
Erectile Dysfunction

PRESENTOR

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Erectile dysfunction,

- Definition
- ED is the persistent inability to attain or maintain penile erection sufficient for sexual intercourse.

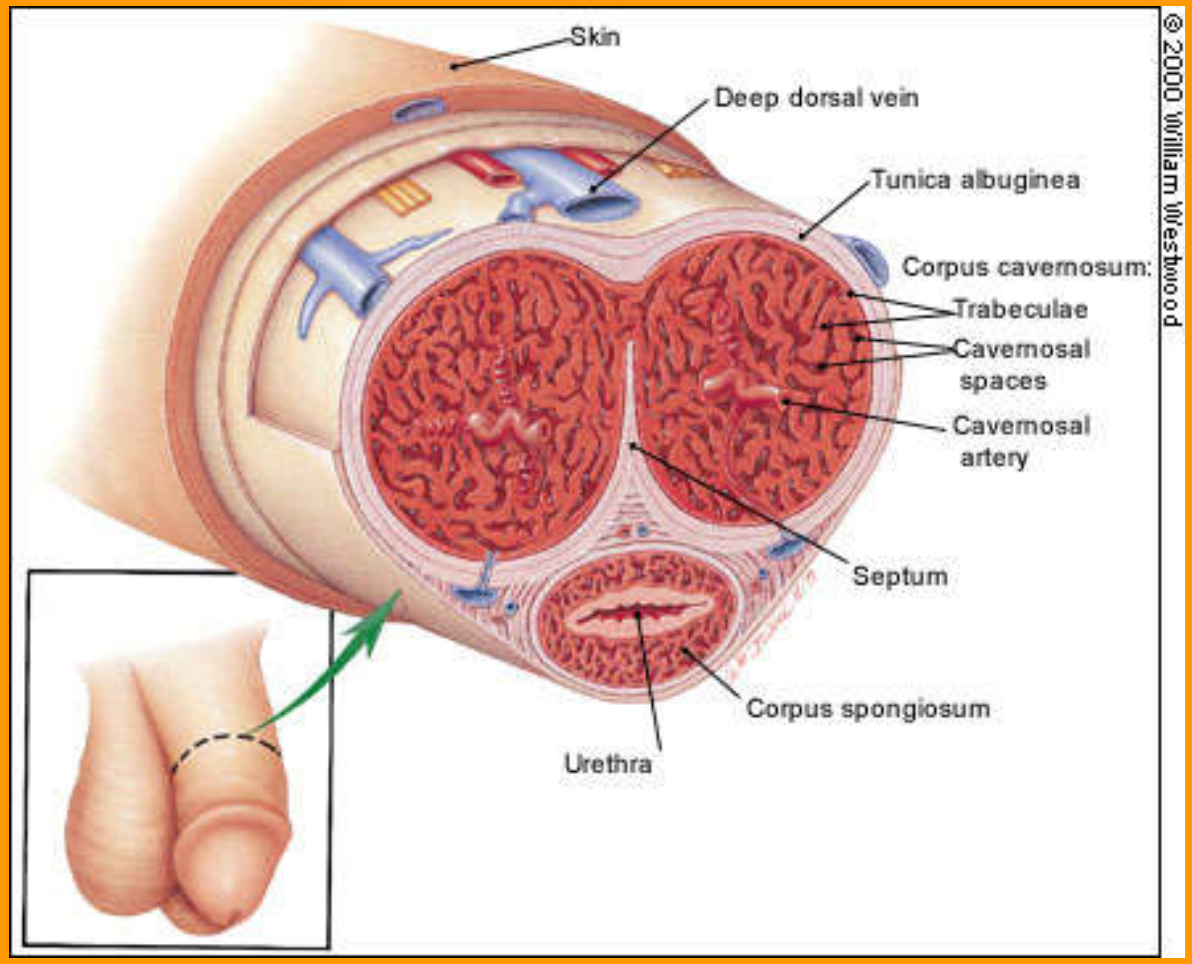
ANATOMY

The penis consists of:

1. Two parallel cylinders of erectile tissue, the corpora cavernosa, and
2. A smaller, single ventrally placed cylinder, the corpus spongiosum, which surrounds the urethra and distally forms the glans penis.

- The corpora cavernosa are composed of a meshwork of interconnected cavernosal spaces lined by vascular endothelium. They share an incomplete septum that allows them to function as a single unit.

DIAGRAM


Skin
Deep dorsal vein
Tunica albuginea
Corpus cavernosum:
Trabeculae
Cavernosal spaces
Cavernosal artery
Septum
Corpus spongiosum
Urethra
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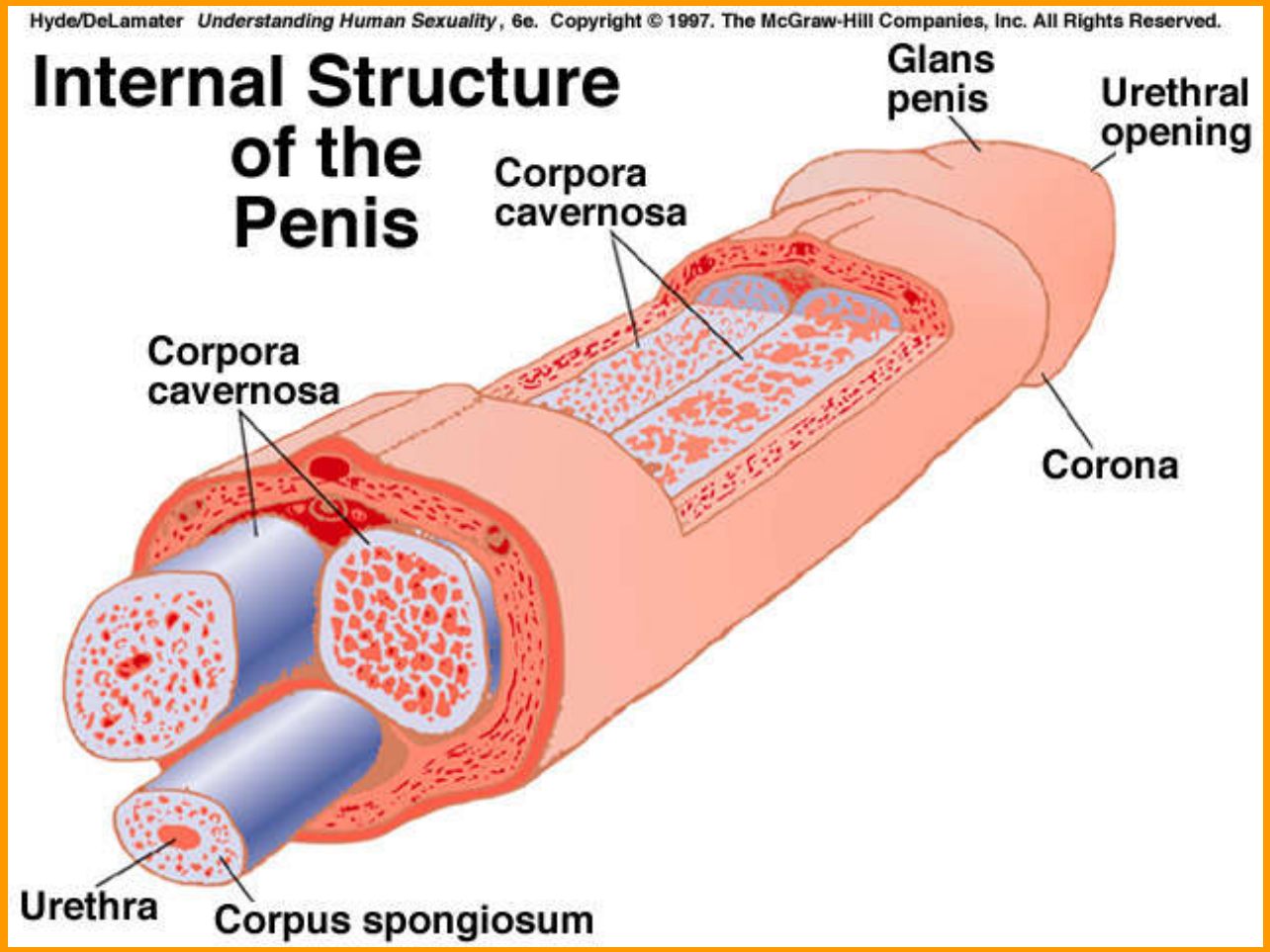
NERVE SUPPLY

- Autonomic and somatic nerves innervate the penis.
- Parasympathetic nerve fibers originate from sacral segments of the spinal cord, while Sympathetic nerves originate from lower thoracic and upper lumbar segments.
- Somatic sensory and motor fibers enter and leave the sacral cord and innervate the penis and the perineum via the pudendal nerve.

DIAGRAM


Hyde/DeLamater Understanding Human Sexuality, 6e. Copyright © 1997. The McGraw-Hill Companies, Inc. All Rights Reserved.
Internal Structure of the Penis
Glans penis
Urethral opening
Corpora cavernosa
Corpora cavernosa
Corona
Urethra
Corpus spongiosum

Physiology of Erection

- Normal male sexual function requires an interaction among
  - vascular,
  - neurologic,
  - hormonal, and
  - psychologic systems.

Multisystem Phenomenon

- The initial obligatory event required for male sexual activity, the acquisition and maintenance of penile erection, is primarily a vascular phenomenon, triggered by neurological signals and facilitated only in the presence of an appropriate hormonal milieu and psychological mindset.

Mechanisms

- Penile erection can be elicited by at least two distinct mechanisms,
- Central psychogenic and
- Reflexogenic, which interact during normal sexual activity.

Mechanism

- Psychogenic erections are initiated centrally in response to auditory, visual, olfactory or imaginary stimuli.
- Reflexogenic erections result from stimulation of sensory receptors on the penis which, through spinal interactions, cause somatic and parasympathetic efferent actions.

STAGES OF SEXUAL RESPONSE

The male sexual response may be divided into four distinct stages

- **Excitement**
- **Plateau**
- **Orgasm**
- **Resolution**

Excitement stage

- Erotic stimuli result in increased blood pressure and continuous blood flow to penis resulting an erection during the excitement stage.
- Penile erection occurs when blood flow to the corpora cavernosa and spongiosum increases as a result of dilatation of the urethral artery, the deep artery of the penis and the dorsal artery of the penis.

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- Psychogenic stimuli transmitted to the limbic system stimulate thoracolumbar (sympathetic nerves
- This in turn results in dilatation of the arteriolar vessels that supply the corpora cavernosa and spongiosum.

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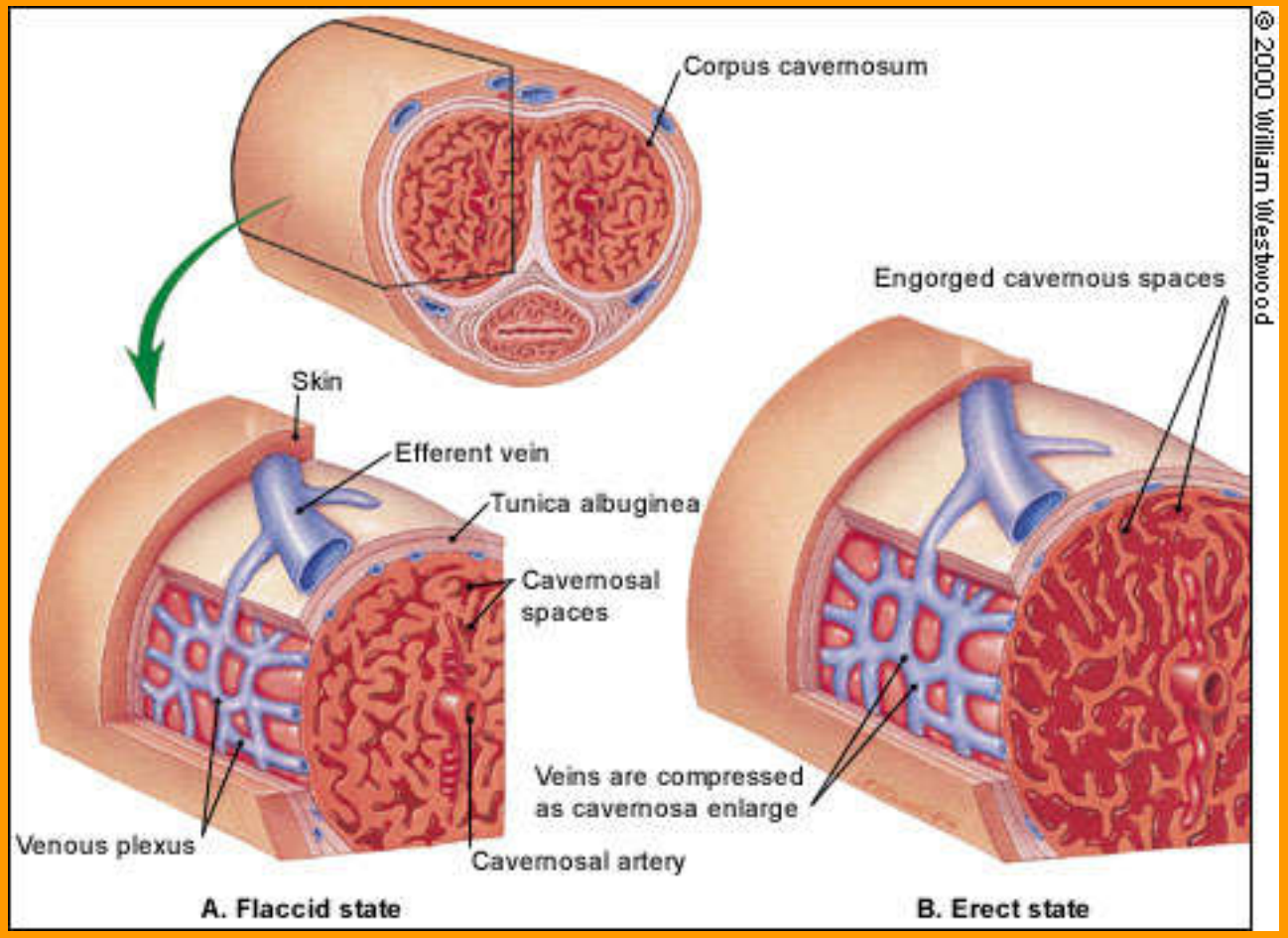
- **In addition to the thoracolumbar centre, sacral parasympathetic (S2-4) nerves may be activated by direct genital stimuli. The afferent limb for the spinal reflex arc is located within the pundendal nerves.**

Diagram


Corpus cavernosum
Skin
Efferent vein
Tunica albuginea
Cavernosal spaces
Venous plexus
Cavernosal artery
A. Flaccid state
Engorged cavernous spaces
Veins are compressed as cavernosa enlarge
B. Erect state
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Erection

- Inflow of blood increases as result of dilatation of arteries.And out flow is decreased
- 1)Closure of valves in penile veins
- 2) When penis grow in volume with the filling of lacunars spaces veins are constricted in between two layer of fascias.

ORGASM

Orgasm occurs in two stages.

- The first stage consists of reflex activity of the thoraco- lumbar sympathetic nerves, resulting in rhythmic contractions of the vas deferens, prostate and seminal vesicles.

Orgasm

- These contractions propel sperm and seminal fluid into the posterior portions of the urethra.
- Simultaneously, the bladder neck contracts by closure of the internal sphincter. So that semen does not enter the urinary bladder.

EJACULATION

- The presence of semen in the urethral bulb leads to a feeling of impending or inevitable ejaculation. The second stage of the organism phase is the ejaculatory reflex.

Ejaculation

- is mediated through the pundendal nerves, which induces rhythmic contractions of the bulbocavernosus and ischiocavernosus muscles. These contractions force semen out of the penis.
- During ejaculation, impulses are transmitted through the anterolateral tracts of the spinal cord to the cerebral cortex where orgasm is perceived.

EJACULATION

- The the urethral presence of semen in bulb leads to a feeling of limpending or inevitable ejaculation. The second stage of the organism phase is the ejaculatory reflex.

Resolution phase

- Following orgasm, the blood in the corpora cavernosa and spongiosum rapidly leaves the penis, resulting in a return to the flaccid state.

Pathophysiology

Penile erections involve an integration of complex physiological processes involving the

- CNS
- Peripheral nervous system
- Autonomic nervous system
- Hormonal
- Vascular systems

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- Any abnormality involving these systems, whether from medication or disease, has a significant impact on the ability to develop an erection, and experience orgasm.

Role of Trabeculae muscles

The degree of contraction of corpus cavernosal smooth muscle determines the functional state of the penis. The balance between contraction and relaxation is controlled by central and peripheral factors that involve many transmitters and transmitter systems.

Trabeculae muscles

- **At the cellular level, smooth muscle relaxation occurs following the release of acetylcholine from the parasympathetic nerves.**

LOCAL TRANSMITTERS

The nerves and endothelium of sinusoids and vessels in the penis produce and release transmitters and modulators that control the contractile state of corporal smooth muscles.

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The nitric oxide (NO) pathway is of critical importance in the physiological induction of erections. The drugs currently used to treat erectile dysfunction were developed as a result of experimental and clinical work that demonstrated that NO released from nerve endings relaxes the vascular and corporal smooth muscle cells of the penile arteries and trabeculae, resulting in an erection.

NO is produced by the enzyme nitric oxide synthase (NOS)

NITRIC OXIDE

- nNOS and eNOS are considered constitutive forms because they share biochemical features.
- These 2 subtypes use the biochemical pathway that targets cyclic guanosine monophosphate (cGMP).
- They are involved in the regulation of neurotransmission and blood flow, respectively.

CENTRAL EFFECTS OF NITRIC OXIDE

- Increasing evidence indicates that NO acts centrally to modulate sexual behavior and to exert its effects on the penis.
- NO is thought to act in the medial preoptic area and the paraventricular nucleus.
- Injection of nitric oxide synthase inhibitors prevents the erectile response in rats that have been given erectogenic agents.

CNS control

- Erections occur in response to tactile, olfactory, and visual stimuli. The hypothalamic and limbic pathways play an important role in the integration and control of reproductive and sexual functions.

CNS control conti

- The medial preoptic center, paraventricular nucleus, and anterior hypothalamic regions modulate erections and coordinate autonomic events associated with sexual responses.
- Afferent information is assessed in the forebrain and relayed to the hypothalamus

Hormonal influences

- The testicular hormone testosterone plays an integral role in normal male sexual function. The onset of adolescent nocturnal erections coincides with the pulsatile release of gonadotropin releasing hormone (GnRH) and luteinizing hormone (LH) activation of Leydig cell testosterone secretion.

Endocrine Role

- Testosterone deficiency results in impotence in experimental animals and men, and sexual potency returns when testosterone levels are normalized. This effect is the result of two actions of testosterone.
- Testosterone acts through psychogenic channels to enhance libido.
- Testosterone is necessary for maintenance of intrapenile nitric oxide synthase levels.

Etiology

Vascular

Endocrinology

Neurological

Psychiatric

Psychological

RISK FACTORS

- **Diabetes**
- **Smoking**
- **Hypertension**
- **Alcoholism**
- **Obesity**
- **Vascular disease**
- **Hyperlipidemia**
- **Cardiac problem**
- **Hypogonadism**
- **Hyperprolectinemia**
- **Hypertension**
- **Medicines and drugs**
- **Surgery**

- Psychiatric diseases
- Anxiety,Depression,
- Situational
- Lack of exercise
- PME
- Partner
- Ageing
- Pyronie,s disease

Smoking

Smoking

- Smoking exacerbate the the risk of impotence associated with cardiovascular disease and medication.
- Smoking can both active and passive.

Mechanism

- Vasoconstriction
- Vascular insult in form of micro injuries leading to plaque formation.
- Shortening of length and elasticity of penis
- Withdrawal effects and anxiety

Diabetes

- ED has been reported to occur in at least 50% of men with diabetes.Impotence may be present as first sign of diabetes in 12% of patients.Impotence is present in almost in all patients with diabetes who have manifestation of of diabetic neuropathy such as bladder dysfunction or decrease testicular sensation.ED is more associated with diabetic retinopathy ,peripheral neuropathy ,cardiovascular diseases,high levels of gylosylated HB,and use of antihypersive drugs.

MECHANISM

- Psychological
- Androgen secretion
- Peripheral nerve activity
- Endothelial cell function
- Smooth muscle contractility
- NO formation is impaired
- Advance gylcosylation end product act as oxidation agent and quench NO

Diabetes, implicated in ED

- Small arterials and arteriolar effect
- Neurological demyelinization
- Sinusoidal smooth muscle deterioration.longer the duration of diabetes less pronounce is neurogenic relaxations
- Up regulation of entdothelin receptor
- Impaired NO and prostacyline release.

Endocrine including Andropause

- Effects of androgens is well established in their on libido an sexual behavior but mechanism on erection is unclear.
- DHEA 0.5mg/ml associated with 16% impotence with levels of 5 to 10mg/ml it is 6.5% and 3.4%.Probability of complete impotence increase as DHEA decease while overall and moderate impotence probability remain unchanged.This support hypothesis that minimal impotent person would become complete impotent with decrease of DHEA levels.DHEA is an important predictor of cardiovascular diseases.

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- Treating hypogonadal person with exogenous testosterone increase rigidity and and nocturnal erection events.

Cardiovascular diseases and hypertension

- CVA ,M I,HYEPRTENSION ,HYPELIPIDEMIA ,LOW LEVEL OF HDL ATERIOSCLEROSIS,PERPHIRAL VASCUALR DISEASES HAVE HIGH CORELATION WITH ED.
- Fears , high drive of SNS are important factors
- 8%TO10% OF UNTREATED HYPERTENSIVE PATEINT WERE FOUND IMPOTENT.
- Venous leakage

Hypertension

- Thus, patients with hypertension are more likely to have ED; and patients with ED are more likely to have hypertension.
- The mechanism by which hypertension may cause ED is likely related to endothelial dysfunction associated with hypertension. Long-standing hypertension may cause oxidate stress, endothelial cell injury and its sequella, including the inability of the arteries, arterioles and sinusoids of the corpus cavernosum to dilate properly.

- It is also possible that non-endothelium-dependent impairment of vasodilatation such as damage to smooth muscle cells contributes to ED in the hypertensive patient. Another contributing factor may be the antihypertensive medicines themselves. For example, thiazide diuretics and beta blockers are known to worsen ED.[6], [7] In general, the calcium channel blockers and angiotensin converting enzyme inhibitors do not worsen ED compared to placebo.

- Of note, there are some small reports suggesting that angiotensin receptor blockers may actually improve ED.8 The mechanisms for the worsening of ED by thiazide diuretics has been postulated to be secondary to alterations in electrolytes, serum zinc deficiency or volume depletion, but the exact mechanism remains elusive.9 If ED is indeed related to a thiazide, patients may show improvement after several weeks of stopping the thiazide and switching to an antihypertensive agent that does not worsen ED

Medicines

- Diuretic ,
- H2 antagonist
- Cytotoxic drugs
- Antidepressant
- Antipsychotic
- Antihypertensives

Chronic diseases

- Parkinson's 60%
- MS 40% TO 80%
- MDD twice as common
- Chronic renal failure 40%
- Hepatic failure
- COPD

SURGERY AND TRAUAMA

- Spinal cord injury
- Prenial injury
- Prostactomy

INVESTIGATIONS

- Blood and urine complete
- Semen analysis
- Blood glucose fasting and random
- Lipid profile
- Serum Prolectin
- Free testosterone
- DHEA
- Color Doppler scan of penis

TREATMENT

- Oral agents.
- Phospho di estrase antagonists
- YOHIMBINE Oral testosterone
- Trazodone
- Koraen RED ginseng
- Gingo biloba
- DHEA
- L ARGININE
- Phenyl alanine
- Dopamine agonist drugs
- Testosterone Stimulator

Injectables

- I/C injections
- PAPAVERINE, + PHENTOLAMINE
- ALPRASTODIL

LOCAL APPLICATONS

- MINOXODINE
- NITROGYLERINE
- TESTOTERON PATCHES
- LOCAL ALPRSATODIL with name of topiglan

Vacuum constriction pump

- MUSE Medicated Urethral System for Erection.
- SURGICAL
- Implants,Rigid and Inflatable
- Revacularization
- Ligation of viens,
- Surgery for pyronies disease or after fracture penis


THANK YOU