Physiology of erection and ejaculation

Physiology of erection

- **Vascular mechanism of erection:**
  - *Flaccidity:* the smooth muscle of the corpora cavernosa (CC) contracts tonically and blood flow is minimal, only for tissue nourishment.
  - *Erection:* arterial vasodilation occurs along with the relaxation of smooth muscle and expansion of the CC. There is also compression of the subalbugineal venous plexus and emissary veins, reducing the venous outflow (corporal veno-occlusive mechanism).

- **Neurological pathways of erection:**
  - *Autonomic peripheral pathways:* the T\textsubscript{11}-L\textsubscript{2} sympathetic fibers (which produce detumescence) and the S\textsubscript{2}-S\textsubscript{4} parasympathetic fibers (which produce erection) are channeled through the cavernous nerves.
  - *Somatic peripheral pathways:* the dorsal penile nerve (a branch of the pudendal nerve) and the motor fibers of the Onuf nucleus (S\textsubscript{2}-S\textsubscript{4}) conduct penile sensitivity to the ischiocavernosus and bulbocavernous muscles, necessary for rigid erection and ejaculation.
  - *Higher centers:* the medial preoptic area and paraventricular nucleus of the hypothalamus, hippocampus, and cerebral cortex. These centers regulate the 3 types of erection:
    - Psychogenic erections: triggered by impulses from the brain.
    - Reflexogenic erections: in response to tactile genital stimulation.
    - Nocturnal erections: during REM sleep.

- **Neurotransmitters:**
  - *Flaccidity:* due to tonic smooth muscle contraction of the CC, adrenergic neurotransmission, and endothelial factors (*angiotensin II, PGF\textsubscript{2α}, and endothelins*).
  - *Erection:* the main neurotransmitter of erection is *nitric oxide (NO)*, which ↑ the production of cGMP, a relaxant of CC smooth muscle. Other neurotransmitters implicated are parasympathetic cholinergics and those mediated by *VIP* (vasoactive intestinal peptide). The erection ceases when NO release stops, phosphodiesterases (especially phosphodiesterase 5) degrade cGMP, and the sympathetic discharge of ejaculation occurs.

- **Sexual hormones:** testosterone maintains libido, but its ↓ does not always produce ED.

Physiology of ejaculation

- **Pathways and centers of ejaculation:**
  - *Afferent pathway:* sensory stimuli from the glans ascend along the pudendal nerve to the hypogastric plexus and the sympathetic ganglia T\textsubscript{10}-L\textsubscript{2}. From there they rise to the CNS.
  - *Ejaculation centers:* there are 3 centers in the CNS that regulate ejaculation:
    - The medial preoptic area of the hypothalamus.
    - The paraventricular nucleus of the hypothalamus.
    - Periaqueductal gray matter.
  - *Efferent pathway:* this dopaminergic route conducts the motor stimulus to the T\textsubscript{10}-L\textsubscript{2} sympathetic ganglia and from there to the S\textsubscript{2}-S\textsubscript{4} fibers of the pudendal nerve. This pathway is centrally and peripherally modulated by the serotoninergic neurons of the paragigantocellular nucleus, which inhibits ejaculation. SSRI drugs ↑ serotonin levels.

- **Phases of ejaculation:**
  - 1\textsuperscript{st} phase (emission): smooth muscle contraction of the prostate, seminal vesicles, vas deferens, and epididymis produces emission of semen to the posterior urethra. Here a pressure chamber forms between the internal (bladder neck) and external sphincters.
  - 2\textsuperscript{nd} phase (ejaculation): rhythmic contractions of the pelvic floor and bulbocavernosus and ischiocavernosus muscles cause the pressure chamber to open.
  - 3\textsuperscript{rd} phase (orgasm): sensory stimulus transmitted to the CNS.