Diagnostic and Therapeutic Options for the Management of Ischemic and Nonischemic Priapism

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Priapism is defined as persistent penile erection continuing beyond, or unrelated to, sexual stimulation. Proper diagnosis encompasses at least 2 very different pathophysiologic processes. Ischemic priapism ("low flow") is a disorder of venous outflow and/or stasis. Nonischemic priapism ("high flow") is a disorder of arterial flow. We present 2 cases that review each condition. The first case highlights a 32-year-old man with a medical history of sickle cell disease who presented to the emergency department complaining of a persistent, painful erection that had continued for 18 hours. The second case describes a 24-year-old man with no significant medical history who sustained trauma to his pelvis while skateboarding. Although the initial evaluation of both types of priapism is similar, pathophysiology and resulting interventions differ for each, underscoring the importance of proper diagnosis.
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Priapism, a sustained erection of the penis, has a lengthy historical footprint. Named after the well-endowed Greek god Priapus, it was first described in the medical literature in 1616. Despite this, priapism is a disease entity whose pathophysiology and management continue to evolve. Just as delineation of ischemic from nonischemic priapism resulted in the formulation of distinct treatment algorithms, it is expected that new insights and discoveries at the cellular level will continue to alter our understanding and management in the years ahead. Here we present sample cases of priapism, review the current literature, and discuss the available diagnostic and therapeutic options.

Definition and Pathophysiology

The development of a normal erection can be explained in simple biomechanical terms. Following stimulation of the corporal smooth muscle, blood flow is increased into the corporal sinusoids as a result of this corporal smooth muscle relaxation. When a sufficient amount of this incoming blood within these sinusoids raises the intracorporeal pressure to a level that will passively compress the egressing subtunical veins, veno-occlusion occurs. As such, it appears as if the amount of veno-occlusion that occurs within the corporal sinusoids parallels the amount and degree of relaxation within the corporal smooth muscle. Therefore, to decrease veno-occlusion (and hence increase outflow of blood from the sinusoids), one must decrease the corporal smooth muscle relaxation. Because an erection itself is dependent on the balance between inflow and outflow of blood within the sinusoids, it is apparent that a persistent erection or priapism may occur as the result of such an imbalance between the inflow and/or outflow of blood within this space.

Priapism is actually defined as a persistent penile erection continuing beyond or unrelated to sexual stimulation.[1] Current scientific evidence demonstrates that when an erection lasts longer than 4 hours, it merits evaluation and possible treatment because of the potential detrimental effects of the erection on the viability and future function of the corporal tissue.[1,2]

The diagnosis of priapism encompasses at least 2 very different pathophysiologic processes. Ischemic priapism ("low flow") is primarily a disorder of venous outflow and/or stasis. This is primarily due to persistent corporal smooth muscle relaxation that continually compresses the subtunical veins, thereby preventing any outflow from the sinusoids. If the intracorporeal pressure is above mean arterial pressure, which it usually is in such a condition, the cavernosal arteries that run through the middle of each corpus cavernosum are also passively compressed such that no inflow of blood is occurring. In contrast, nonischemic priapism ("high flow") is a disorder of arterial inflow. This latter condition is due primarily to a high inflow of blood in tandem where there is very little, if any, smooth muscle relaxation. In this situation, the high inflow of blood clears as rapidly as it can from the sinusoids and the blood only "backs up" into the sinusoids if inflow is greater than outflow. It is this difference in pathophysiology between the 2 conditions that underscores the starkly different etiologies, presentation, and management of ischemic versus nonischemic priapism.

An additional entity, recurrent or stuttering priapism, is a subtype of ischemic priapism reserved for those who experience recurrent painful erections with intervening periods of detumescence. Each of the episodic erections typically lasts less than 4 hours, often building toward and culminating in a long-standing ischemic erection. The pathophysiology of recurrent priapism was long thought to be repeated episodes of persistent veno-occlusion. Recent work suggests that the cause of this persistent and intermittent disorder of veno-occlusion may be dysregulation of the phosphodiesterase type 5 (PDE5) within the nitric oxide-cyclic guanosine monophosphate (cGMP) signaling pathway in the corporal tissue, although definitive scientific proof for this relationship is still lacking.

Ischemic Priapism

Case 1

A 32-year-old black man with a medical history of sickle cell disease presented to the emergency room complaining of a persistent, painful erection for the past 18 hours. Sexual excitement prompted the erection; however, detumescence did not occur after the cessation of intercourse. The patient presented only after the pain became unbearable, and at the time of evaluation was in obvious discomfort. Examination revealed an erect penis with rigid corpora cavernosa. Palpation of the tense corpora exacerbated the pain. Of note, both the glans of the penis and the corpora spongiosum were soft. Laboratory analysis was within normal limits with the exception of an elevated reticulocyte count. Aspiration of the corpora cavernosa demonstrated dark, viscous blood. The corresponding blood gas analysis confirmed hypoxia and acidosis.

Presentation, work-up, and diagnosis. As evident in Case 1, ischemic priapism usually presents as a fully erect, usually painful erection. The coupled corpora cavernosa are firm, taut, and tender to palpation. As the disorder is related to venous congestion and/or suboptimal outflow of the corpora cavernosa, both the corpus spongiosum and glans penis are spared and will be soft, owing to their distinct venous drainage.

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idiopathic. Hematologic abnormalities resulting in priapism include sickle cell disease, protein C deficiency, lupus, polycythemia vera, and malignancy such as leukemia. Drugs known to precipitate priapism include the illicit (cocaine, marijuana, and alcohol) and the prescribed (antihypertensives, antidepressants, psychoactive medications, and anticoagulants). Men whose treatment regimen for impotence includes intracavernosal injection of vasoactive agents (alprostadil, papaverine, and others) also commonly can present with priapism.

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Initial evaluation of a patient with a presentation of any type of priapism should include a history of the priapic episode (duration of erection, degree of pain, usage of drugs or medications prior to onset), a complete medical and sexual history including prior episodes of priapism and any applied interventions, a physical examination to include not only the penis but also the perineum, laboratory testing to include a complete blood count with differential, reticulocyte count and hemoglobin electrophoresis if considering a hemoglobinopathy, and toxicology of the urine if indicated.

Although the differentiation of ischemic from nonischemic priapism may be suggested based on history and physical examination, confirmation must be obtained with cavernosal blood gas analysis and/or color duplex ultrasonography of the penis. Cavernosal blood gas can be obtained by introducing the standard blood gas needle (ie, "butterfly") into the lateral aspect of 1 of the corpora cavernosa anywhere along the penile shaft. Visual inspection of the blood gas aspirate in ischemic priapism will reveal dark, viscous blood. Subsequent laboratory analysis will demonstrate acidic, hypoxic blood (Table 1).[1] If performed, color duplex ultrasound of the perineum and penis in ischemic priapism will demonstrate little to no flow in the cavernosal arteries.

However, it is important to realize that to date there is no evidence that the oxygenation of the cavernosal tissue itself occurs from the blood within the sinusoids and, like every other tissue in the body, this process must occur via the capillaries that perfuse the corporal tissue. When an erection occurs and the intracorporeal pressure is such that it is at or above mean arterial pressure, the flow of blood through the cavernosal artery, which is located within the lumen of the cavernosa, is relatively nonexistent and as such there is also very little, if any, blood perfusing the cavernosal tissue. What actually happens to that oxygen within the erythrocytes of the blood trapped within the sinusoids of the priapic corpora still remains a mystery.

Intervention. Once the diagnosis of ischemic priapism has been made, emergent intervention is required. Without detumescence and the restoration of arterial inflow to the cavernosal tissue, cavernosal hypoxia (via the capillaries) will be ongoing, ultimately resulting in interstitial edema, corporal smooth muscle fibrosis, and clinical erectile dysfunction.[3] Even a single prolonged erection can cause such pathologic alterations at both the structural and molecular level in the corpora cavernosa. Histopathologically, edema of the cavernous tissue and its trabecular framework at 4 hours is followed by a denuding of the sinusoidal endothelium at 24 hours, followed by smooth muscle cell necrosis and transformation into fibroblastic-like cells at 48 hours.[4] The changes set the stage for the progressive fibrosis and erectile dysfunction of the penis that are the hallmarks of priapism's sequelae.

Although intervention after 72 hours of an erectile state may relieve the unwanted erection and pain, little benefit is thought to remain in terms of potency preservation.[2] In fact, the combined work of a number of investigators has demonstrated that the hypoxia and acidosis seen in the priapic penis at even 4 hours can result in irreversible dysfunction of the cavernosal smooth muscle, and therefore the erectile apparatus.[5-11]

The predominant intervention in ischemic priapism is to achieve smooth muscle constriction or detumescence via corpora cavernosal aspiration/irrigation with concomitant usage of a sympathomimetic agent. Aspiration and irrigation of the cavernosa with

Table 1
Laboratory Analysis for Blood Gas Values

Source	pH	pO_2 (mm Hg)	pCO_2 (mm Hg)
Normal mixed venous blood	7.35	40	50
Aspirate in ischemic priapism	< 7.25	< 30	> 60
Aspirate in non ischemic priapism	~ 7.40	> 90	< 40

normal saline promotes evacuation of the viscous, hypoxic blood. Sympathomimetics promote detumescence by contracting the smooth muscle, thereby opening the emissary veins and increasing venous outflow.[12] Aspiration alone without the use of sympathomimetics, with or without corporal irrigation, resolved the erection in up to 36% of patients in summary data compiled by the American Urological Association (AUA) Guideline Panel.[1] The addition of a sympathomimetic injection increased efficacy to 81%, whereas injection of a sympathomimetic alone without aspiration/irrigation yielded a 58% resolution rate.[1]

A number of sympathomimetic agents have been studied to determine efficacy. These agents include epinephrine, norepinephrine, and phenylephrine. Although all of these agents carry the risk for systemic effects such as hypertension, bradycardia, tachycardia, palpitations, and cardiac arrhythmias, phenylephrine is the preferred agent for use in the treatment of priapism given its selectivity for α-adrenergic receptors and its limited cardiovascular effects. Regardless of which agent is used, however, noninvasive monitoring of patients during and after administration is recommended.

Although specific regimens for corporal aspiration and irrigation differ, one commonly used at our institution to treat an ischemic priapism is as follows:

After a penile block is applied, a dilute solution of phenylephrine is prepared by adding 1 mL of phenylephrine (10 mg/mL) to 99 mL of normal saline for a final concentration of 100 μg/mL. A 19-gauge butterfly needle is then inserted into the lateral aspect of one of the corpora cavernosa, and 1 or 2 mL of solution (ie, 100-200 μg of phenylephrine) is injected intracavernosally. If detumescence is not apparent within 2 minutes, an additional 1 or 2 mL of the phenylephrine solution is injected. This is repeated at 2-minute intervals until detumescence is achieved, with no more than 10 mL of total solution injected (ie, 1000 μg of phenylephrine).

If detumescence does not occur with phenylephrine, the cavernosa should be irrigated with normal saline, with or without the addition of heparin. If there is difficulty aspirating the irrigate, a second 19-gauge butterfly needle can be placed on the opposite side of the shaft away from the first butterfly needle. To facilitate involvement of the entire cavernosa, 1 needle should be placed proximally with the contralateral needle placed distally.

With regard to priapism that is secondary to an underlying systemic disorder, such as sickle cell disease and other hematologic malignancies, intracavernosal intervention should proceed concurrently with systemic treatment. For example, for the patient in Case 1, data suggest that systemic measures alone (ie, hydration, oxygenation, blood exchange transfusions, analgesia, and alkalinization) have reduced efficacy when compared with concomitant systemic and cavernosal therapies.[1]

Surgical Interventions

Distal shunts. In the event that aspiration/irrigation with the use of a sympathomimetic agent fails, additional surgical intervention may be required. The next step involves the creation of a shunt distally between the corpora cavernosa and the glans of the penis. The distinct venous drainage of the corpora spongiosum (and its distal continuation, the glans penis) and the corpora cavernosa allows the congested cavernosa to drain.

A number of different types of shunts have been described, including the Ebbehøj, Winter's, and Al-Ghorab. The Ebbehøj shunt involves insertion of a scalpel through the glans penis lateral to the meatus into the underlying distal end of one or both of the rigid corpora cavernosa.[13] The Winter's shunt involves the same maneuver, however, with a large biopsy needle substituted for the scalpel.[14] Finally, the Al-Ghorab shunt involves a transverse incision into the glans between the corona and superior aspect of the urethral meatus, with the incision carried down to excise the tunica albuginea off the tip of the corpora cavernosa.[15] A summary of the efficacy and reported postintervention impotence as compiled by the AUA Guideline Panel is reported in Table 2.[1] Although the Al-Ghorab is the most effective of the distal cavernoglanular shunts, it is also the most invasive and has a high rate of reported impotence, leading some authors to suggest that it should only be performed after an Ebbehøj or Winter's shunt has been attempted. Whereas the reported impotence

Table 2
Reported Summary of Efficacy of Distal Cavernoglanular Shunts

Type of Shunt	Number of Publications	Number of Patients	Resolution	Impotence
Ebbehøj	15	52	73%	14%
Winter's	79	235	66%	25%
Al-Ghorab	11	23	74%	25%

Table 3
Reported Summary of Efficacy of Proximal Shunts

Type of Shunt	Number of Publications	Number of Patients	Resolution	Impotence
Cavernospongious shunt	69	142	77%	49%
Cavernosaphenous shunt	83	160	76%	52%

is most attributable to the pathologic consequences of priapism itself, impotence secondary to failure of spontaneous closure of a distal shunt can be successfully corrected by formal shunt closure.[16]

Although a distal surgical shunt should not be used as a first-line intervention, note that in patients whose priapism has exceeded 48 hours, aspiration/irrigation with the use of a sympathomimetic agent is less likely to result in resolution. This is based on the nonresponsiveness of the cavernous smooth muscle to sympathomimetics secondary to the duration of hypoxia and acidosis. Although the consensus is that nonsurgical measures still warrant an attempt, it may be necessary to proceed fairly quickly to formal surgical intervention.

Proximal shunts. In some instances of ischemic priapism, particularly those of duration longer than 72 hours, the ongoing hypoxia and acidosis may have resulted in enough edema and tissue death within the cavernosa that creation of a distal shunt fails to resolve the priapism. In these instances, a more proximal shunt may be required. Examples of proximal shunts include a spongiocavernosus (ie, Quackels or Sacher) and a cavernosaphenous (ie, Grayhack) shunt. The former involves creation of an anastomosis between each proximal cavernosum to the corpus spongiosum via a perineal incision.[17] The Grayhack shunt, rarely used today because of the ease of the spongiocavernosus shunt, involves anastomosing the saphenous vein to the ipsilateral proximal cavernosal body.[18] A summary of the efficacy and reported postintervention impotence as compiled by the AUA guideline panel on priapism is reported in Table 3.[1]

Although proximal shunts have reasonable efficacy for resolution of the priapism, these interventions are not only more time intensive and surgically complex, but raise the potential for significant complications. Anastomosis of the cavernosa to the spongiosum has resulted in reports of urethral fistulas and cavernositis.[19] Likewise, draining the cavernosa via the saphenous vein(s) has resulted in pulmonary embolism.[20] Recently, tunneling of the cavernosa from distal to proximal has been suggested as a method by which to increase the efficacy of a distal shunt in cases of severe edema and necrosis throughout the corporal body.[21] These authors describe a modification of the Ebbehøj scalpel incision, the so-called T-shunt, which is 2 incisions per corpora cavernosa. A scalpel is inserted through the glans penis, with 1 horizontal and 1 vertical incision made at the distal end of each cavernosa in the shape of a T. This is followed by insertion of a 20 to 24 Fr straight urethral sound or dilator from the stab incision through the cavernosum all the way proximally to the crura. In this way it is presumed that flow from proximal to distal in the cavernosa is facilitated, increasing the likelihood that the entirety of corpora cavernosa may be drained via the distal shunt. The ultimate goal of this or similar measures is to avoid formal creation of a proximal shunt.

Prevention

Given the morbidity of ischemic priapism as it relates to fibrosis and erectile dysfunction, much focus has been placed on preventing future episodes. This is particularly true as it pertains to those with recurrent (ie, stuttering) priapism. A number of systemic therapies have been proposed, including oral use of terbutaline, digoxin, baclofen, and hormonal agents. To date, evidence supporting the use of these agents is limited to case reports or small case series. Terbutaline, a β-adrenergic agonist, is the exception. Three randomized trials have evaluated its efficacy in achieving detumescence in men presenting with pharmacologically induced erections. Although its mechanism of action is not clear, terbutaline did demonstrate increased success versus placebo in 2 of the 3 trials, with detumescence rates ranging from 36% to 42%.[22] Whether these results would translate to those with recurrent or stuttering priapism remains to be seen.

Estrogens, gonadotropin-releasing hormone agonists (GnRH), and antiandrogens are hormonal agents that have been used in the treatment of recurrent priapism, particularly in those patients with sickle cell disease. In a randomized, controlled trial diethylstilbestrol (DES) eliminated priapism episodes in 9 patients initially randomized to DES (4 patients) versus placebo (5 patients, with crossover).[23] Dosing varied per patient, ranging from 5.0 mg orally daily to 2.5 mg orally per week. Priapric attacks recurred after cessation of DES in 5 of the 9 patients (55%). Given the

increased risk of thromboembolic events with long-term estrogen therapy evidenced in the obstetrics-gynecology literature (including coronary artery disease and cerebrovascular accidents), only short-term use should be considered. With regard to GnRH agonists, 2 case reports describe the use of leuprolide acetate.[24,25] Monthly dosing of leuprolide acetate (7.5 mg intramuscular [IM]) resulted in reduced episodes of priapism. One of the 2 patients was treated for 4 months without recurrence on cessation; the other recurred after 1 year of therapy and elected to continue with injections. Likewise, the antiandrogen bicalutamide, an inhibitor of the androgen receptor, has been reported to reduce priapism episodes in those with recurrent priapism and sickle cell anemia.[26] Initial dosing of bicalutamide was 50 mg orally daily, tapering to 1 tablet every other day depending on frequency of priapism episodes and development of side effects (breast tenderness or swelling). Relative to GnRH analogs, libido and the ability to obtain a normal erection appear better preserved with the use of this antiandrogen.

Several investigators, including Burnett and colleagues, have proposed a new therapy for recurrent priapism—PDE5 inhibition. Although counterintuitive, preliminary data from his group support the use of chronic and daily PDE5 inhibitors in reducing priapism recurrences.[27] How could a medication designed to promote erections assist in preventing its prescribed effect? As described in their dysregulatory hypothesis, the structural and molecular changes that occur within the ischemic cavernosa may cause alterations in endothelial nitric oxide/cGMP signaling pathway.[28] In particular, decreased endothelial nitric oxide bioavailability, via lower steady state levels of cGMP, leads to down-regulation of the set point of PDE5 function. As a result, neuronal stimulation of the penis leads to levels of PDE5 that are insufficient to degrade cGMP, resulting in a prolonged erection. Continuous, long-term PDE5 inhibition thereby affects recurrent priapism by re-establishing PDE5 regulatory control. These investigators have reported the success of this chronic PDE5 treatment in 7 patients.[27]

In patients for whom oral therapies of any kind are not effective in reducing the priapism episodes, self-administration of sympathomimetics intracorporeally at the beginning of a priapic episode is a treatment option. Although not preventative, it does decrease the time to, and associated logistics of, seeking medical care with each episode. For many patients, this self-treatment with these vasoconstrictors is similar to what impotent patients use when they use intracorporeal vasoactive injections for the treatment of their erectile dysfunction.

In patients for whom oral therapies of any kind are not effective in reducing the priapism episodes, self-administration of sympathomimetics intracorporeally at the beginning of a priapic episode is a treatment option.

Nonischemic Priapism

Case 2

A 24-year-old Asian man without significant past medical history sustained a trauma to his pelvis while skateboarding. Two weeks later he presented to the emergency room complaining of a persistent erection over the past 24 hours. The erection was not painful. Examination of the penis revealed a partially rigid phallus that was nontender to palpation. The corpora cavernosa were partially rigid. The corpus spongiosum and glans penis were soft. Examination of the perineum revealed bruising consistent with the patient's history of trauma. Aspiration of the cavernosum demonstrated bright red blood and a normal arterial blood gas profile. Color Doppler ultrasound demonstrated flow within the cavernosal arteries with an area of turbulent flow within the right cavernosum consistent with a cavernosal artery to sinusoid fistula.

Presentation, work-up, and diagnosis. As evident in Case 2, nonischemic or high-flow priapism typically presents as a partially erect, nontender erection. The disorder is a result of up-regulated arterial inflow, often secondary to an arterial fistula within the corpus cavernosum. The most common etiology is trauma to the penis or perineum; however, the duration of time from trauma to presentation can be quite variable. We have had patients with recurrent high-flow priapism who have refused intervention and continue years later to still have normal on-demand erectile function. The initial work-up is the same as presented previously for ischemic priapism, and once again, the diagnosis of ischemic priapism should not be rendered prior to obtaining a cavernosal blood gas and/or duplex ultrasound. Cavernosal aspiration will demonstrate bright red blood with subsequent laboratory analysis confirming an arterial source (Table 1). Color duplex ultrasound of the penis and perineum (best performed in dorsal lithotomy position) will demonstrate normal to increased flow within the cavernosal arteries. An arterial fistula or pseudoaneurysm may also be demonstrated.

Intervention. Nonischemic priapism is not a surgical emergency. As the erection is secondary to arterial inflow and there is no restriction in the

outflow of blood, the acidosis and hypoxia seen in ischemic priapism are absent. Patients are thus at low risk for permanent damage. Observation will result in spontaneous resolution in approximately 60% of patients, and thus should be the initial management.[1] Spontaneous resolution is even more likely in those without an underlying anatomic abnormality such as a fistula or pseudoaneurysm.

For those patients with persistent nonischemic priapism desiring intervention, selective arterial embolization has become the primary treatment modality. A number of absorbable materials (ie, autologous blood clot and gelatin) and permanent materials (ie, coils, polyvinyl alcohol, and acrylic glue) have been described in the literature. Historically, although rates of resolution were similar between absorbable and permanent materials (74% and 78%, respectively), associated erectile dysfunction was significantly higher in those treated with permanent materials (5% vs 39%, respectively).[1] With the advent of more selective embolization techniques, reported erectile dysfunction rates have begun to decrease. Additionally, permanent materials such as microcoils may be a useful adjunct in those situations where embolization with an absorbable material does not have a durable effect,[29] but in our opinion these permanent products should only be used as a last resort and where the patient has been given informed consent as to the probability of permanent erectile dysfunction.

Note that, other than the initial aspirate for blood gas analysis, interventions such as aspiration/irrigation, injection of sympathomimetics, and the creation of shunts are not warranted in nonischemic priapism. Occasionally, however, a high-flow priapism will be so severe that the inflow of blood is so much greater than the inability of the corpora to drain, that some patients may present with a picture of a rigid penis. In this situation, which is rare, aspiration of the corpora may be instituted. We have also seen a case where we have converted an ischemic priapism to a high-flow priapism presumably due to an injury to a cavernosal artery secondary to the irrigating needle used during the treatment of the ischemic priapism.

Other than the initial aspirate for blood gas analysis, interventions such as aspiration/irrigation, injection of sympathomimetics, and the creation of shunts are not warranted in nonischemic priapism.

Conclusions

Although the initial evaluation of both types of priapism is similar, the pathophysiology and intervention differ for each, thus necessitating proper diagnosis. An important consideration when distinguishing between the 2 types of priapism is the sequelae of fibrosis and erectile dysfunction more often associated with ischemic priapism. As knowledge of the molecular and cellular processes involved with priapism evolves, it is expected that the therapeutic options will similarly be refined to not only reduce the immediate clinical condition but also to prevent or reduce the incipient fibrosis and smooth muscle dysfunction that may accompany the disorder. ■

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Main Points

- Priapism is defined as a persistent penile erection continuing beyond or unrelated to sexual stimulation. When an erection lasts longer than 4 hours, it is considered priapism and warrants evaluation and possible treatment. Although the initial evaluation of both types of priapism is similar, the pathophysiology and intervention differ for each, thus necessitating proper diagnosis.
- Ischemic priapism ("low flow") is primarily a disorder of the venous outflow and/or stasis. Nonischemic ("high flow") is a disorder of arterial inflow. An important consideration when distinguishing between the 2 types of priapism is the sequelae of fibrosis and erectile dysfunction more often associated with ischemic priapism.
- For diagnosis of ischemic priapism, emergent intervention is required with the predominant course to achieve smooth muscle constriction via corpora cavernosal aspiration/irrigation with concomitant use of a sympathomimetic agent. In the event that aspiration fails, surgical intervention may be required utilizing distal or proximal shunts.
- Nonischemic priapism is not a surgical emergency. Observation will result in spontaneous resolution in 60% of patients; for patients who require intervention, selective arterial embolization is the primary treatment modality.

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