Lecture 2

* Charectaristics of the sensory reseptors:

1) Differential sensitivity ⟶ the receptor differentiate between pain, touch, pressure..... (the modality)

2) receptive Feild ⟶ The area at which the stimuli affects ⟹ receptive field size $\propto \frac{1}{\text{density / acuity}}$ of the receptors

↑size of the receptive field ⟶ ↓acuity (discriminative) ⟶ ↓precise perception.

3) Lateral inhibition :- An inhibatory interneuron inhibits an adjacent neuron to inhance contrast between wanted and unwanted information e.g (The vision within the retina)

⟹ precise localization.

The process occurs by :- 1) Neuro-transmitters (mostly GABA / and it can be excitatory or inhibatory) 2) Receptors ⟶ determines the outcome ⟹ for GABA it's chloride channels).

4) Labeled line principle : each afferent fiber is labeled with a specific receptor within the cerebral cortex

so the brain can decode the stimulus location and type as each stimuli travel with a specific afferent (ascending) pathway to a specific area in somatosensory cortex

* The stimuli intinsity ⟹ ↑intensity ⟶ ↑magnitude of receptor potential ⟶ ↑frequency of action potential ⟶ ↑size of affected area ⟶ ↑receptors respond.

↑Temporal Summation ⟶ action potential frequancy ↑Spatial summation ⟶ increasing in receptors number.

* Receptors adaptation : constant intensity of a stimuli ⟶ ↓magnitud + frequency of the receptors potential ⟶ ↓ action potential as the Na channels return to resting.

it varies in : 1) Rate (slow, fast) 2) Extent (sudden, gradual).

Fast adapting ⟶ phasic, Rate receptors (eg:- pacinian corpuscle. receptors). slow ⟶ tonic (eg:- pain receptors).

* Tactile sensation:-

1) Touching ⟶ receptors on the skin or the direct underlying layer.

2) pressure ⟶ damage of the tissue ⟶ deep layer.

3) vibration ⟶ repeatitive touching.

4) itching 5) Tickling. ⟶ Superficial.

Types of receptors:-

1) pacinian corpusles ⟹ more deep, for pressure and vibration (why?) ⟶ Rapid adaptation

2) Messiner corpusles ⟹ more superficial, for touching, tickling

Tactile receptors : A and C

Aα Aβ Aγ Aδ

C

Receptor	A	C
mylein	✓	X
Diameter	big	small
velocity	high	low

Aβ ⟹ specialized receptors. for example Messiner's corpuscles.

free ending Aδ ⟹ myelinated.

C ⟹ Unmyelinated ⟶ transmit itching and tickling ⟹ superficial.

- slower than the specialized and myelinated. free ending
- can be stimulated mechanicaly or chemicaly (like itching for dialysis patients).

position receptors :- proprioception. : * Knowing the weight destrimination (weight of objects or weight on earth)

* important for Knowing the body position in state or dynamic. without the usage of our eyes.

} So by this the muscles effort can be determined.

* The body position is determined by the joints angles so all the receptors near the joints participating in this process.

* proprioception devided into 2 types :-

1) static position sense ⟹ concious perception of the orientation of different body parts with respect of each one to the another.

2) Rate of movement senses ⟹ Kinesthesia or dynamic proprioception

* Muscles spindles and pacinian corpuscles ⟹ detecting rapid rate changes ⟹ they're responsible for detecting the rate of movement ⟹ detected by Aα fibers.

* Thermoreceptors: all of them are free nerve ending, but they sense different degree.

cold receptors ⟶ A_δ more than C nerve fibers

warm receptors ⟶ C more than A_δ nerve fibers.

nociceptors (pain receptors).

all existed at the superficial skin and the underlying layer ⟹ free nerve ending so they are slow

* The larger the surface area the more accurate the senses of thermal changes.

Cold receptors are more than the warm.

* Transduction of thermal sensation not physical effect on the nerve endings it's chemical effect that lead to metabolic rate changes.

* Warm sensation transfer through ⟶ Transient receptor potential (TRP) Channels through vanilloid transient receptor potential (TRPV).

spicy food has a compound that's called capaicin ⟶ activating vanniloid

* cold sensation transfer through TRPM 8 which can be activated by mental.

Lecture 3

* Somatosensory pathways:

At each synapsing level we have integration (processing) → brain stem, thalmus.

A] first order neuron → from the sensory receptors through the spinal nerves and the cranial nerves to the brain stem were it synapse with the second order neuron.

↳ part of the CNS and the PNS (all the sensory neurons (Except the 1st.) are part of the CNS) + it ascends ipselaterally

B] Second order neuron → brain stem → thalmus we it synapse with the third order neuron. ⇒ the second order neuron decussate befor reaching the thalmus → Ascending contralaterally

C] Third order neuron → thalmus → primary somatosensory

1] posterior (dorsal) column - Medial Leminiscus ⇒ Sensory from the PNS → CNS ⇒ pro sensation Touch Vibration pressure proprioception → high conducting velocity.

it devides into: gracilis fasiculi → medialy + transmit info from the Lower part of the body (below T6).

gracilis coneatus → laterally + transmit info from the upper part of the body (T6 + above) } As we go up we go more medialy. Important for localization.

* first order neuron → from the PNS to the medulla. + it synapse with the second order neuron at the neucli (gracilis + coneatus).

* Second order neuron → at the medulla it decussate → to the thalmus where it synaps with the third order neuron.

* Third order neuron → from the thalmus → primary somatosensory area of the cerebral cortex.

2] Anterolateral spinothalmic pathway → Anterior and lateral horn of the spinal cord to the thalmus ⇒ slow conducting velocity (unmylinated+small)

↳ PTIT → pain Temperature Itch Tickle Crude touch Sexual sensation. Spinal nerves → Anteriolateral of the ventral horn of the gray matter of the spinal cord → thalmus → cerebral cortex

* first order neuron → from the dorsal root ganglia → shortly synapse with the second order neuron at the dorsal gray horn of the spinal cord (Doesn't ascend ipsilaterally).

* Second order neuron → At the dorsal gray matter of the spinal cord → decussate at the spinal cord → Anterior or lateral spinothalmic tract.

* Third order neuron → thalmus → somatosensory area of the cortex.

3] Trigemenospinal pathway → face, oral, Teeth, nasal cavity

* first order neuron → from the trigeminal nerve to the pons and the medulla.

* Second order neuron → decussate at the level of pons and medulla → Thalmus.

* Third order neuron → Thalmus → primary somatosensory area of the cerebral cortex.

* The brain areas:

1+2 → non specific info to control the excitability of the cerebral cortex } Brodmann area 1, 2, 3 → primary somatosensory area.

2+3 → to the opposit area of the cortex

4 → recieving incoming fibers and send them to the other layers.

5+6 → containing pyramidal cells for the descending/moter fibers to the PNS

1] primary somatosensory area ↳ detailed area for pinpoint locations

2] Secondary somatosensory area ↳ not well understood work for association

3] Somatosensory area for association ↳ to give a meaning for the sensation.

} for integration and perception + located posterior to the parietal lobe Posterior to the central gyrus.

The anterior area is responsible for the muscles, joints and tendons information → so it's close to the primary somatosensory area (S1).

" posterior " " " " " light movement → so it's close to the Somatosensory association area

Homonculus → lips and finger appearing large as they're very detailed processing containing lasts of sensory receptors.

* Because of the decussation hemisphere recieving sensory from the right side

Lecture 4 (pain)

	Fast pain	Slow pain
Receptors	Free nerve ending	Free nerve ending
Fibers	A_δ	C
Chronic	X	↘
Stimulus	Mechanical, Thermal	Chemical (inflammation).
Neurotransmitters.	Small molecules (glutamate).	neuropeptides.
pain nature	sharp/localized	Dull, poorly localized. more unpleasant. (suffering).
Site of pain	Not felt on the deeper tissues	Skin + deeper tissues.
Velocity	6-30 m/sec	0.5-2 m/sec.

Small molecule → stored in viscles at the nerve terminals once the action.p reach the nerve ending they get released (e.g glutamate) → Fast.

peptides → produced within the axon (e.g Substance P) → slow

2 pathway for pain transmition to the cortex:-

Fast. sharp pain pathway Slow Chronic pain pathway.

pain calssification:-

1] Somatic → Cutaneous (skin) or Deep (muscles + joints).
2] Visceral → Scarce distributed receptors. (Small intestine).
3] parietal → Sharp and localized. (pleura).
4] Neuronal → hyperalgesia + allodynia.

} cause:-
- pain intensity and rate of the area damaged.
- Localization and innervation density
- Autonomic responses.

* e.g:- Small intestine → ischemia would be more painful than dissection. why?

As the area affected in the case of ischemia is larger so we have Spatial Summation. (more receptors are affected).

Visceral pain:- Transmited through C fibers → slow, chronic, Suffering pain, aching

*Most important difference between viscera and the surface that the localized pain in the viscera rarely causes severe pain while the diffused pain causes the severe pain.

* some organs are completely insensitive like the liver paranchyma and the alveoli in the lungs while the liver's capsule, bile duct and the lung's pleura are highly sensitive.

* visceral pain is reffered pain → e.g pacrease injury causes back pain

parietal pain:- from the viscus the pain destributes to the parietal (pleura, pericardium, peritoneal).

* More localized pain + sharp as the parietals are supplied by extensive pain fibers from the peripheral nerves so the sensations are directly conducted through the local spinal.n

* Withdrawl reflex:- it's a protective spinal reflexes that triggers a rapid motor reflexes so it reduces the tissue damage → it's weak or lost in diabetic patients.

* Neospinothalmic tract:- for the fast pain transmition
  - The fibers Terminate in → Reticular formation.
  - → Ventrobasal Lamina (VBL) → through the dorsal coloumn- medial Laminuscal (most of them). ⇒ The same neuron order pathway
  - → posterior neucleus on the thalmus.

* Activation of the dorsal coloumn- medial laminuscal can cause sharp exact localized pain.

Slow pain ⇒ Through C fibers.

first order neuron dorsal root ganglia/synapse at the substantia gelatinosa with the second order neuron → second order neuron decussate at the substantia gelatinosa (at lamina I, II, III of the spinal cord) → Thalmus synapse with the third →

Third terminates at the intralaminar neulei in the subcortical region → as the slow pain needs emotional response ⇒ some end at the Reticular formation for concious awarness of pain.

* palespinothalmic pathway ≈ 10% - 25% go to the thalmus if not the fiber go to:
  - Reticular neucli at the mesencephalon, pons, medulla.
  - tectal area deep to the inferior and Superior colliculi.
  - periaqueductal that surround the aqueduct of slyvius.

brain lower part (intelaminal + ventral neucli) important in suffering type of pain.

Why we can't sleep during a severe pain → because of the arousal effect in the whole brain → Reticular formation.

* Localization is imprecise the pain can be localized on one organ not a specific point on it (e.g the hand not a specific point on the hand).

* So This ↑ phenomen explanis the diffuse, multi connectivity of this pathway

* Visceral pain → Dermatomal is orginated at the area from where it's embryonically orginated (e.g heart is orginated at the neck and the upper thorax).

☆ Heart + left shoulder are orginated from the same embryonic origin or dermatom.

☆ convergence → the ascending sensory neuron from the skin interfere with the second order neurons of the visceral organs → 1st order neuron share the same spinal segement and synapse at same spinal cord level.

☆ Labeled line phenomenon → The brain interprets the signals coming from the heart for example are coming from the left shoulder

* Endogenious Anelgesia ⇒ Stress inhibiting the pain HOW?!

Stress activates → paraventricular nuclei (in the thalmus) releasing → beta endorphins synapse with neuron → periaqueductas gray area (PAG) releases → Enkephalins synapse with neuron → Nucleus raphe magnus (NRM).

Sending signals to the spinal cord releasing → Serotonin local neuron → presynaptic inhibition of the pain pathway releasing of → Enkaphlins → presynaptic and postsynaptic inhibition of pain coming through C∂s fibers.

Released in the spinal cord.

periaqueductal + paraventricular → mesencaphalon + Upper part of pons → sending signals to NRM → lower part of pons + Upper part of the medulla.

Reticular nuclei + paragigantocellularis → lateral medulla → Releasing second signals to the dorsolateral coloumn in the spinal cord.

In the dorsal horn the analgesia inhibits pain signals before reaching the brain.

Control of pain:

1] Lateral inhibition: Activating Aβ fibers on the pripheral tactile receptors.

2] Stress: By activating the endogeneous analgesic system.

3] phamacological.

4] Exercise: it generates type of stress → activating the endogeneous analgesic system.

5] Acupuncture: vital energy called qi flows through body pathways called meridians → Activation of sensory neuron that releases neurotransmitters that work as analgesics as enkephalins + endorphins and dynophins.