensation **e.g. touch will cause pain.**
Muscular Pain	Less blood flow in the muscles **(ischemia).**
Causalgia	Burning pain.
Stress induced analgesia	Mild degree of pain is not felt if the other part of the body has excessive pain. **It's a well known phenomenon seen when the soldier is wounded in battle field but feels no pain until the battle is over. The cause is not known may be it is similar to Gate control hypothesis.**
Phantom pain	Pain felt in an amputated part long after amputation was done
Thalamic Syndrome	Obstruction of the thalmogeniculate branch of the posterior cerebral artery Affects posterior thalamic nuclei. Prolonged severe pain.
Trigeminal neuralgia	It is excruciating intermittent pain by stimulation of trigger area in the face **e.g. Washing of face, combing hair, blast of air on face.** It results from compression of trigeminal nerve root by blood vessels.

CHRONIC PAIN :

❖Chronic pain can be considered as bad pain because it persist long after injury and is often refractory to pain killers.
❖Chronic pain caused by nerve injury is called neuropathic pain.

NEUROPATHIC PAIN :

❖Caused by the damage to peripheral nerve.
❖The distal cut end develops a scar tissue forming rounded ball (neuroma) which is sensitive to pressure.
❖Repeated activation causes continuous pain.
❖Examples post herpetic neuralgia and diabetic neuropathy.

SITE & MECHANISM OF PAIN RELIEF :

- ✓Block production of inflammatory mediators.e.g. Aspirin & nonsteroidal antiinflammatories.
- ✓Sympathectomy can be useful. (cut the sympathetic innervation to the organ)
- ✓Exogenously administration of opoid like drugs.
- ✓Electrical stimulation of the dorsal column can alleviate pain originating below site of stimulation.
- ✓elective activation of large diameter afferent fibers by transcutaneous electrical nerve stimulation.
- ✓Stimulation of brainstem sites or administration of drugs which can modify serotoninergic or adrenergic neurons e.g. antidepressants.

And by the end of this lecture .. We finished our team work for the CNS block .. Good luck our future doctors

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CNS Block