

# Penile Doppler Ultrasound for Erectile Dysfunction: Technique and Interpretation

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**OBJECTIVE.** Erectile dysfunction (ED) is a common medical condition that has a high prevalence and incidence worldwide and may have a significant impact on both physical and psychosocial health. The purpose of this article is to review the role of penile Doppler sonography in the assessment of ED.

**CONCLUSION.** Penile Doppler sonography is an essential tool for differentiating between vascular and nonvascular causes of ED; therefore, radiologists must be familiar with the imaging protocol, the limitations of the technique, and interpretation of its findings, to warrant an accurate diagnosis and appropriate patient management.

**P**enile erection is a complex phenomenon that is coordinated by the interaction of the arterial, venous, and nervous systems. A defect or incoordination in any of these systems may result in erectile dysfunction (ED), which is defined as the persistent inability to attain or maintain penile erection sufficient for sexual intercourse [1–3]. ED is a common medical disorder that primarily affects men older than 40 years old, is strongly related to age, and has a high prevalence, having been estimated to affect approximately half of men between 40 and 70 years old [4]. ED may have a significant impact on both physical and psychosocial health and is closely related to early endothelial dysfunction; therefore, ED acts as a potential warning sign of cardiovascular disease.

The advent of new oral therapies, such as phosphodiesterase type 5 inhibitors, has significantly changed the diagnostic and therapeutic approach to ED over the past decades. Penile Doppler sonography (US) continues to play an essential role in the diagnostic workup of ED, mainly for patients with poor or no response to oral therapy. Doppler US examination is combined with an intracavernosal injection of vasoactive agents to exclude anatomic abnormalities and assess penile hemodynamics.

In the present article, we describe the scanning protocol and interpretation of different parameters and imaging findings in the diagnosis and classification of ED.

## Penile Anatomy

The penis comprises three cylindric endothelium-lined cavernous bodies, which consist of two corpora cavernosa located dorsally and the corpus spongiosum found ventrally. The corpora cavernosa are the main erectile bodies, and the corpus spongiosum distally forms the glans penis and contains the penile or pendulous portion of the urethra.

The male urethra is subdivided into prostatic, membranous, bulbar, and penile portions. The penile portion of the urethra extends from the suspensory ligament to the external urethral meatus, where an ampullar dilatation called the fossa navicularis is found [5].

Both the corpora cavernosa and the corpus spongiosum are surrounded by the tunica albuginea, a thick fibrous sheath comprising inner (circular) and outer (longitudinal) layers [6]. The corpus spongiosum is enveloped with a much thinner sheet because it lacks the outer longitudinal layer. The tunica albuginea separates the corpora cavernosa in the median plane, forming the septum of the penis, which contains multiple fenestrations in its proximal segment and allows free communication between the sinusoids of both sides.

Outside the tunica albuginea, the deep fascia layer (Buck fascia) surrounds the three corpora, and the superficial fascia of the penis (Dartos fascia) encompasses all the components of the penis (Fig. 1).

Arterial supply to the penis is provided by the internal pudendal artery, which is a

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branch of the anterior division of the internal iliac artery. The internal pudendal artery divides into three branches: the dorsal penile artery (supplying the glans penis, distal corpus spongiosum and penile skin), the cavernosal artery (supplying the corpora cavernosa), and the bulbourethral artery (supplying the urethra and proximal corpus spongiosum). The cavernosal artery divides by the helicine arteries, which directly communicate into the lacunar spaces of the corpora cavernosa. Multiple anatomic variations of the arterial penile distribution occur frequently and may be as high as 50% [7, 8].

Venous drainage from the three corpora is characterized by a triple drainage system involving deep, intermediate, and superficial systems [9]. The superficial system is contained in the Dartos fascia and comprises the superficial dorsal vein, which drains into the external pudendal vein. The intermediate system contains the circumflex and deep dorsal veins, which lie underneath the Buck fascia and drain into the prostatic venous plexus of Santorini and the internal pudendal vein. The deep system consists of the cavernosal and crural veins, which join the deep dorsal veins, the prostatic venous plexus, and the internal pudendal vein. The emissary veins receive the subtunical venular plexus, and they open into both the intermediate and deep venous systems (Fig. 2).

### Sonographic Penile Anatomy

The US appearance of the penis may vary in flaccid and erect states. On US, the corpora cavernosa manifest as well-demarcated cylindrical structures with intermediate echogenicity and homogeneous echotexture. The corpus spongiosum usually has higher echogenicity compared with the corpora cavernosa.

The tunica albuginea appears as a thin, linear hyperechogenic structure (generally less than 2 mm thick) encircling the corpora. Between the corpora cavernosa, a region of posterior acoustic shadowing is often seen, which corresponds to the penile septum [8, 10].

Within both corpora cavernosa, the cavernosal arteries are seen as narrow tubular structures with an echogenic wall in the longitudinal plane and as echogenic dots in the transverse plane. The dorsal veins appear at the dorsal aspect of the penis as anechoic compressible tubules and usually have detectable flow on color Doppler US (Fig. 3).

### Physiologic Profile of Penile Erection

Penile erection is a complex phenomenon that is based on the coordinated interaction of the arterial, venous sinusoidal, and nervous systems.

When the penis is in a flaccid state, the helicine arteries and the smooth sinusoids of the corpora are normally contracted; therefore, low-volume inflow and outflow exist as a result of the high-resistance vascular bed. When sexual stimulation occurs, the endothelium and the parasympathetic nerve terminals release nitric oxide, which is the primary neurotransmitter involved in penile erection. The nitric oxide triggers a molecular cascade that raises intracellular cyclic guanosine monophosphate levels and decreases intracellular calcium levels, leading to trabecular muscle smooth cell relaxation. This relaxation subsequently increases blood flow, trapping the incoming blood by the expanding sinusoids. The corpora become engorged, and the subtunical venular plexus and emissary veins are compressed against the tunica albuginea, limiting the venous outflow caused by passive venoconstriction and maintaining penile erection (Fig. 4).

Penile detumescence primary occurs when the adrenergic receptors on smooth muscles are activated and cyclic guanosine monophosphate is degraded (by phosphodiesterase type 5), allowing a rise in intracellular calcium. As a consequence, the smooth muscle cells contract, which permits drainage of the trapped blood on the lacunar spaces and reverses venoocclusion [11, 12].

### Erectile Dysfunction Causes and Initial Evaluation

ED is defined as the persistent inability to achieve, maintain, or both achieve and maintain a penile erection sufficient to engage in satisfactory sexual activity. Such dysfunction may have a significant impact on quality of life, affecting both physical and psychosocial life.

The cause of ED may be organic (neurogenic, hormonal, vasculogenic, or drug induced), psychogenic, or mixed (Table 1). ED most commonly has a mixed cause that involves both psychogenic and organic components [1]. The disorder is strongly related to age, and epidemiologic data have shown a high prevalence and incidence of ED worldwide. Among other studies, the Massachusetts Male Aging Study revealed that the combined prevalence of mild and moderate ED in men 40–70 years old is 52% [12].

Cardiovascular disease shares with ED the same risk factors, mainly hypertension, hypercholesterolemia, diabetes, and smoking. Several epidemiologic studies have reported that ED is a marker of endothelial dysfunction and can be an early manifestation of cardiovascular disease (especially coronary and peripheral vascular disease) [13–17].

The initial evaluation of any patient with ED includes a detailed medical and psychosexual history. The use of validated question-

**TABLE 1: Classification of Causes of Erectile Dysfunction**

Category	Conditions
Vasculogenic	Arterial: macroangiopathy or microangiopathy (e.g., atherosclerosis and trauma) Venous: corporal venoocclusive mechanism dysfunction Sinusoidal: failure to relax (e.g., fibrosis)
Neurogenic	Central causes: degenerative disorders (multiple sclerosis or Parkinson disease), stroke, CNS tumors, and spinal cord injury Peripheral causes: diabetes mellitus types 1 and 2, polyneuropathy, chronic renal failure, surgery (major surgery of pelvis and retroperitoneum, radical prostatectomy, urethroplasty, and other surgeries)
Hormonal	Hypogonadism, hyperprolactinemia, hyperthyroidism, hypothyroidism, Cushing disease, panhypopituitarism
Drug induced	Antihypertensive agents, antidepressants, antipsychotic agents, antiandrogens, recreational drugs (alcohol, heroin, marijuana, anabolic steroids, or other recreational drugs)
Anatomic or structural	Peyronie disease, penile fracture, hypospadias, epispadias, or micropenis
Psychogenic	Depression, anxiety, stress, or partner-related issues

naires, such as the International Index for Erectile Function, helps to assess all sexual function domains and the potential impact of a specific treatment modality [17].

A focused physical examination assessing the possible causes of and contributors to ED must be made to evaluate for signs of hypogonadism, penile deformities, prostatic enlargement, and cardiovascular and neurologic status. In addition, basic laboratory tests should be performed, including a glucose-lipid profile and a hormonal profile that includes collection of an early-morning sample to determine the total testosterone level [1, 18].

This stepwise approach to evaluating ED may benefit the decision-making process with regard to choosing the most appropriate treatment option [1, 12].

## Sonography for Erectile Dysfunction

US is the imaging method of choice for initial evaluation of the penis because it can assess anatomy and dynamic blood flow; is highly available, minimally invasive, and cost effective; and is well tolerated by the patient.

There are three principal US modalities that can be used during a penile study. The first, gray-scale or B-mode US, evaluates the penile anatomy and nonvascular abnormalities, such as plaques, fibrosis, tunica albuginea defects, masses, and fluid collections. The second modality, color Doppler US, allows simultaneous display of moving blood superimposed on a gray-scale image. It is used for the assessment of vascular flow and its direction. The third modality, spectral Doppler US, displays blood flow velocity over time as a waveform, so it is a graphic representation of the flow. It allows evaluation of the speed and direction of the flow. Spectral Doppler US can be displayed simultaneously on a gray-scale image (duplex US) or with the addition of color Doppler (triplex US).

For patients with ED, the main role of imaging is to differentiate between vascular and nonvascular causes [19].

The significance of penile Doppler US has decreased since the introduction of an oral phosphodiesterase type 5 inhibitor (sildenafil) in 1998, because good response to this effective drug involves correct functioning of arterial and venoocclusive mechanisms [13].

At present, penile Doppler US is principally used to evaluate the integrity of the vascular mechanism and exclude underlying arterial or venous insufficiency in patients with poor or no response to oral therapy (i.e., phosphodiesterase-5 inhibitor agents). ED is

a common complication after pelvic surgery (mainly related to prostate, rectal, or bladder cancers) and it may be caused by vascular injuries, neurogenic injuries, or a combination of both; therefore, penile Doppler US can be performed to confirm organic ED before penile prostheses surgery is performed [20].

Penile Doppler US is also recommended in the evaluation of posttraumatic ED, penile fibrosis, and penile curvature (Peyronie disease [PD]), ED occurring after priapism, candidates for a penile implant, primary ED, and medicolegal situations [21].

Recent data have documented that penile Doppler US is useful for detecting silent cardiovascular disease in men with ED, and it may be a valid tool to improve risk stratification of these patients [22, 23].

Arteriography and dynamic infusion cavernosometry or cavernosography remain the diagnostic reference standard for arteriogenic and venogenic dysfunction, respectively, and they should be performed only for patients who are being considered for vascular reconstructive surgery [1, 2].

## Ultrasound Protocol

When used to evaluate ED, penile US must be performed with the penis in both flaccid and erect states. Therefore, intracavernosal injection of vasoactive drugs is required.

Before the examination is initiated, the method and possible complications should be explained in detail to the patient, and verbal and written informed consent may be obtained in instances when applicable, especially with regard to the risk of priapism developing after intracavernosal injection. A calm, private, and comfortable environment is essential to ensure acceptable results.

The study should be performed using a high-frequency linear array (7.5–12 MHz), with the patient in supine position. The entire penis should be scanned through its ventral aspect in longitudinal and transverse views [24].

First, morphologic evaluation of the penis is performed using gray-scale or B-mode US to rule out anatomic abnormalities. Then, erection is pharmacologically induced by injection of intracavernosal vasoactive drugs into the lateral side of the proximal third of the penis. The most common vasoactive agent currently used is 10–20 µg of prostaglandin E1 [25, 26].

During the induced erection, dynamic assessment with color and spectral Doppler US is performed at the origin of both cavernosal

arteries. Spectral sampling of the cavernosal arteries is obtained at 5-minute intervals until maximal peak systolic velocity and minimal end-diastolic velocity values are reached (which generally requires up to 20 minutes, with a maximum of 30 minutes). It is important to maintain the narrowest possible box sampling and, in particular, a Doppler US angle of 30–60° during the examination, for the examination to be deemed valid.

## Doppler Ultrasound Diagnostic Criteria

The spectral waveform of the cavernosal arteries undergoes normal variations during erection. In the flaccid phase, the spectral waveform is monophasic with minimal diastolic flow. During the filling phase, there is an increase in both systolic and diastolic flow, followed by the tumescence phase in which there is the appearance of a dicrotic notch at the end of systole; hence, a low-resistance waveform is obtained. Progressively, the end-diastolic velocity decreases to zero (i.e., the tumescence phase), leading to diastolic flow reversal and corresponding to the full erection phase. During rigid erection, reduction in the peak systolic velocity (PSV) and disappearance or inversion of diastole are seen [5, 11, 26] (Fig. 5).

The hemodynamic parameters commonly used in Doppler US are as follows: PSV, end-diastolic velocity (EDV), and the resistance index (RI) (Table 2). The PSV and EDV are expressed in centimeters per second. The PSV corresponds to the maximum flow rate during systole, and the EDV defines the residual flow in a vessel at the end of the diastolic phase. The RI reflects the peripheral resistance to blood flow and is expressed using the following formula:  $RI = (PSV - EDV) / PSV$ .

Through evaluation of Doppler US parameters, the vascular response can be classified as normal or pathologic (denoting arterial insufficiency or venous incompetence). A PSV of 30 cm/s or greater indicates arterial competence after adequate pharmacologic stimulation. The EDV and RI assess the integrity of the venoocclusion mechanism, and the normal cutoff values are an EDV less than 5 cm/s and an RI greater than 0.8 [26, 27].

The PSV is the most accurate indicator of arterial disease. A PSV value of less than 25 cm/s is diagnostic of arterial insufficiency (Fig. 6). Intermediate PSV values (25–30 cm/s) are not specific [28]. A difference of greater than 10 cm/s between the PSV of both cavernosal arteries suggests arteriogenic ED [17, 29] (Fig. 7). High-velocity jets and

**TABLE 2: Penile Doppler Ultrasound Diagnostic Criteria**

Parameter	Diagnostic Values	Characteristics
Peak systolic velocity	> 30 cm/s: Normal 25–30 cm/s: Nonspecific < 25 cm/s: Arterial dysfunction A difference of > 10 cm/s between the peak systolic velocity of both cavernosal arteries suggests arterial dysfunction	Best Doppler ultrasound indicator of arterial dysfunction
End-diastolic velocity	< 5 cm/s: Normal > 5 cm/s: Venous dysfunction (if the patient has normal arterial function)	Best Doppler ultrasound indicator of venous dysfunction
Resistive index	> 0.9: Normal < 0.8: Venous dysfunction	Must be evaluated until maximal peak systolic velocity and minimal end-diastolic velocity are reached

damped waveforms on Doppler US are indicative of proximal arterial stenosis [17].

EDV is the best Doppler US indicator of venous dysfunction. In association with a normal arterial response, an EDV greater than 5 cm/s suggests venoocclusive ED, which manifests as persistent diastolic flow [38] (Fig. 8). Elevated EDV usually involves a decrease in the RI; hence, an RI of less than 0.80 is indicative of inadequate corpora blood retention (i.e., venous dysfunction) [18]. Continuous flow in the deep dorsal veins may also be seen on Doppler US during all phases, but it is not a requisite for diagnosis.

Objective gradation of penis rigidity must be reflected in the radiologic report, based on five stages, with stage 1 denoting no response; 2, mild tumescence; 3, tumescence without rigidity; 4, partial rigidity sufficient for penetration; and 5, complete rigidity [21].

A structured report is recommended (see Appendix 1) for the following reasons: it may improve the clarity and quality of the information, it reduces diagnostic mistakes, and it provides better communication between the radiologist and the referring clinician.

### Limitations and Complications of the Technique

In patients with arterial insufficiency, venous competence cannot be assessed using Doppler US.

Young patients may have false-positive findings of venous incompetence because of a suboptimal response to injection of prostaglandin E1 resulting from high anxiety and increased sympathetic drive. In this case, the study can be completed with administration of an intracavernosal injection of 2 mg of phentolamine ( $\alpha$ -adrenergic antagonist) [29].

Intracavernosal injection of prostaglandin E1 is not recommended for patients with a

penile prosthesis, a history of priapism, or conditions predisposing to priapism (e.g., sickle cell disease, myeloma, or polycythemia) [31].

Iatrogenic priapism (involuntary painful erection persisting for more than 4 hours) is the most important complication associated with the procedure. The incidence of priapism is low ( $\approx 1\%$ ) and can usually be successfully managed with a conservative approach (e.g., pharmacologic reversal with an  $\alpha$ -adrenergic agonist vs corpora aspiration) [32, 33].

Other possible complications are hypotension, dizziness, pain, hematoma, and long-term fibrosis.

The persistent absence of cavernous artery blood flow or a resistance index greater than 1.00 (absent diastolic flow) has been shown to be an objective predictor of priapism. If these findings are noted during ultrasound examination, patient management involves observation or elective treatment [19, 33].

### Causes of Secondary Vasculogenic Erectile Dysfunction

#### Peyronie Disease

PD, or induratio penis plastica, is a chronic benign fibrotic alteration of the penis that is of unknown cause and is characterized by the development of fibrous plaques or nodules within the tunica albuginea, typically on the dorsal aspect. Eventually, it can cause penile deformity, and it is the most frequent cause of painful penile erection [34]. PD usually affects men between 40 and 60 years old, and it can be associated with other fibrotic conditions (e.g., Dupuytren contracture and Ledderhose disease) and with vascular ED, both arterial incompetence and venous leakage [35].

Although MRI is the preferred modality for the assessment of PD, penile US is very

useful in the diagnosis and follow-up of PD. Imaging is required to assess the size and location of the plaques, detect small nonpalpable lesions or involvement of the penile septum, or evaluate disease progression.

On gray-scale US, penile plaques usually are seen as focal hyperechoic thickening of the tunica albuginea, which becomes more apparent after stimulation with vasoactive drugs, with or without calcification [19]. Plaques may sometimes present as a hypoechoic lesion associated with thickening of pericavernous tissue, because of the predominating fibrosis and interstitial edema (in initial stages) (Fig. 9).

In the acute phase, which usually lasts for 12–18 months, an increased Doppler signal indicating hyperperfusion around the plaques may exist and is considered a sign of inflammation in the active state of the disease [6].

Sonoelastography is used for estimation of tissue stiffness, and some studies have shown its utility in identifying plaques that cannot be detected by gray-scale US, especially noncalcified fibrous plaques [36–38].

#### Penile Fracture

Penile fracture is usually caused by trauma to an erect penis, most commonly during sexual intercourse, causing rupture of the tunica albuginea. In the acute setting, patients typically have a history of a cracking sensation, pain, and a suddenly swollen penis with loss of erection.

Penile trauma can lead to long-term complications, such as localized fibrous plaque formation or corpora scarring and vasculogenic ED [39].

The diagnosis of penile fracture is usually clinical, but US is a useful imaging tool, both in the acute and chronic phases, allowing localization of any tunical rupture. On US,



penile fracture is seen as a hypoechoic breach in the tunica albuginea, in particular along the longitudinal view of the penis, and an associated collection may be seen [17] (Fig. 10).

Segmental fibrosis can result in untreated albugineal disruption, limiting corpus cavernosum expansion and, consequently, ED, mainly as a result of failure of the venoocclusive mechanism [39].

## Priapism

Priapism is a rare abnormality defined as a painful erection that lasts for more than 4 hours. It can be classified as high-flow (non-ischemic) or low-flow (ischemic) priapism. High-flow priapism is commonly associated with pelvic or penile trauma, leading to fistula formation between the cavernosal artery and the cavernosal sinusoids. Low-flow priapism is the most frequent type, and it is a medical emergency because of the risk of tissue necrosis resulting from sinusoidal thrombosis and venoocclusion. If untreated, low-flow priapism can lead to irreversible tissue damage and permanent ED [39].

On US, cavernosal fibrosis is seen as fine echogenic strands located around the cavernosal arteries and replacing the sinusoids of the corpora cavernosa, generally without appreciable changes after injection of vasoactive drugs, and Doppler US parameters usually indicate venogenic dysfunction [21].

## Conclusion

Despite the fact that introduction of oral phosphodiesterase type 5 inhibitors has given rise to changes in the clinical and diagnostic management of ED, penile Doppler US still plays an important role in the assessment of ED. It allows baseline evaluation of the functional anatomy while providing real-time assessment of the dynamic changes experienced in response to the dosing of vasoactive medications.

Becoming familiar with the sonographic protocol, the limitations of the technique, and interpretation of imaging features of penile Doppler US are essential to differentiating between the vascular and nonvascular causes of ED and therefore determining appropriate management of the patient.

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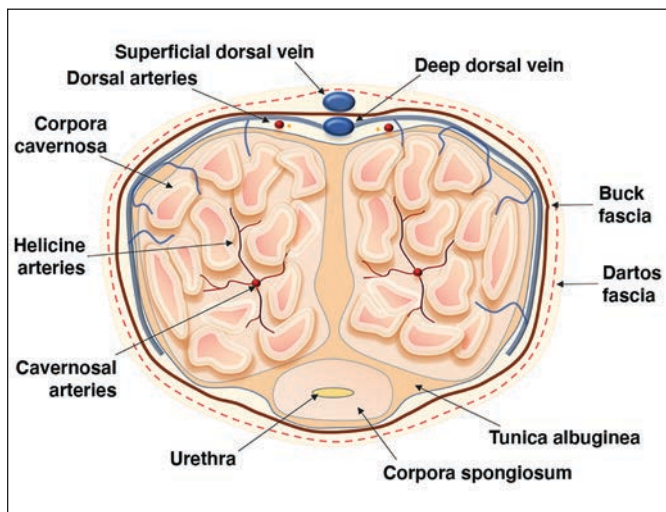
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## APPENDIX I: Checklist for Reporting Penile Doppler Ultrasound

1. Nature and dose of vasoactive agent(s) used
2. Vascular assessment
  - a. Cavernosal arteries

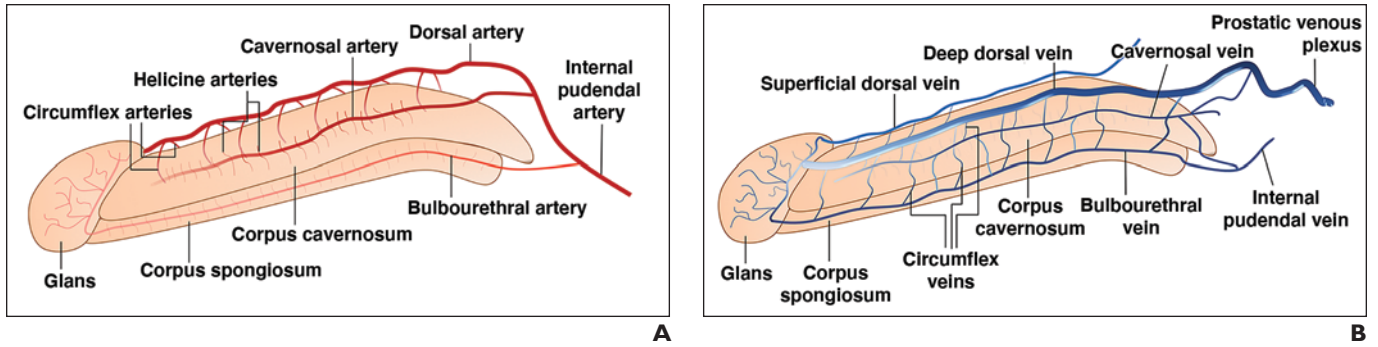
Minutes After IV Contrast Injection	Peak Systolic Velocity (cm/s)		End-Diastolic Velocity (cm/s)		Resistive Index	
	Right	Left	Right	Left	Right	Left
0						
5						
10						
15						
20						
≥ 30						

- b. Has maximal peak systolic velocity been reached and when? Has continuous flow in the deep dorsal vein been shown during all erection phases?
3. Nonvascular abnormalities: calcifications, fibrosis, albuginea defects, and other abnormalities
4. Degree of erection achieved
  - a. No response
  - b. Mild tumescence
  - c. Tumescence without rigidity
  - d. Partial rigidity sufficient for penetration
  - e. And complete rigidity
5. Other comments: deformity shown during the examination, immediate complications, and other comments



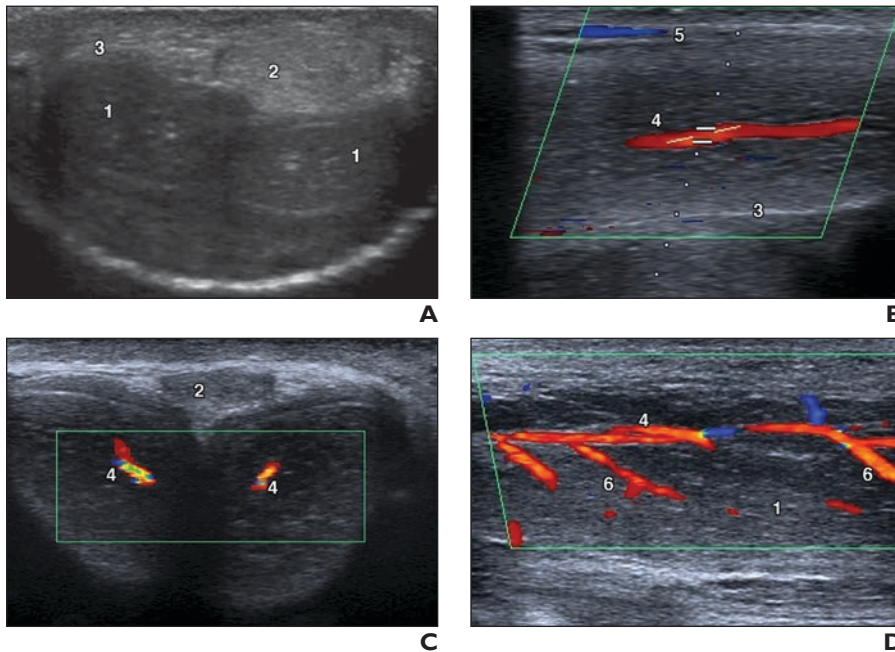
**Fig. 1**—Normal anatomy of penis. Transverse drawing shows corpora cavernosa and corpus spongiosum surrounded by tunica albuginea. Cavernosal arteries and helicine arteries are located within corpora cavernosa, and corpus spongiosum contains urethra. Buck fascia encircles three corpora, deep dorsal vein, and dorsal arteries, and Dartos fascia encompasses all components of penis. Superficial dorsal vein is located between Buck fascia and Dartos fascia.

## Penile Doppler US for ED



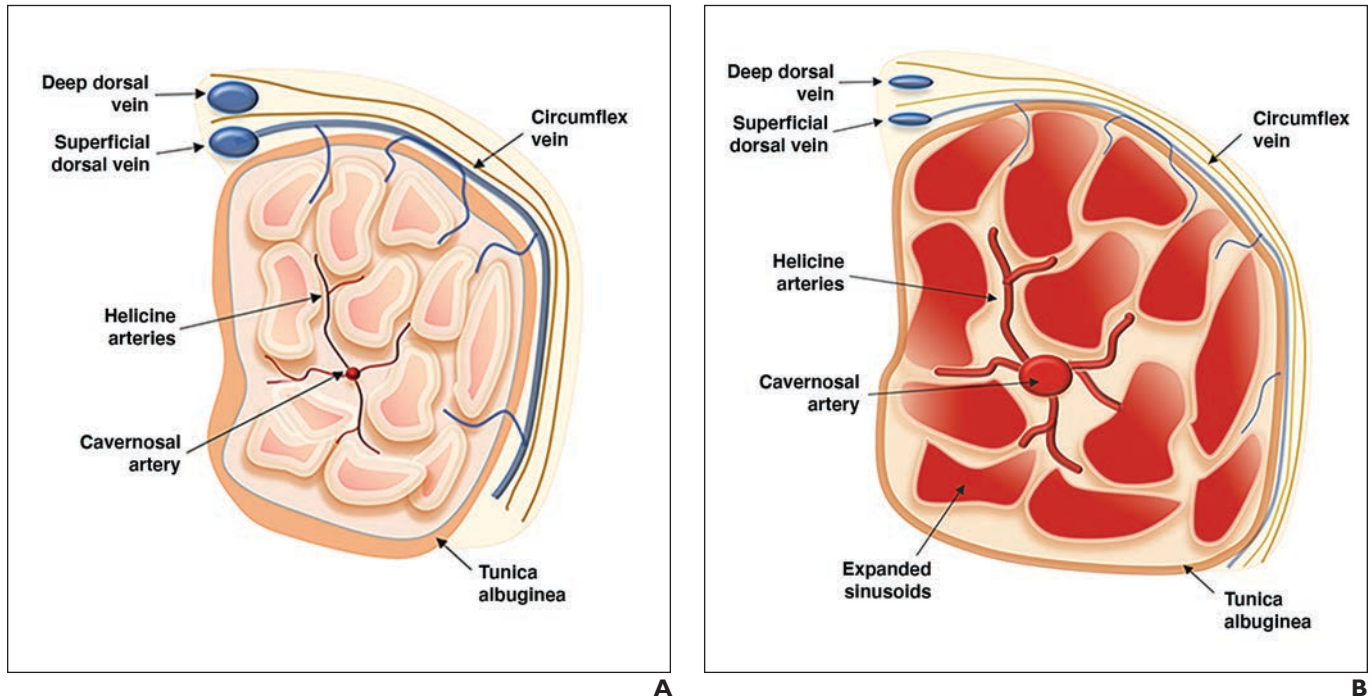
**Fig. 2**—Penile vasculature.

**A and B,** Drawings show arterial supply (**A**) and venous system (**B**) of penis.


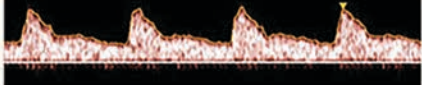
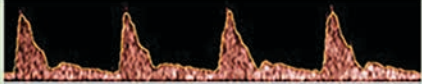


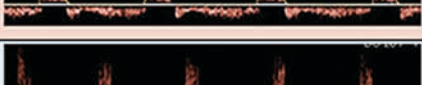


**Fig. 3**—Sonographic images of anatomy of penis. **A–D,** Ultrasound images obtained in transverse (**A and B**) and longitudinal (**C and D**) planes show corpora cavernosa (1), corpus spongiosum (2), tunica albuginea (3), cavernosal artery (4), deep dorsal vein (5), and helicine arteries (6).





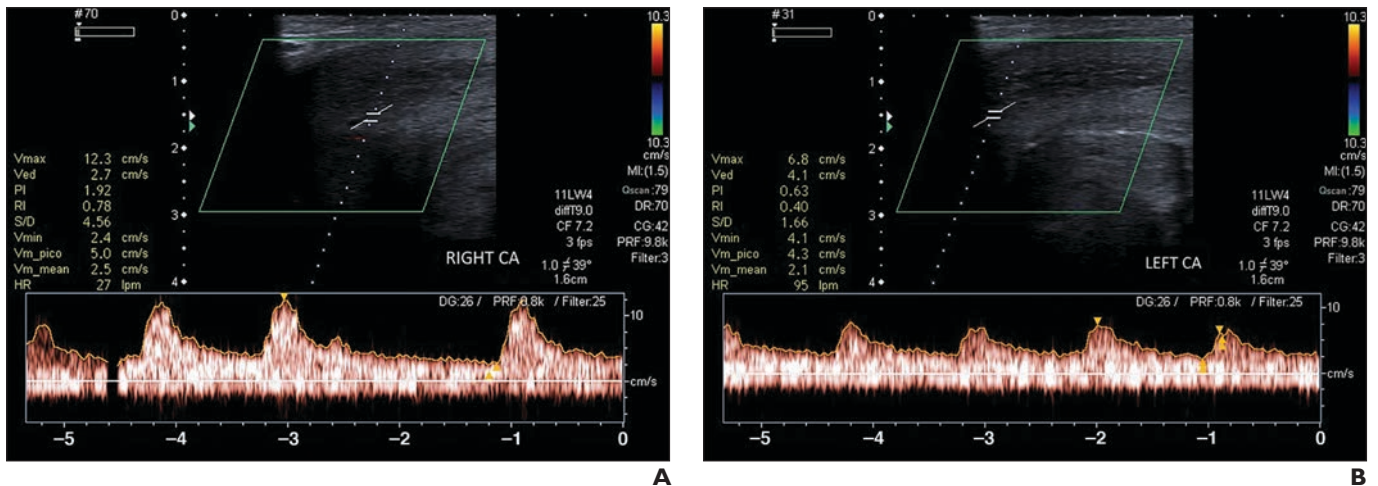
**Fig. 4**—Physiologic changes in corpus cavernosum during erection.  
**A**, Illustration of flaccid penis shows that helicine arteries and smooth muscle cells of sinusoids are normally contracted.  
**B**, Illustration shows erect penis after parasympathetic stimulation. Smooth muscle cells relax and, consequently, helicine arteries and sinusoidal smooth muscles expand, filling cavernosal spaces and decreasing venous outflow.

ERECTION PHASES	SPECTRAL DOPPLER PHASES	SPECTRAL DOPPLER WAVEFORM
FLACCID	Phase 0: monophasic with minimal diastolic flow	
FILLING	Phase 1: increased peak systolic velocity and end-diastolic velocity	
TUMESCENCE	Phase 2: decrease in diastolic flow with classic dirotic notch	
	Phase 3: decrease in diastolic flow approaching 0 cm/s	
FULL ERECTION	Phase 4: diastolic flow reversal	
RIGIDITY	Phase 5: decrease in peak systolic velocity and end-dystolic velocity	

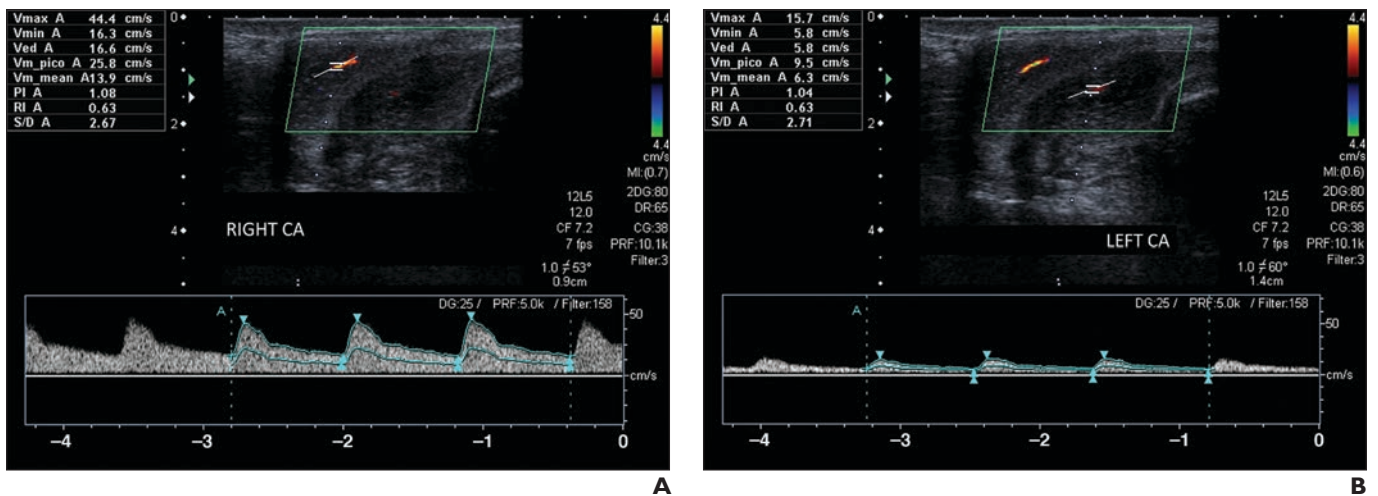
**Fig. 5**—Correlation between erection phases and spectral waveforms.



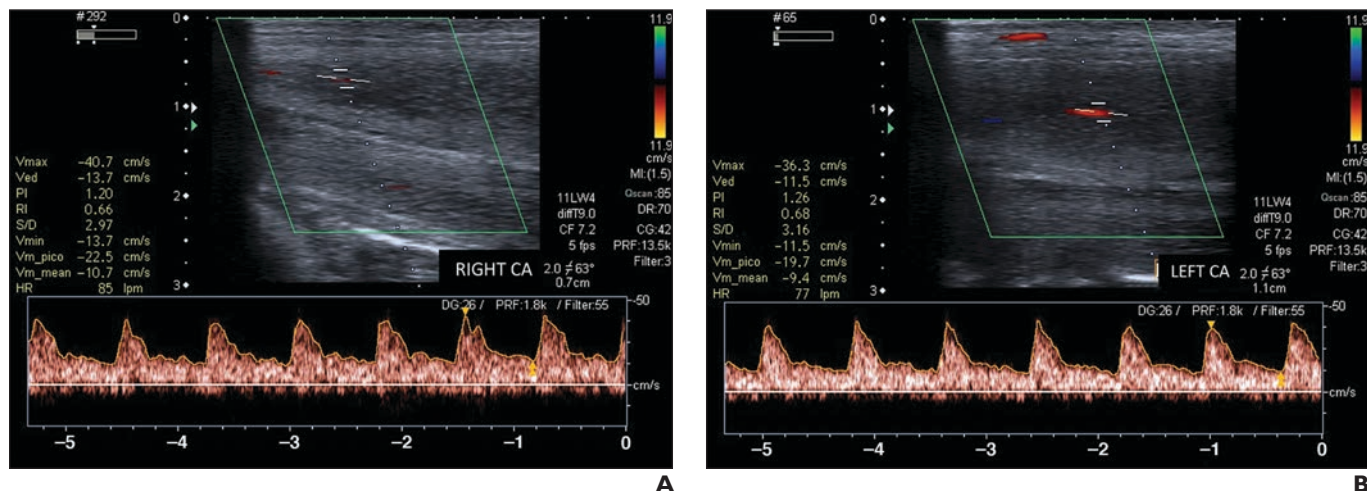
## Penile Doppler US for ED



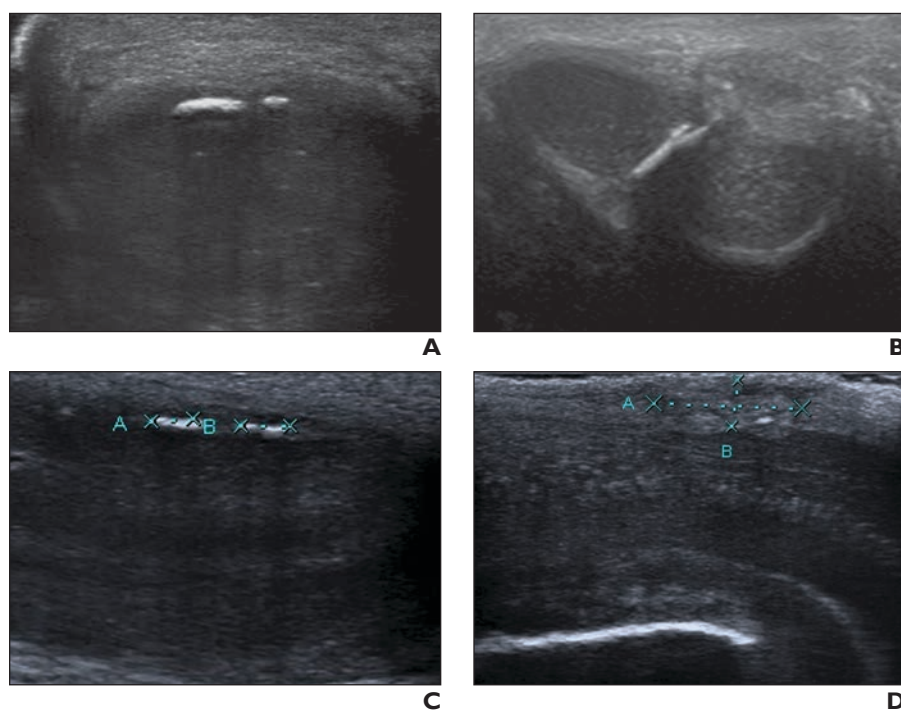
**Fig. 6**—66-year-old man with arteriogenic erectile dysfunction. Vmax = peak systolic velocity, Ved = end-diastolic velocity, PI = pulsatility index, RI = resistance index, S/D = systolic/diastolic ratio, Vmin = minimum velocity, Vm\_pico = mean peak velocity, Vm\_mean = mean velocity, HR = heart rate, CA = cavernosal artery, LW = linear transducer, diff = differential, CF = color flow, fps = frames per second, MI = mechanical index, DR = dynamic range, CG = color gain, PRF = pulse repetition frequency. **A** and **B**, Penile Doppler ultrasound images obtained after intracavernosal administration of 10 µg of prostaglandin E1 show flows with low speed in right (**A**) and left (**B**) cavernosal arteries, both with maximum peak systolic velocity of less than 25 cm/s at all intervals and low-resistance spectral waveforms, compatible with arterial insufficiency.



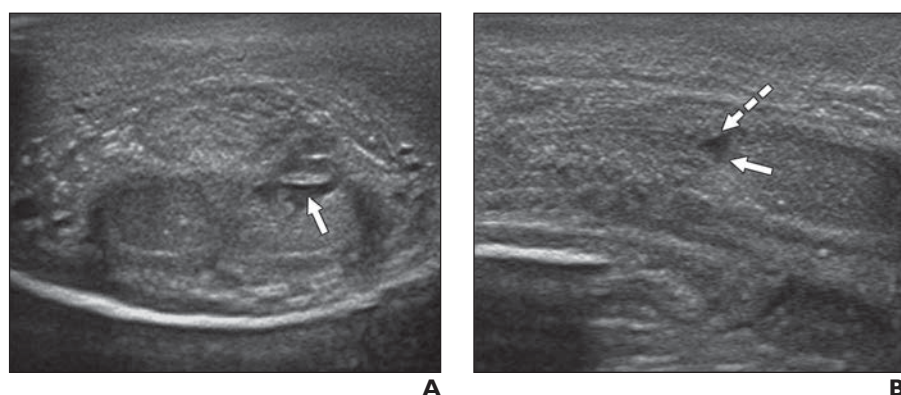
**Fig. 7**—55-year-old man with unilateral cavernosal artery insufficiency. Vmax = peak systolic velocity, Vmin = minimum velocity, Ved = end-diastolic velocity, Vm\_pico = mean peak velocity, Vm\_mean = mean velocity, PI = pulsatility index, RI = resistance index, S/D = systolic/diastolic ratio, CA = cavernosal artery, L5 = linear transducer, CF = color flow, fps = frames per second, MI = mechanical index, DG = depth gain, DR = dynamic range, CG = color gain, PRF = pulse repetition frequency. **A** and **B**, Penile Doppler ultrasound images obtained after intracavernosal injection show difference of more than 10 cm/s between peak systolic velocity of two cavernosal arteries. Maximal peak systolic velocity in right cavernosal artery (**A**) was 44.4 cm/s and that in left cavernosal artery (**B**) was 15.7 cm/s, which are findings suggestive of left arterial insufficiency.



**Fig. 8**—38-year-old man with venogenic erectile dysfunction. Vmax = peak systolic velocity, Ved = end-diastolic velocity, PI = pulsatility index, RI = resistance index, S/D = systolic/diastolic ratio, Vmin = minimum velocity, Vm\_pico = mean peak velocity, Vm\_mean = mean velocity, HR = heart rate, CA = cavernosal artery, LW = linear transducer, diff = differential, CF = color flow, fps = frames per second, MI = mechanical index, DR = dynamic range, CG = color gain, PRF = pulse repetition frequency. **A and B**, Penile Doppler ultrasound images show normal arterial inflow parameters in right (**A**) and left (**B**) cavernosal arteries (peak systolic velocity, > 30 cm/s) with end-diastolic velocity greater than 5 cm/s and RI less than 0.75, suggestive of venous incompetence.



**Fig. 9**—Peyronie disease. **A–D**, Gray-scale ultrasound images obtained in transverse (**A and B**) and longitudinal (**C and D**) planes show curvilinear hyperechoic lesions with posterior acoustic shadowing in region of tunica albuginea in dorsal aspect of both corpora cavernosa (**A and C**). Calcified plaque is seen in penile septum (**B**), and fibrous plaque appears as nodular hypoechoic lesion with focal calcifications in tunica albuginea (**D**). Dashed lines A and B in image in **C** denote calcified plaques in dorsal tunica albuginea of corpora cavernosa. Dashed lines A and B in image in **D** delimit fibrous plaque in dorsal aspect of corpora cavernosa.



**Fig. 10**—46-year-old man with penile fracture. **A and B**, Gray-scale Doppler ultrasound images obtained in transverse (**A**) and longitudinal (**B**) planes show focal defect in tunica albuginea (dashed arrow, **B**) and small collection in adjacent corpus cavernosum (solid arrow, **A and B**).