Physiology of erection and ejaculation

- Innervation
  - Autonomic: sympathetic nerves originating from T11–L2, and parasympathetic nerves originating from S2–4, join to form the pelvic plexus. The cavernosal nerves, i.e., parasympathetic are branches of the pelvic plexus that innervate the penis. Parasympathetic stimulation causes erection; sympathetic activity causes ejaculation and detumescence (loss of erection).
  - Somatic: somatosensory (afferent) information travels and enters the spinal cord at S2-4. From S2-4, the somatic efferent (i.e., somatomotor) innervate the ischiocavernosus and bulbocavernosus muscles of the penis.
  - Central: medial preoptic area (MPOA) and paraventricular nucleus (PVN) in the hypothalamus are important centres for sexual function and penile erection
Each corpus cavernosum comprises a thick fibrous sheath, the tunica albuginea, which surrounds the erectile tissue. Each corpus has a centrally running cavernosal artery, which supplies blood to the multiple lacunar spaces, which are interconnected and lined by vascular endothelium.
Mechanism of erection

- Neuroendocrine signals from the brain, created by audiovisual or tactile stimuli, activate the autonomic nuclei of the spinal erection centre (T11-L2 and S2-4). Signals are relayed via the cavernosal nerve to the erectile tissue of the copora cavernosa. This triggers increased arterial blood flow into sinusoidal spaces (secondary to arterial and arteriolar dilatation). The result is expansion of the sinusoidal spaces against the tunica albuginea. Rising intracavernosal pressure and contraction of the ischiocavernosus muscles produces a rigid erection.

- Following orgasm and ejaculation, vasoconstriction due to increased sympathetic activity produces detumescence.

3 types of Physiological erections can occur:
1. nocturnal
2. Psychogenic
3. reflexogenic
Ejaculation

- Tactile stimulation of the glans penis causes sensory information travel to sympathetic nuclei. Sympathetic efferent signals cause contraction of smooth muscle of the epididymis, vas deferens, and secretory glands, propelling spermatozoa and glandular secretions into the prostatic urethra. There is simultaneous closure of the internal urethral sphincter directing sperm into the bulbourethra (emission), but preventing sperm entering the bladder. Rhythmic contraction of the bulbocavernosus muscle (somatomotor innervation) leads to the pulsatile emission of the ejaculate from the urethra.
Phases of erectile process

0 **Flaccid phase** Cavernosal smooth muscle contracted; sinusoids empty; minimal arterial flow

1 **Latent (filling) phase** Increased cavernosal artery flow; penile elongation

2 **Tumescent phase** Rising intracavernosal pressure; erection forming

3 **Full erection phase** Increased cavernosal pressure causes penis to become fully erect

4 **Rigid erection phase** Further increases in pressure + ischiocavernosal muscle contraction

5 **Detumescence phase** Following ejaculation, sympathetic discharge resumes; there is smooth muscle contraction and vasoconstriction; reduced arterial flow; blood is expelled from sinusoidal spaces
Factors influencing cavernosal smooth muscle

- Nitric oxide (NO)
- Vasoactive intestinal peptide (VIP)
- Prostaglandin E₁ (PGE₁)

Decrease in calcium → RELAXATION (erection)

- Noradrenaline (NA)
- Endothelin-1
- Prostaglandin F₂ (PGF₂)

Increase in calcium → CONTRACTION (flaccidity)
Secondary messenger pathways involved in erection
MALE SEXUAL DYSFUNCTION

is the inability to attain or maintain a penile erection sufficient for sexual intercourse, may involve also problems with emission, ejaculation or orgasm.

- **Premature (rapid) ejaculation**: persistent or recurrent occurrence of ejaculation with minimal sexual stimulation
- **Retarded ejaculation**: Is undue delay in reaching a climax during sexual activity.
- **Retrograde ejaculation**: backflow of semen into the bladder during ejaculation owing to an incompetent bladder neck mechanism.
- **Anorgasmia**: is the inability to achieve an orgasm during conscious sexual activity
Aetiology

- **Inflammatory** Prostatitis
- **Mechanical** Peyronie's disease
- **Psychological** Depression; anxiety; relationship difficulties; lack of attraction; stress
- **Occlusive vascular factors**  
  - *Arteriogenic*: hypertension; smoking; hyperlipidaemia; diabetes mellitus; peripheral vascular disease
- **Venogenic**: impairment of veno-occlusive mechanism (due to anatomical or degenerative changes)
- **Trauma**  
  - Pelvic fracture; spinal cord injury; penile trauma
- **Extra factors**  
  - Iatrogenic: pelvic surgery; prostatectomy
- **Neurogenic**
  - CNS: multiple sclerosis (MS); Parkinson's disease; multi-system atrophy; tumour
  - Spinal cord: spina bifida; MS; syringomyelia; tumour
  - PNS: pelvic surgery or radiotherapy; peripheral neuropathy (diabetes, alcohol-related)
* Chemical

Antihypertensives (beta-blockers, thiazides, ACE inhibitors)
Anti-arrhythmics (amiodarone)
Antidepressants (tricyclics, MAOIs, SSRIs)
Anxiolytics (benzodiazepine)
Anti-androgens (finasteride, cyproterone acetate)
LHRH analogues
Anticonvulsants (phenytoin, carbamazepine)
Anti-Parkinson drugs (levodopa)
Statins (atorvastatin)
Alcohol

* Endocrine Hypogonadism; hyperprolactinaemia; hypo and hyperthyroidism; diabetes mellitus
**Diagnosis**

**History**

- **Sexual**: onset of ED (sudden or gradual); duration of problem; presence of erections (nocturnal, early morning, spontaneous); ability to maintain erections (early collapse, not fully rigid); loss of libido; relationship issues (frequency of intercourse and sexual desire, relationship problems).

- **Medical and surgical**: hypertension; cardiac disease; peripheral vascular disease; diabetes mellitus; endocrine or neurological disorders; pelvic surgery, radiotherapy, or trauma (damaging innervation and blood supply to the pelvis and penis).

- **Drugs**: enquire about current medications and ED treatments already tried (and outcome).

- **Social**: smoking, alcohol consumption.

- An organic cause is more likely with gradual onset (unless associated with an obvious cause such as surgery, where onset is acute); loss of spontaneous erections; intact libido and ejaculatory function; existing medical risk factors; and older age groups. The International Index of Erectile Function (IIEF) or ED intensity scale can be used to quantify severity.
* **Examination**

  * **Full physical examination** (CVS, abdomen, neurological); digital rectal examination to assess prostate; external genitalia assessment to document foreskin phimosis and penile lesions (Peyronie's plaques); confirm presence, size, and location of testicles. The bulbocavernosus reflex can be performed to test integrity of spinal segments S2â€“4 (squeezing the glans causes anal sphincter and bulbocavernosal muscle contraction).

* **Investigation**

  * Blood tests: U&E; fasting glucose; PSA; serum testosterone; sex hormone binding globulin; LH/FSH; prolactin; thyroid function test; fasting lipid profile.
PSYCHOLOGICAL EVALUATION

Nocturnal penile tumescence and rigidity testing
Useful for diagnosing psychogenic ED

Penile Duplex ultrasonography
Penile arteriography
Cavernosography
1. **Lifestyle Changes**

* Regular exercise, a healthy diet, smoking cessation, and limiting use of alcohol can reduce the risk of ED.

* Perineal compression on penile arteries from long-distance cycling may also represent a modifiable risk factor for ED. Changing the bicycle seat or riding practices will often improve erectile function.
2. Changing Medications

When a patient complains of sexual dysfunction after taking a particular medication, it is important in many situations, changing the medication to a different class of agent. Antihypertensive agents, alpha-adrenoceptor antagonists, calcium channel blockers, and angiotensin-converting enzyme (ACE)-inhibitors may reverse ED in some patients.

Antidepressants may lead to sexual dysfunction benefit from watchful waiting, substitution (bupropion, nefazodone, buspirone, mirtazapine), drug holidays, selective serotonin reuptake inhibitor (SSRI), dosage reduction.
3. Psychosexual therapy
Aims to understand and address underlying psychological issues, and provides information and treatment in the form of sex education, improving partner communication skills, cognitive therapy, and behavioural therapy (programmed re-learning of couple's sexual relationship).

4. Oral medication

**Phosphodiesterase type-5 (PDE5) inhibitors:** sildenafil (Viagra); tadalafil (Cialis); vardenafil (Levitra). PDE5 inhibitors enhance cavernosal smooth muscle relaxation and erection by blocking the breakdown of cGMP. Sexual stimulus is still required to initiate events. Side-effects: headache; flushing; visual disturbance. Contraindications: patients taking nitrates; recent myocardial infarction; recent stroke; hypotension; unstable angina.

**Dopamine receptor agonist:** apomorphine. Apomorphine is administered sublingually, and acts centrally on dopaminergic receptors in the paraventricular nucleus of the hypothalamus to enhance and co-ordinate the effect of sexual stimuli.
5. Androgen replacement therapy

Testosterone replacement is indicated for hypogonadism. It is available in oral, intramuscular, pellet, patch, and gel forms. In older men, it is recommended that PSA is checked before and during treatment.

6. Intraurethral therapy

**Alprostadil (MUSE).** Synthetic prostaglandin E1 (PGE1) pellet administered into the urethra via a specialized applicator. Once inserted, the penis is gently rolled to encourage the pellet to dissolve into the urethral mucosa, from where it enters the corpora. Side-effects: penile pain; priapism; local reactions.
7. Intracavernosal therapy

alprostadil/Caverjet (synthetic PGE1); papaverine (smooth muscle relaxant) ± phentolamine (alpha-adrenergic antagonist). Training of technique and first dose is given by health professional. Needle is inserted at right angles into the corpus cavernosum on the lateral aspects of mid-penile shaft. Adverse effects: pain; priapism; haematoma

8. Vacuum erection device.

9. Penile prosthesis
THE END

THANKS