Male Reproductive Tract: Penis Structure—Erection

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Glossary

Detumescence The inverse of tumescence, detumescence describes a state of the penile corpora cavernosa in which blood from erection-related veins drains back into the larger circulatory system.

Ejaculation An action of gushing fluid composed of semen and sperm, initiated by peristalsis from the semiferous tubule along the epididymis, seminal vesicle, ejaculation orifice, prostate urethra, external sphincter, membranous urethra and penile urethra to the urethral meatus.

Erection rigidity Refers to varying grades of erectile rigidity, with peak rigidity (a full erection) characterized by a bone-like hardness in the penis.

Intracavernosal pillars Fibroelastic tissues distributed between the 10 or 2 o'clock to 6 o'clock positions of the inner tunical layer of the corpora cavernosa. The pillars are intertwined with collagen bundles and elastic fibers.

Nitric oxide activities A key neurotransmitter to relax smooth muscle of the vascular endothelium within the sinusoidal wall of the paired corpora cavernosa, the lone corpus spongiosum, and the glans penis.

Parasympathetic activity The part of the autonomic nervous system that is responsible for regulating the penile sinusoids into tumescence, which results from increased penile arterial supply and decreased venous outflow when active; in the autonomic nervous system, it is the counterpart to sympathetic activity.

Penile arterial inflow An extent of penile artery supply to the sinusoids of the penis, it is largely routed to the corpora cavernosa where arterial supply speed varies from 2–3 mL/min to 60–80 mL/min.

Penile cavernosal-sinusoidal trapping A state of the penile sinusoids in which the penile arterial supply exceeds venous drainage, resulting in sinusoidal expansion.

Smooth muscles One of the muscle categories, the *smooth* muscles scatter along the vascular wall, gastrointestinal tract, reproduction tract, sinusoidal walls, etc.

Sympathetic tone The part of the autonomic nervous system that is responsible for regulating the penile sinusoids into detumescence resulting from increased venous outflow and decreased penile arterial supply; in the autonomic nervous system, it is the counterpart to parasympathetic activity.

Tunical outer longitudinal layer A tenacious collagen layer surrounding the corpora cavernosa, the collagen fibers in organized arrays interlaced with elastic fibers that form an irregular, latticed network on which the collagen fibers rest. This tissue determines drainage speed and the penile morphology and is the pivotal coat to protect an implanted prosthesis.

Tumescence Inverse of detumescence, tumescence describes a state of the penile corpora cavernosa characterized by sinusoidal expansion resulting from increased penile arterial supply and decreased venous drainage of erection-related veins.

Venous drainage A term that describes the return of sinusoidal blood back to the larger systemic circulatory system following an erection.

The Human Penile Structure and Erections

Comparative Anatomy in Mammals

In male animals, the os penis (the penis bone) exists in most species to achieve the best chance of paternity; this durable structure increases the coital frequency and often the duration as well. The penis bone, which is attached to the tip of the penis rather than the base, provides structural support for male animals that engage in prolonged intromission. The longest sexual intercourse was performed by a couple of rattlesnakes (*Crotalus L.*) that made love for 23 h and 15 min (Fig. 1). The coital duration is not proportionally correlated, though: for instance, while a male lion's os penis allows him to engage in an impressive 250 copulations in 4 days, each copulation can only last for a minute (which is still longer than that of a male rat, for whom copulation lasts only a few tenths of second). However, the male's hardy os penis makes it easy to get geared up for the next willing female shortly after his previous ejaculation. Although mounting is always required in mating behaviors, a male dog can swing its penis in a right-about direction after successful mating. This facility is particularly impressive, since there is neither a truly synovial joint nor a synarthrosis inside the canine penis. The answer might lie in the peculiar anatomy of the canine penis, which is composed of a long os penis in the front and a stout and minimally expandable corpora cavernosa at the rear.

Humans, woolly monkeys, and spider monkeys are the only primates lacking this hardy piece of anatomy. How do human males manage to function (sexually) without an os penis if, as conventional wisdom suggests, no os analog structure exists? It is a good question, but the human penis may have no need for an os penis given that erection rigidity is attained through a hydraulic
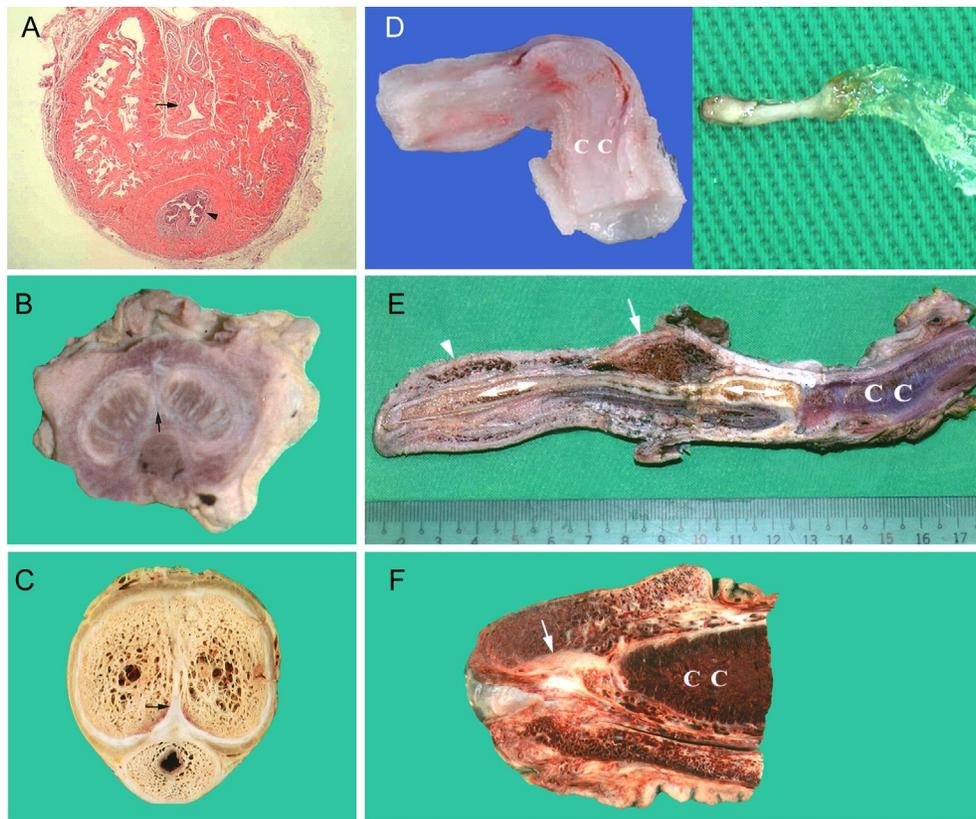

Fig. 1 Comparison of penile structure in different species, cross-sections of the corpora cavernosa and longitudinal aspect of the glans penis are from A to C and D to F, respectively. (A) In rats, the corpus cavernosum, devoid of the medial septum and intra-cavernosal pillars, is positioned between the deep dorsal vein (*arrow*) and the urethra (*arrowhead*) (H & E stain, reduced from 25 ×). (B) In dogs, a complete septum (*arrow*) and abundant intracavernosal pillars are obvious (1 ×). (C) In male humans, an intracavernosal pillar is not uncommonly encountered (not demonstrated). A septum (*arrow*) is significant, but incomplete and dorsally fenestrated. Note the clear delineation of the inner circular and outer longitudinal layers of the tunica albuginea (1 ×). (D) In rats, a short os penis is positioned between the glans penis and the corpus cavernosum (left panel, 7 ×). The amount of glanular tissue is scanty. The junction between the glans penis and the corpus cavernosum looks like a knee joint, which provides a flipping action during mating. The short os penis (right panel, 1 ×) can be better demonstrated after clearing and alizarin red S staining because only bony tissue can be preserved. (E) In dogs, the os penis (*double-headed arrow*) is enveloped with a unique glans penis of two compartments (*arrowhead* and *arrow*, respectively). Similar to the rat penis, the corpora cavernosa are not intromitted. However, they are reinforced with abundant intracavernosal pillars and a complete septum (1 ×). (F) In male humans, the distal ligament (*arrow*) within the glans penis is obvious and should be regarded as a ligamentous structure rather than a mere collection of sinusoids. The distal ligament is an aggregation of the outer longitudinal layer of the tunica albuginea and acts as a buttress of the glans penis (1 ×). Courtesy reproduction from *Journal of Andrology*, 26(5):624–628, 2005.

system rather than a bony structure. To know the relation of structure and peculiar function in the human penis, it is advisable to first know the comparative penile anatomy in varied species.

Recently a study was done on rat, dog, and human penises. In the rat's penis, although there is a short os penis, performing a flipping action with a rigid erection would be challenging, as neither a significant septum nor intracavernosal pillars is found in the corpus cavernosum (Fig. 1A). In dogs, with their long os penis, there is a complete septum between the paired corpora cavernosa as well as abundant intracavernosal pillars located behind the os penis (Fig. 1B). In human beings, for extraordinary hydraulic extensibility there is an incomplete septum with dorsal fenestration and moderately abundant intracavernosal pillars in the corpora cavernosa (Fig. 1C), which allows free communication between the two corppora cavernosa.

The anatomical structure of the glans penis varies between different species. In the glans penis of both rats (Fig. 1D) and dogs (Fig. 1E), there is a bony structure, i.e., the os penis. In these mammals, the os penis is the only supporting structure in the glans penis; there is no synovial joint in the species. In contrast to these os penises, the human glans penis contains an os analog, named the distal ligament (Fig. 1F). This newfound structure is inelastic and formed through an aggregation of the outer longitudinal layer of the tunica albuginea, in which neither a vascular component nor nerve tissue is seen. Moreover, many tributaries of supporting structures radiate directly from the distal ligament.

In histological sections, the os penis is composed mainly of type I collagens interlocked with type III collagens both in rats (Fig. 2A) and dogs (Fig. 2B) while connecting the os penis with its fibrous envelope. In humans, the distal ligament is composed of type I collagens (Fig. 2C), and neither osteocytes nor chondrocytes, characteristics of the bony structure, are found. The distribution of intracavernosal pillars is intermediate between the rat and dog; its pronounced wavy appearance results from extraordinary extensibility via the elastic component (Fig. 2D). Moreover, in the tunica albuginea of the corpora cavernosa, elastic fibers are sparse in the canine penis, few in the murine penis, and abundant in the human penis.

During mating, only the long os penis, associated with its glans penis, is intromitted, but the corpora cavernosa are not because these are deeply buried in the male body and can only act as a support of the long os penis. Therefore, either the junction between the os penis and the corpora cavernosa or that of the os penis to the glans penis is extraordinarily strong.

In dogs, the overwhelmingly abundant intracavernosal pillars and a complete septum will enhance the strength of the corpora cavernosa. In rats, the mounting duration is so short that these enhancing micro-architectures are not necessary. The resulting insufficient rigidity may, in turn, facilitate the flipping movement of the intromitted penis for the removal the semen plug deposited by a previous male. In human beings, the corpora cavernosa are intromitted. The intracavernosal pillars and the septum may increase dispensability and may, therefore, be mandatory in order to establish sufficient rigidity by congestion of the sinusoids if longer coital time is required. This suggests that a man may be susceptible to premature ejaculation if his penis is erect but
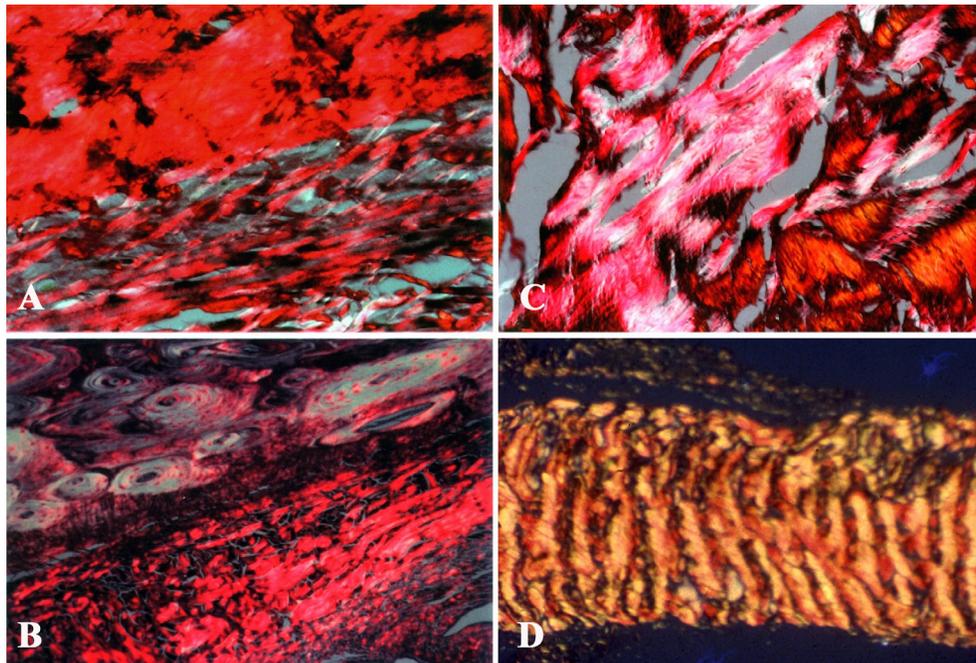

Fig. 2 Histology of the os penis, os analog, and intracavernosal pillars of the human penis. (A) In rats, the type I collagen with a reddish appearance is the major component of the os penis, which can be regarded as a woven bone (upper). The interlocking type III collagen with a greenish appearance is uncommon in the os penis proper, but substantial in its envelope (lower) (Picrosirius red stain, 400 ×). (B) In dogs, the appearance of a lacuna is characteristic. Osteocytes as well as chondrocytes are seen (Picrosirius red, 100 ×). (C) The os analog of human beings is similar to the os penis of a dog. Type I collagen is also the major component, with type III collagen as an interlocking ingredient. However, there are neither osteocytes nor chondrocytes (Picrosirius red, 400 ×). (D) In the human penis, the distribution of intracavernosal pillars is intermediate between the rat and dog (Fig. 1B); its pronounced wavy appearance results from an extraordinary extensibility via elastic component (Picrosirius red, 25 ×). Courtesy reproduction from *Journal of Andrology*, 26(5):624–628, 2005.

insufficiently rigid. A complete septum meets the requirement for establishing a pair of sufficiently strong corpora to buttress a long os penis such as that found in dogs, and the absence of this structure will be sufficient to support a short os penis whose mounting time is as short as a few of tenths second, as it is in rats. In human beings, the medium septum with its unique dorsal fenestration is observed, and the paired corpora cavernosa may be regarded as an ideal milieu to apply Pascals' law which states that pressure applied to any part of the enclosed fluid at rest is transmitted undiminished to every portion of the fluid and to the walls of the containing vessel. It appears to be enforced with intracavernosal pillars; sufficient erectile rigidity could not otherwise be reached. Not surprisingly, therefore, one donor artery is sufficient in attempting arterial reconstruction. Plenty of elastic components in the tunica albuginea of the corpora cavernosa thus increase the erectile capability of the penis. Overall, this implies that not only the increment of erectile length, but also the erectile girth of the human structure may, therefore, expand.

Essential Structures for Erection Rigidity in the Human Penis

Human males lack an os penis but instead possess an os analog—the distal ligament—located within the glans penis. It is complemented by the human penis's evolved hydraulic system. However, this particular evolution is not without its disadvantages because a hydraulic system is vulnerable to insufficient rigidity if veno-occlusive dysfunction exists. Unsurprisingly, it is common for physicians to encounter males who suffer from erectile dysfunction, which is defined as difficulty (or inability) to attain or maintain an erection of sufficient rigidity. It is also not uncommon for young boys to question their parents and ask why the penis is sometimes soft like a worm and sometimes hard like a bone. Although a functional brain is a prerequisite for healthy penile sexual function, a rigid erection can be a purely mechanical event arising within penis itself—some males can sustain a full, rigid erection resulting from reflexogenic activity (originating between the 2nd and 4th vertebrae of the sacral spine) despite sustaining an injury to the upper spinal cord.

It is necessary to describe the indispensable structures for achieving erectile rigidity. There are six essential structures to reach this aim: healthy sinusoids, a functional tunica albuginea (especially the outer longitudinal layer), intact intracavernosal pillars, an integral distal ligament, and normal ischiocavernosus and bulbospongiosus muscles. The seamless interplay of the tunica albuginea and cavernosal sinusoids is the primary step of a penile erection (Fig. 3). The cavernous artery delivers blood to two types of branches: firstly, to the outer capillaries, which are responsible for supplying nutrition to the penis in the flaccid state as well as the smooth muscle and nerve fibers; secondly, to the inner helicine arteries (Fig. 1, left), which open directly into cavernous spaces without entering capillaries, which are then emptied into the post-cavernous venules. These inner arteries are for fulfilling the erectile function; they are shaped like corkscrews and allow the penis to elongate and expand without compromising flow. Multiple layers of smooth muscle surround the helicine branches. This muscle is contracted while flaccid, allowing only small amounts of blood into the lacunar spaces. After the appropriate stimulus, muscle relaxation occurs and the arteries dilate and straighten, increasing blood flow and dilating the lacunar spaces and, in turn, applying pressure against the outer longitudinal layer of the tunica albuginea to reduce the blood drainage, which eventually results in a rigid erection. Application of real world physics would assume that the corpora cavernosa comprise an ideal vessel in which to apply Pascal's law, which states that pressure applied to any part of an enclosed fluid at rest is transmitted undiminished to all walls of the containing vessel. The cavernosal sinusoidal
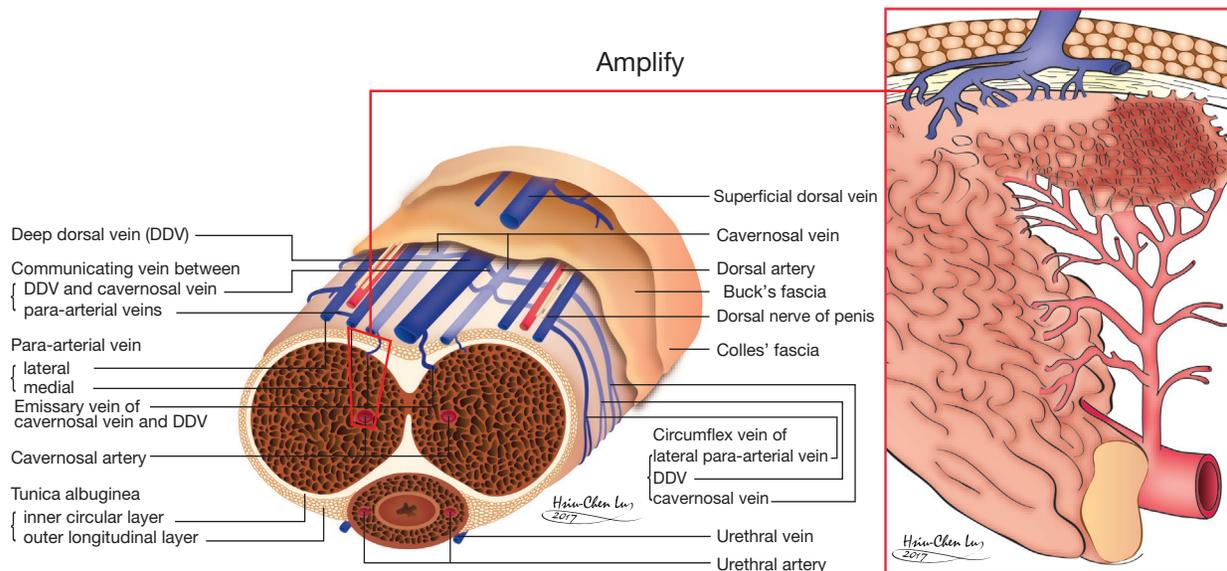

Fig. 3   An illustration of the relationship between sinusoids and emissary veins in human corpora cavernosa. Left: A cross-section of the human corpora cavernosa. Right: An amplified view of an emissary vein and its supplying sinusoids. Their anatomical relation is like a cluster of grapes, where the emissary veins are regarded as the grape stems and each sinusoid as the fruit.

expansion and decreased venous drainage result from tunical synchronizing limitation. The tunica albuginea is a bi-layered structure with a 360° inner circular and a 300° outer longitudinal layer which is the buttress against the sinusoidal wall to seal off the emissary veins which pass obliquely along the inner and outer longitudinal layer, an extension from the ischiocavernosus and bulbospongiosus muscles. The 360° inner layer encircles a cavernosal membrane to completely contain the erectile sinusoids and—together with the intra-cavernosal pillars—supports the cavernosal sinusoids. The distal ligament is an os penis analog and is arranged centrally and acts as a trunk of the glans penis to maintain patency of the distal urethra and preserve the range of ejaculation; it helps maintain the integrity of the entire penile structure while an intracavernosal pressure surge is conveyed distally. These structures, and their respective functions, are all indispensable for achieving penile erection and physiology.

Important Neuronal Center and Neurotransmitter for Erection

It is difficult, if not impossible, to enjoy a love life without a healthy working central nervous system. This is especially true for sexual activity because erection, emission, ejaculation, and orgasm are specifically coordinated by corresponding areas in the brain. The medial preoptic nucleus, paraventricular nucleus of the hypothalamus, and the limbic system, via the action of oxytocin, melanocortin and dopamine, are responsible for stimulating the spinal autonomic centers to cause erections. The sympathetic spinal center is located in the 12th thoracic vertebra to the 2nd lumbar vertebra and the parasympathetic spinal center is located in the 2nd to 4th vertebrae of the sacral spine. The baseline sympathetic tone originates from the intermediolateral gray matter of the thoracolumbar spinal cord in the 1st thoracic vertebra to 2nd lumbar vertebra. As a rule, a functional penis requires a functional heart. Penile sympathetic stimulation flows through several pathways, including the sympathetic chain ganglia (in the 1st thoracic vertebra) which also supply such structures as the heart and vascular system via the splanchnic nerve in the 5th thoracic vertebra to the 12th lumbar vertebra. The autonomic nerve fibers innervate the helicine arteries. The cholinergic nerve endings stimulate the nitrous oxide-synthase (NOS) and therefore the release of nitrous oxide (NO), which is the principal neurotransmitter causing penile erection.

Adrenergic nerve fibers and receptors are present in the cavernous trabeculae and surround the deep penile arteries. Sympathetic contraction is thought to be mediated by activation of postsynaptic alpha-adrenergic receptors and modulated by presynaptic alpha-adrenergic receptors. Noradrenaline is the major neurotransmitter controlling penile flaccidity and tumescence. Acetylcholine is required for vascular smooth muscle relaxation, and cholinergic nerves have been demonstrated within the cavernosal smooth muscle and surrounding penile arties.

Non-noradrenergic, non-cholinergic (NANC) neurons release NO, which in turn increases the production of cyclic guanosine monophosphate (cGMP), a second messenger, causing cavernosal smooth muscle relaxation. Other neurotransmitters, including vasoactive intestinal peptide (VIP), calcitonin gene-related peptide (CGRP), prostaglandins, and other peptides, are also thought to be involved in the erectile process. Therefore, it is understandable that both sexual stimulation such as sexual thoughts (psychogenic stimuli) and genital touch (reflexogenic stimuli) can produce a penile erection.

Human Erection Mechanism

Erection Physiology

The human erection results from a complex interplay of psychologic, hormonal, neurologic, vascular, and cavernosal contributors with a neuronal and endothelial nitric oxide effect. A firm erection requires good arterial blood inflow, trapping of sinusoidal blood, and limiting venous outflow of erection-related veins. It is commonly agreed to use six phases for describing an erection cycle. In order to provide a comprehensive overlook, the Table 1 Summary details the entire erection process. The autonomic nervous system controls penile state, which varies between flaccid and erectile states; two system sub-branches, the sympathetic and parasympathetic, are located in the 12th thoracic to 2nd lumbar vertebra and in the 2nd to 4th sacral spine, respectively. The higher center for the autonomic system is in the thalamus, and, unsurprisingly, the sympathetic and parasympathetic systems are also appointed to the thoracolumbar and craniosacral systems. The penis is usually kept in a spongy-soft flaccid state (Fig. 4) by a baseline sympathetic tone, which precipitates release of norepinephrine from penile adrenergic nerves, resulting in tonic contraction of the smooth muscle cells of the sinusoidal trabecula and penile arterioles as well. The human penis itself is a hydraulic system which depends on

Table 1   Summary of six phases for the erection process

Phases

Flaccid: Resting state with minimal arterial inflow at around 2–3 mL/min, which is good for physiological purposes
Latent: Sexual stimulation, either central or local, causes increase in arterial inflow which could elevate to 60–80 mL/min
Tumescence: Rapid elongation and girth expansion of penis resulting from sinusoidal expansion to limit the outflow of erection-related veins[a]
Full erection: Complete penile expansion characterized by high intra-corporeal pressure and decreased arterial inflow
Rigid erection: Ischiocavernosus and bulbospongiosus muscle contraction with further surge in intracavernosal pressure
Detumescence: Increased venous outflow and decreased arterial inflow until a flaccid state is resumed

[a]Penile erection-related veins (PERV) include one deep dorsal vein, two cavernosal veins, and two pairs of para-arterial veins.
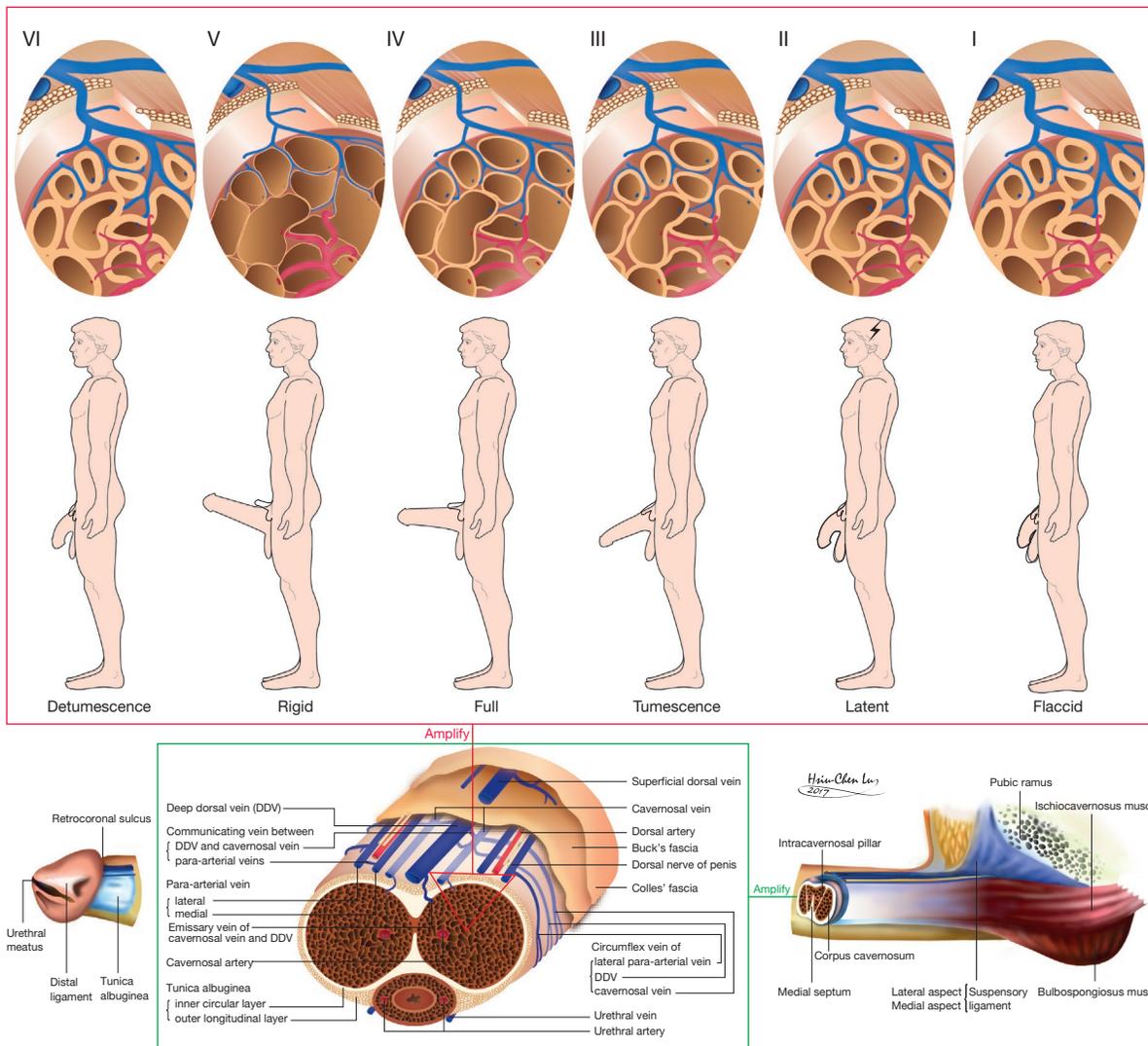
Fig. 4 A schematic illustration of the human erection process in six-phase cycle. Bottom: Lateral aspect. The penis leans upon the suspensory ligament, which is an extension of the linea alba. It is capped by the glans penis. Proximally, the corpus spongiosum is held by the bulbospongiosus muscle, in which the fibers are mostly transverse. The corpora cavernosa are surrounded by the tunica albuginea, which is a bi-layered structure consisting of an inner circular and an outer longitudinal layer with multiple sublayers. The intracavernosal pillars, which may be considerably larger distally, are a continuation of the inner circular layer. The corpus cavernosum is entrapped in the ischiocavernosus muscle, with the muscle fibers aligned in the longitudinal direction. Note the distal ligament within the glans penis. In the middle, a cross-section of the corpora cavernosa depicts the anatomy of the penile shaft. Uppermost: Pictures I–VI depict, step by step, that the penile helicine artery and cavernosal sinusoids dilate, thereby blocking the emissary veins; also depicted is the cavernosal sinusoids' response when a central sexual arousal ensues. The erection process is divided into six phases: flaccid, tumescence, latent, full, rigid, and detumescence, with the final step leading back to a flaccid state.

the vascular system; thereafter, this smooth muscle contraction results in significant resistance to arterial blood inflow and prevents cavernosal sinusoids from expanding, thereby keeping the penis in the flaccid state (flaccid phase). Not surprisingly, norepinephrine is released when a man experiences pain or stress, which in turn fades an erection even if the penis is in a fully erect state. Norepinephrine also likely inhibits the release of nitric oxide, thereby further inhibiting stimulation of erections; consequently, the male penis is kept in a flaccid state. Its counterpart parasympathetic system plays a role in re-starting the erection process.

Whenever sexual arousal ensues—originating either from the brain or from peripheral stimulation (latent phase)—there is a rapid impulse increasing parasympathetic activity which overrides the basal sympathetic tone. Increased parasympathetic tones result in a decrease in norepinephrine levels and an increase in acetylcholine release, which in turn increases nitric oxide synthase activity; subsequently, nitric oxide releases from both non-adrenergic, noncholinergic neurons and endothelial cells of vascular tissue and cavernosa sinusoids. Then, nitric oxide enhances, or inhibits the degradation, of both intracellular second messengers—cGMP (cyclic guanosine monophosphate) and cAMP (cyclic adenosine monophosphate). A profound smooth muscle relaxation (especially in the cavernosal sinusoids of the corpora cavernosa) results in encouraging helicine artery supply, increasing sinusoidal

expansion, and limiting venous drainage outflow of erection-related veins. It is well-elucidated that increasing nitric oxide neurotransmitters stimulates adenylate cyclase to elevate intracellular levels of cGMP. Both cGMP and cAMP are potassium channel openers that lead to hyperpolarization within the cells and, subsequently, cause closure of voltage-dependent calcium channels, which, along with intracellular organelle sequestration of calcium, leads to decreased intracellular calcium levels. An intracellular calcium drop causes the corporal smooth muscle relaxation, which enhances active dilation of the penile arteries, arterioles, and sinusoids, resulting in increased arterial inflow and sinusoidal expansion and in limited penile venous outflow of the erection-related veins. This ultimately leads to elevated intracavernosal pressure against the outer tunical layer of the corpora cavernosa.

Fulfilling effective erectile function requires a delicately synchronized cavernosal smooth muscle action, which is made possible by the presence of gap junctions located in the membranes of adjacent muscle cells. It is very likely that this synchronized erectile activity plays a pivotal role in the erection process because cavernosal smooth muscle tissue is only sparsely innervated by nervous tissue. These gap junctions allow the free exchange of a second-messenger molecules and larger ions such as calcium, which is indispensable for coordinated smooth muscle erectile activity. Active dilation of the cavernous and helicine arteries of the penis is the first event to occur during erection. This is closely followed by trabecular smooth muscle relaxation, which increases the compliance of the sinusoids, causing them to expand to accommodate the increased blood inflow. This expansion causes compression of the sub-tunical venous plexuses and emissary veins between the compliant venous sinusoids and the surrounding noncompliant tunica albuginea—particularly the outer longitudinal layer—thereby reducing venous outflow from the penis (tumescence phase). The rapid increases in penile arterial inflow results in pressure elevation within the penis that is comparable to systemic arterial levels. These arterial inflows include the bulbourethral artery, the cavernosal artery, and the dorsal artery to supply the sinusoids in the corpus spongiosum, corpora cavernosa, and glans penis, respectively or jointly. An intracavernosal pressures exceeding 90 mmHg can be regarded as the starting point of erection, and then further rapid elevation in pressure will cause a full penile erection (Full erection phase); in particular, provided that the patient does not suffer from venous leakage, further contraction of the ischiocavernosus and bulbospongiosus muscles raises the pressure several hundred millimeters mercury just within the corpora cavernosa (rigid erection phase). However, the sinusoidal pressure of the corpus spongiosum and glans penis sustains much lower intra-sinusoidal pressure due to the virtual absence of outer tunical layer and intracavernosal pillars. This pressure difference between these anatomical structures guarantees a smooth ejaculation and also provides a "cushion" protection for a sexual partner.

Ejaculation is facilitated by rhythmic contractions of the bulbocavernosus muscles, which contribute into the ventral thickening of the tunica albuginea, positioned at around the 5 and 7 o'clock positions. An intracavernosal pressure surge can be detected because of this anatomical relationship. Ejaculation is a typical sympathetic tone climax which results in noradrenaline secretion surge, which in turn, causes smooth muscle contraction and of course detumescence, ultimately returning the penis back to a flaccid state (detumescence phase). The reciprocating cycle of penile erections described in the preceding paragraphs is perpetual unless and until a male suffers from erectile dysfunction.

Tunical Outer Longitudinal Layer Is a Key Structure for the Erection Mechanism

Although the human penile anatomy and erection physiology have been extensively studied, we may still not thoroughly understand this unique organ. In the last few decades we have endeavored to shed light on the penile tunical and venous anatomy with the discovery of a bi-layered tunica and an intriguing model of penile erection-related veins. The former substantiates a model of the tunica albuginea of the corpora cavernosa as a bi-layered structure with a 360° complete circular layer (smooth muscle component) and a 300° incomplete outer longitudinal coat (skeletal muscle component) in contrast to a single inner circular layer. (This anatomical discovery raises additional questions: How can the erection process be fulfilled if no outer longitudinal layer exists? How can emissary veins be sealed off when passing the tunica if there is just one single tunical layer?) The latter proves that one deep dorsal vein, two cavernosal veins, and two pairs of para-arterial veins (as opposed to one single deep dorsal vein) are situated between Buck's fascia and the tunica albuginea for corpora cavernosa drainage. Thus, the corpora cavernosa communicates with the larger circulatory system via the penile arterial and venous vasculature.

During the Second World War, a critically injured soldier exhibited a rigid postmortem erection following the administration of an intracavernous blood transfusion at 25 mL/min. This incident implies that the human erection could prove to be, at its core, a mechanical matter. While it is generally accepted that the human erectile mechanism involves an intricate interplay among hormonal, vascular, neurological, sinusoidal, pharmacological, and psychological factors, the mechanism itself and the relative contribution of each respective component remain somewhat unclear, and merit further study. To identify a research model for differentiating venogenic factors from psychogenic factors in the ED contributor list, cadavers were deliberately chosen. A dynamic cavernosometry was performed in 48 cadavers for various hemodynamic studies (Table 2). Between 2002 and 2003, five fresh cadavers were obtained for demonstrating the pivotal role of the penile erection-related veins in a penile erection. That study was criticized for using normal saline with a speed of 150 mL/min, which far exceeds the arterial inflow rate of 60–80 mL/Min in human penile physiology. A second criticism questioned whether and how we could prove that the sinusoidal cells were totally dead. Although normal saline is used for volume expansion in medical purposes, the viscosity coefficient is zero, and, consequently, the infusion rate will be abnormally high. We therefore use 10% colloid and defrosted cadavers to address the two concerns. We investigated the role of venous outflow to isolate the key determinant of erectile function. The dynamic infusion cavernosometry and cavernosography were conducted on 15 defrosted human cadavers, both before and after the meticulous removal and ligation of penile veins. Preoperatively, an infusion rate of more than 28.1 mL/min (14.0–85.0 mL/min) was required to induce a rigid

Table 2   Summary of hemodynamic study on 48 valid human male cadavers in varied purposes from 2002 to 2013

Grouping	Cadaver		Infusion rate or volume			Purpose of study for penile erection-related veins[b]
	No.	Study time	Fluid	Preoperative[a]	Postoperative	
Fresh	5	2002–2003	Normal saline	1050 mL	89 mL	The pivotal role in erection
Defrosted	15	2009–2010	10% colloid	28.1 mL/min	7.3 mL/min	The principal component in erectile rigidity
Defrosted	7	2010–2012	10% colloid	30.2 mL/min	2.8 mL/min	Venous drainage of the corpora cavernosa
Defrosted	11	2011–2013	10% colloid			Role in penile veno-occlusive Mechanism
Total	48					

[a]The PERV stripping surgery.
[b]Penile erection-related veins (PERV) include one deep dorsal vein, two cavernosal veins, and two and pairs of para-arterial veins.

erection. Following surgery, we obtained the same result at a rate of 7.3 mL/min (3.1–13.5 mL/min) across the entire sample. The rigid erection is a mechanical phenomenon contingent on venous competence, and penile veins are the principal component for erectile rigidity. Similarly, seven cadavers were studied for exploring the venous drainage of the corpora cavernosa. Eventually, 11 valid defrosted cadavers were studied via electrocautery ($n = 6$) or ligation ($n = 5$); after exposing the defrosted cadaveric penises to eletrocautery with varied intracavernous pressure, we were able to prove (indirectly) that the outer tunica acts as the determinant structure of the veno-occlusive mechanism.

Clinical Relevance of Penile Erection

Diseases of the Penile Vascular System

There are penile arterial insufficiency and penile venous occlusive dysfunction in this disease entity. The failure to attain and/or maintain a rigid erection is a common symptom of penile arterial insufficiency or veno-occlusive dysfunction. Poor penile arterial function is commonly regarded as a major cause of vasculogenic erectile dysfunction; however, penile veno-occlusive dysfunction may be the far more prevalent one. The former is frequently regarded as an early sign of cardiovascular disease. Either arterial reconstruction or endovascular treatment are feasible options. Generally, any measure that benefits cardiovascular health will potentially improve penile arterial function. The prevalence of penile veno-occlusive dysfunction may be more widespread than commonly believed. Most impotent males suffer from this problem; one set of treatment options is characterized by venous intervention. Penile venous stripping is superior among these options, which also includes conventional venous ligation, venous coil embolization, and venous sclerotherapy.

Diseases of the Penile Cavernosal Sinusoids

The corpora cavernosa comprise the most ideal milieu in the human body in which to apply the Pascal's law. It is therefore the structure that is most vulnerable to malfunction resulting from penile arterial insufficiency, cavernosal fibrosis, and excessive venous drainage. The cavernosal fibrosis may be the most ill-defined disease despite extensive research. In the natural physiology, penile cavernosal sinusoids are not vulnerable to the aging process because the sinusoidal walls are at rest most the time, which distinguishes them from other end artery organs such as the retina (which typically rests only at night) and the kidney (which works 24 h/day). Penile implants remain the gold standard if cavernosal fibrosis is so severe that all other medical treatments fail.

Diseases of the Penile Spongiosal Sinusoids

The corpus spongiosum is the structure for guarding the passage of urine and ejaculate. A tethered penile ventral deviation happens when a fibrosis of the corpus spongiosum is severe enough to constrain the elongation ability; such a deviation also potentially constrains the passage of ejaculate.

Diseases of the Glans Sinusoid

The glans penis should not be completely rigid when the penis is erect; such rigidity would diminish the "cushion" effect between the penile shaft and the vaginal wall and thereby, as well, the patency of urethral ejaculation. However, some patients present concerns about an excessively soft glans, and questions regarding this matter are frequently asked in clinical setting. The character of the glans sinusoids is specific and divergent from that of the sinusoids in the corpora cavernosa and corpus spongiosum, respectively. Excessive venous drainage should be ascribed if a patient complains his glans is too supple, although a psychogenic origin cannot be overlooked.

Diseases of the Tunical Albuginea

The erectile morphology is determined by the tunica albuginea, which is the major component of the penile fibroskeletal system. Penile dysmorphology may be due to congenital penile curvature and acquired Peyronie's disease. In addition, a severe ventral curvature is not uncommonly presented because a tethering force is generated from a fibrotic corpus spongiosum, e.g., one affected by urethral manipulation syndrome, which is a term that describes urethral fibrosis caused by insertion of a catheter or other endoscopic instrument. In the most severe cases, surgical intervention is still the best viable option.

Diseases of the Distal Ligament

Although the distal ligament—an os analog in the human penis—is a relatively newfound structure, it is deemed an indispensable structure of penile integrity. This structure is so paramount that a traumatic disruption, although rare, causes the inability to engage in penile coitus despite otherwise intact erectile function of the corpora cavernosa. A surgical repair is required if traumatic disruption occurs.

Priapism

Priapism describes a situation in which the human penis remains in a rigid erectile state for over 6 h despite the absence of sexual stimulation. It is not uncommon to encounter in a clinical setting. It is commonly categorized into ischemic, non-ischemic, and recurrent ischemic types. The most prevalent is ischemic priapism, and it occurs when blood does not adequately drain from the corpora cavernosa; it is generally painful, resulting in a corporeal rigidity but a pale and non-engorged glans penis. On the contrary, in non-ischemic priapism—as the name suggests—the penile erection veins are not completely blocked. Non-ischemic priapism typically results from a shunt formed between the corpus spongiosum and an artery or disruption of the nerve of the parasympathetic nervous system, which enhances penile arterial inflow.

Medical history and physical examination is the first impression of the priapism which is further documented by sonography and cavernosal blood gas analysis. The most common cause of ischemic priapism is sickle cell disease followed by medication such as selective serotonin re-uptake inhibitors (SSRIs), cocaine, cannabis, and intracavernous injection agents such as papaverine or prostaglandin. Treatment strategy aims on terminating the underlying cause. In treating patients with ischemic priapism, it is typically advised to administer a nerve block (for pain relief) followed by aspiration of blood from the corpus cavernosum. A cavernosal measurement is advised by cold normal saline irrigation or, if that has no effect, then a phenylephrine injection. Non-ischemic priapism is often treated with cold packs and compression. Shunting surgery may be required if standard measures are not effective. Sequelae include erectile dysfunction and penile deviation.

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