Management of Priapism
2021 Update

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KEYWORDS

• Priapism • Management • Shunt • Guidelines • Penile prosthesis

KEY POINTS

- Priapism management, Intracavernosal shunting, Penile prosthesis
- Ischemic priapism, Non ischemic priapism, Stuttering priapism, Priapism management, Shunting

INTRODUCTION

Priapism is defined as a penile erection that lasts longer than 4 hours after completion of sexual activity or that is unrelated to sexual activity.[1] Priapism is a disorder of the hemodynamic systems that control erection and detumescence of the penis. Priapism is relatively rare, with an incidence of 1.5 per 100,000 person-years.[2] Contemporary management of priapism is directed toward 3 goals: resolution of the acute event, preservation of the erectile function, and preventing recurrence.[3]

There are 2 pathophysiologic variants that are essential to distinguish in the emergent setting: ischemic (low-flow) and nonischemic (high-flow).[4] Each variant has specific etiologies, diagnostic criteria, and management. Ischemic priapism may be conceptualized as a compartment syndrome of the penis. Emergent categorization of ischemic versus nonischemic versus recurrent ischemic priapism (RIP) (stuttering priapism) is essential because ischemic priapism may result in penile fibrosis and erectile dysfunction (ED).[1] Although the priapic erection provokes anxiety in both patient and provider, nonischemic priapism does not mandate emergent intervention.[5]

Patients with suspected priapism should be evaluated by an emergency medicine physician and ideally a urologist. A complete medical and sexual history should be documented. Points to discuss with the patient include the erection onset, duration, presence or absence of penile pain, antecedent genital or perineal trauma (straddle injury), prior episodes of prolonged erection, and general erectile quality.[4] Medical history should be directed at risk factors for priapism, especially prescribed of intracavernosal injectables, hematologic diseases (eg, sickle cell disease [SCD], glucose-5-phosphate dehydrogenase deficiency, hereditary spherocytosis, and leukemia), medications (eg, psychotropic medications, especially trazodone), use of oral phosphodiesterase type 5 (PDE5) inhibitors, over-the-counter medications/supplements for ED, and recreational drugs (cocaine).[4,6] A focused physical examination should be performed. The physical examination is important to determine the degree of penile rigidity and any evidence of perineal trauma.[1] Patients with ischemic priapism present with rigid corporal bodies, the tips of which often are palpable through the typically soft glans penis; the degree of tenderness is a function of the duration of ischemic priapism. In nonischemic priapism, the corporal bodies are partially erect and generally not tender to palpation.[4]

In both ischemic priapism and nonischemic priapism, the precipitating event may be a nocturnal erection that fails to subside. The physiology of ischemic priapism begins with a normal erection; the pathology is related to time-dependent changes in corporal oxygenation, hypercarbia, and acidosis as the erection persists beyond 4 hours to 10 hours.[4]

Nonischemic priapism almost universally is associated with prior pelvic or penile trauma. Straddle injuries may cause disruption of the

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cavernous artery or cavernous sinusoids resulting in arteriolar-sinusoidal fistula. The onset of nonischemic priapism may not be evident for hours to days following the initial trauma. A partial persistent erection typically becomes evident following a nocturnal erection some days to weeks later. Nonischemic priapism is not a compartment syndrome and may not be associated with discomfort other than at the site of a prior injury.[7]

This commentary discusses the current literature and practices surrounding the diagnosis and management of ischemic and nonischemic priapism. Few randomized controlled trials of priapism management exist due to the emergent nature of the condition. There are many published society guidelines, surgical case reports, and basic science investigations. A general understanding of this pathophysiology and penile hemodynamics allows the urologist to safely and effectively triage patients to the appropriate medical and surgical interventions.

ISCHEMIC PRIAPISM ETIOLOGY AND PATHOPHYSIOLOGY

Ischemic priapism is the most common subtype of priapism and accounts for more than 95% of all reported priapism episodes.[7,8] Ischemic priapism is a medical emergency. Failure to achieve prompt and complete detumescence typically results in cavernous thrombosis and smooth muscle necrosis. The natural history of ischemic priapism is days to weeks of penile erection and pain, which progresses to fibrosis and ED.[9] Extraneous factors, such as busy emergency departments, delayed transportation, patient embarrassment, and intoxication, can delay diagnosis. A study of US emergency room visits between 2006 and 2009 identified 32,462 diagnostic codes for priapism (a national incidence of 5.4 per 100,000 men per year). Priapism incidence was 30% higher in summer months, and 13% of visits resulted in hospitalizations.[10]

Ischemic priapism is defined by rigid corpora, minimal or no cavernous nonischemic inflow, and complete occlusion of venous outflow. The result of this hematologic stasis is a progressively acidotic environment within the corpora, secondary to hypoxia and hypercarbia. Aspirated blood from the corpora is dark red due to the absence of bound oxygen. The progressive acidity within the closed compartment activates corporal nociceptors; hence, pain is a key factor in differentiating ischemic priapism from nonischemic priapism.[11]

The hallmark physical finding of ischemic priapism is fully erect, rigid, and tender corporal bodies. The glans and corpus spongiosum may be flaccid or partially engorged but are not rigid. History and physical examination alone may be sufficient for the experienced provider to diagnose ischemic priapism, but corporal aspiration and penile blood gas are confirmatory and warranted when possible. Inquiries into previous episodes of priapism and interventions for such are important and may help diagnose and direct evaluation and treatment.

Intracavernous injection (ICI) therapy with vasodilator agents is a common treatment of men with ED refractory to oral PDE5 inhibitors. The incidence of prolonged erection or ischemic priapism following diagnostic penile injection (prostaglandin E1, papaverine, papaverine/phentolamine, or combination of all 3, Trimix) in the office is reported to vary between 1.3% and 5.3%.[12] The likelihood of prolonged erection or priapism following diagnostic alprostadil injection varies with the etiology of ED and may be considerably higher in younger men, and in patients with neurogenic or psychogenic ED.[12] Urologists should be aware that recreational use of ICIs has been reported as the primary etiology of priapism in specific community settings. A retrospective of cases from a major Los Angeles medical center identified 169 events of ischemic priapism over 8 years; 49% of events were related to recreational use of ICIs; 50% of patients using recreational ICIs also were human immunodeficiency virus positive. Only 25% of patients had been prescribed ICI therapy; oral PDE5 inhibitors were reported in 5% of cases; SCD in 4%; and trazodone use in 5%.[7,13]

Multiple classes of medications are well known to cause priapism by themselves or in combination with erection potentiating medications. Although the package inserts for all PDE5 inhibitors commercially available in the United States (ie, avanafil, sildenafil, tadalafil, and vardenafil) warn of prolonged erections, ischemic priapism following PDE5 inhibitor therapy is rare in the average patient with ED. A query of the FDA Adverse Event Reporting System (FAERS) Public Dashboard found 411 cases of PDE5 inhibitor–related priapism since 1998. Drug-induced priapism was 2-times to 2.6-times more likely with antipsychotics or trazodone than PDE5 inhibitors .[14,15]

Another important etiology for ischemic priapism is hematological abnormalities. Blood dyscrasias have been proposed to cause vascular stasis, leading to decreased venous outflow, which prevents detumescence.[7] The blood dyscrasia most often associated with ischemic priapism is SCD.[16] SCD is the result of a mutation in the gene coding for hemoglobin. Boys and men who are homozygous for the hemoglobin S gene are likely to manifest SCD.

The incidence of priapism in patients with SCD under 18 is 3.6% versus 42% in patients over the age of 18 years. Other hematologic pathologies (eg leukemia) have been associated with ischemic priapism in up to 5% of cases.[17]

The pathophysiology of ischemic priapism from SCD is complex. Historically ischemic priapism was attributed to sludging of sickled red cells in the penile sinusoids.[7] Although the primary event in the sickling of red cells is polymerization of abnormal hemoglobin, there are downstream pathophysiologic events related to chronic hemolysis and oxidant damage that may be germane to the pathogenesis of ischemic priapism in SCD. It has been proposed that nitric oxide synthase deficiency (down-regulation cyclic guanosine monophosphate [cGMP] protein kinase 1 and Rho-kinase pathways) in SCD patients leads to an impaired negative feedback loop in the cGMP and PDE5 pathway. The results are erections that are unchecked. This hypothesis has been demonstrated in a rodent model: cGMP is left in an uninhibited state due to a lack of enough PDE5 to allow cGMP breakdown. Oxidative stress, also known to occur in sickle cell anemia, further alters the balance of the nitric oxide/cGMP (NO/cGMP) pathway. These pathways have been proposed as therapeutic targets for stuttering priapism (**Table 1**).[18]

The urologist and emergency provider should be familiar with SCD-associated RIP and with general recommendations for oral or intravenous hydration and pain control in SCD patients. To screen for SCD in the patient presenting with ischemic priapism, current American Urological Association (AUA) guidelines recommend complete blood cell count, reticulocyte count, and hemoglobin electrophoresis.[1] Consultation with a hematologist is recommended before transfusing for anemia or if other complications of vaso-occlusive crisis (VOC) are suspected. Priapism in SCD patients is not necessarily accompanied by acute VOC. VOC generally is thought to result from ischemia induced by vaso-occlusion in the bones and bone marrow. VOC requires the attention of a hematologist because associated complications include fever/infection, acute kidney injury, acute anemia, hepatobiliary complications, acute chest syndrome, and stroke.[7]

Advanced pelvic malignancies may lead to priapism; this commonly is referred to as "malignant priapism." The pathophysiology of malignant priapism may be direct infiltration of tumor implants or occlusion of venous outflow. Magnetic resonance imaging may be most helpful in distinguishing a patient with penile induration due to malignant infiltration of corporal bodies. A recent study of 412 men with idiopathic ischemic priapism found a 3.5% prevalence of a malignant cause.[9,19]

Other etiologies that should be noted but are not discussed further in this article include thalassemias, lymphomas, amyloidosis, scorpion stings, spider bites, spinal cord injury, and medications for attention-deficit/hyperactivity.

STUTTERING PRIAPISM ETIOLOGY AND PATHOPHYSIOLOGY

Stuttering priapism, or RIP, refers to multiple erection episodes, generally sleep-related, that often are painful, last up to 4 hours, and typically affect adolescents and adult men with SCD.[20] Patients often detail a history of stuttering, awakening with prolonged erections with increasing frequency leading up to an episode of true ischemic priapism.[21]

Sleep-related erections are a natural phenomenon in hormonally intact men. Nocturnal penile

Table 1
New molecular targets for treatment of stuttering priapism

Study	Molecular Target	Therapeutic Agent
Burnett and Pierorazio 2011[47]	Nitric oxide, cGMP system	PDE5 inhibitors
Musicki and Burnett 2020[48]	Oxidative stress	Apocynin (NADPH oxidase inhibitors)
Mi et al 2008[49]	Adenosine	ADA-PEG (supplemental enzyme therapy)
Kanika et al 2009[50]	Opiorphins	ODC inhibitor
Musicki, Karakus et al 2018[51]; Morrison, Madden et al 2015[52]; Rachid-Filho, Cavalcanti et al 2009[53]	Androgens	Testosterone replacement therapy; Finasteride
Olujohungbe and Burnett 2013[54]	Adrenergic system	Ephedrine; etilefrine

Abbreviations: ADA-PEG, polyethylene-glycol–modified adenosine deaminase; ODC, ornithine decarboxylase.

tumescence coincides with rapid eye movement sleep; 3 to 5 erections can occur in an 8-hour sleep cycle with erection of varying degrees (tumescence) lasting from 10 minutes to 20 minutes. Unfortunately for men with RIP, the erections can last up to 3 hours to 4 hours and may persist on awakening requiring interventions. RIP needs to be distinguished clinically from the psychological phenomenon, known as sleep-related painful erections (SRPEs). SRPEs are classified as rapid-eye-movement (REM) parasomnias; patients complain of awakening recurrently with painful erections. Awakening from SRPE results in natural detumescence unlike RIP. Nocturnal penile tumescence and rigidity testing in SRPE does not show prolonged erections, and these men have no history of priapism and erectile dysfunction. SRPEs typically are treated with baclofen and other neuroleptic agents.[7]

Treatment options for RIP aim to inhibit specific pathways and factors controlling sleep-related erections. Medications that have been proposed to this effect include ketoconazole, finasteride, antiandrogens (flutamide, bicalutamide, and chlormadinone), gonadotropin-releasing hormone agonists, and diethylstilbesterol. These agents have in common interruption of androgen synthesis and or blockage of androgen receptors resulting in inhibition of nocturnal erections.[22,23]

In vitro studies of antiandrogenic agents also show direct inhibitory effects at the molecular level. They inhibit corporal smooth muscle relaxation by altering cellular calcium transport. The potential side effects of GnRH agonists, antiandrogens, and DES are profound and include gynecomastia, hot flushes, loss of libido, osteoporosis, increased body fat, and insulin resistance. Additionally, DES is associated with risk of deep vein thrombosis.[24]

Ketoconazole is an orally bioavailable antifungal medication. Ketoconazole, at dosages of 200 mg, causes total and free testosterone to fall 60% within hours of dosing. To prevent adrenal insufficiency, patients should be coadministered a corticosteroid. Oral ketoconazole has been shown efficacious in suppressing RIP, with initial dosing of 200 mg, 3 times daily, plus 5 mg of prednisone, for 2 weeks, followed by 200 mg, nightly, without prednisone.[25]

Finasteride and dutasteride are 5α-reductase inhibitors, which block conversion of testosterone to dihydrotestosterone. Finasteride, in dosages from 1 mg to 5 mg daily, and dutasteride, at 0.5 mg daily, both have been shown to reduce frequency and duration of RIP episodes.[26,27]

Direct ICIs of α-adrenergic agents (phenylephrine, metaraminol, and etilefrine) reverse prolonged erections and can terminate an episode of RIP. Self-administration of phenylephrine 100 μg to 200 μg is an effective tool for reliable patients with RIP. Patients started self-administered therapy should be counseled about sympathomimetic side effects including hypertension, headache, and bradycardia.[28,29]

Oral nightly α-adrenergic drugs (pseudoephedrine and etilefrine) potentiate sympathetic pathways causing vasoconstriction of erectile tissues and have been investigated in nightly dosing.[29]

Oral terbutaline has been touted as a treatment of ischemic priapism.[30] Evidence for efficacy of oral terbutaline in management of priapism has not met efficacy standards for priapism according to both the AUA and the European Association of Urology guidelines. This treatment has few side effects but has no significant efficacy in reversing ischemic priapism or preventing RIP and may delay administration of more effective therapy.[1,5]

Finally, daily oral PDE5 inhibitor dosing in SCD patients with RIP has been investigated in a placebo-controlled study. PDE5 inhibitors dosing did not show increased efficacy over placebo during the 8-week trial but sildenafil dosing, at 50 mg, did show a reduction in RIP in the 8-week open-label phase.

Each of those therapies has significant side effects. Antiandrogens suppress testosterone and may have an impact on sexual drive and spermatogenesis. Antiandrogens are contraindicated in prepubescent children and adolescents who have not experienced closure of the epiphyseal plates because this stunts growth. Oral ketoconazole can interrupt androgen synthesis effectively but also interrupts steroidogenesis and combination therapy with prednisone is required.[25] Oral baclofen has shown some efficacy in patients with spinal cord injury or men with SRPE but has little or no impact in neurologically intact men with RIP.[1]

NONISCHEMIC PRIAPISM ETIOLOGY AND PATHOPHYSIOLOGY

Nonischemic priapism, also called high-flow priapism, is a much less common than ischemic priapism. This condition is the result of unregulated cavernous nonischemic inflow following formation of an arteriolar-sinusoidal fistula.[4] In contrast to ischemic priapism, there is appropriate flow of well oxygenated blood through the system, which maintains appropriate pH and oxygenation of the cavernous environment. Unregulated high corporal blood (arteriolar–sinusoidal fistula) does cause partial persistent erection. The etiology of nonischemic priapism most often is blunt perineal

trauma, most often straddle injury related to bicycles, falls, or sports trauma. There are reports of nonischemic priapism after correction of ischemic priapism via T-shunt and corporal snake procedure.[31] Direct penetrating injury to the cavernous artery (most commonly needle laceration during treatment of ischemic priapism) also can cause a cavernous fistula. Sustained erection may begin within 24 hours of trauma but typically the sinusoid fistula takes several weeks to mature.[4]

EVALUATION OF PRIAPISM

Careful history and diligent physical examination, as described previously, are the preliminary keys to differentiating ischemic versus nonischemic priapism. A priapism checklist has been proposed to help urologists and emergency room providers (**Table 2**).[4] Laboratory testing with complete blood cell count (blood cell differential and platelet count) and coagulation panel is advised to evaluate for hematologic abnormalities. A penile blood gas effectively differentiates nonischemic from ischemic priapism.[5] Use of a drug screening panel is indicated if there is concern for psychotropic or recreational drug use. Alcohol intoxication and illicit drug use have been shown to be causative in up to 21% of cases of ischemic piapism.[4] If sickle cell anemia is suspected as the underlying cause, additional laboratory studies should be considered: hemoglobin electrophoresis, reticulocyte count, and lactate dehydrogenase. The AUA guidelines do not recommend directing priapism treatment solely at the underlying cause. Treatment of a sickle cell crisis without concomitant emergent management of ischemic priapism is likely to lead to substantial delay in resolution and higher risk of long-term ED. Hematological consultation and investigations of any abnormal laboratories can be pursued after emergent decompression of the priapism.[1]

Initial diagnostic and therapeutic management begins with corporal aspiration. Blood aspirate from ischemic priapism contains deoxygenated blood that appears dark, like motor oil, versus the oxygenated blood of nonischemic priapism, which is much lighter in color. Initial aspirates should be sent for blood gas analysis of P_{O_2}, P_{CO_2}, and pH. Normal nonischemic blood maintains a P_{O_2} of greater than 90 mm Hg with a P_{CO_2} of less than 40 mm Hg. The pH of normal blood is approximately 7.4. Values concerning for ischemic priapism (**Table 3**) are consistent with profound hypoxemic acidosis, often with a P_{O_2} of less than 30 and a P_{CO_2} of greater than 60. pH is acidotic, often less than 7.25. In line with the pathophysiology of nonischemic priapism, the blood gas values from penile aspirates mirror systemic oxygenated blood values.[4]

Color duplex Doppler ultrasonography (CDDU) of the penis is a diagnostic tool that is recommended as an option (where available) by AUA guidelines.[1] EAU guidelines go 1 step further and specify that CDDU of both the penis and perineum are recommended, can differentiate nonischemic from ischemic priapism, can identify fistula in 70% of cases, and may be used as an alternate to penile blood gas in the evaluation of priapism.[5] Ischemic priapism manifests as little to no blood flow in the cavernosal arteries on CDDU imaging. Nonischemic priapism manifests as normal to high blood flow velocities (numeral) within the arteries. It often may appear turbulent secondary to fistula blood flow. When nonischemic priapism is suspected, CDDU also may be used to look for anatomic abnormality, such as pseudoaneurysm. It is recommended that CDDU be performed

Table 2
Priapism checklist

Finding	Ischemic	Nonischemic
Fully rigid corpora	Common	Sometimes present
Penile pain	Common	Sometimes present
Penile blood gas: low PO_2, high CO_2	Common	Rare
Recent penile injection	Common	Sometimes present
Chronic erection without full rigidity	Rare	Common
Perineal trauma	Rare	Common

Table 3
Penile blood gas diagnostic values

Source	P_{O_2} (mm Hg)	P_{CO_2} (mm Hg)	pH
Normal nonischemic blood	>90	<40	7.4
Normal mixed venous blood	40	50	7.35
Ischemic blood from aspirate	<30	>60	<7.25

with the patient frog legged or in lithotomy, to permit scanning of the perineum as well as the penile shaft.[5]

INITIAL MANAGEMENT OF ISCHEMIC PRIAPISM

A variety of nonspecific home remedies, including exercise, ejaculation, ice packs, cold baths, and cold enemas, have been proposed for ischemic priapism; data on efficacy for these are scant and these are not recommended, particularly if they lead to delay in administration of more appropriate evidence-based therapies. Initial management of ischemic priapism should include administration of a dorsal nerve block with appropriate local anesthetic agent. After administration of local anesthetic, a large-bore needle (19 gauge or higher) is placed into the base of the pendulous shaft. Some centers recommend bilateral placement of 19-gauge needles to facilitate simultaneous irrigation and aspiration. The needles subsequently are used for diagnostic aspiration followed by therapeutic aspiration, aspiration/irrigation, and/or injection of a sympathomimetic drug (Fig. 1).[4,7] It is the authors' practice to utilize a single butterfly needle or angiocatheter at the base of the pendulous shaft, leaving just enough room to manually compress the penis below at the penoscrotal junction. Alternatively, 19-gauge needles can be placed bilaterally at the 9-o'clock or 3-o'clock positions if aspiration/irrigation is to be used. The authors compress the penoscrotal junction and aspirate the pendulous penis until it is completely detumesced. Maintaining the butterfly needle, compression is released and the penis allowed to refill. After several rounds of aspiration, red oxygenated blood consistently is observed. Restoration of the corporal environment with oxygenated blood is required for effective smooth muscle contraction in response to sympathomimetic drugs (phenylephrine, ephedrine, epinephrine, norepinephrine, metraminol, and etilefrine). Phenylephrine is the agent of choice, given higher selectivity of α_1-adrenoreceptors with lower stimulation of β-mediated ionotropic and chronotropic effects on the heart, leading to a more favorable side-effect profile.[32] Resuming finger compression at the penoscrotal junction, the authors begin dosing with phenylephrine in 100-μg to 200-μg aliquots every 5 minutes. Smaller aliquots of phenylephrine

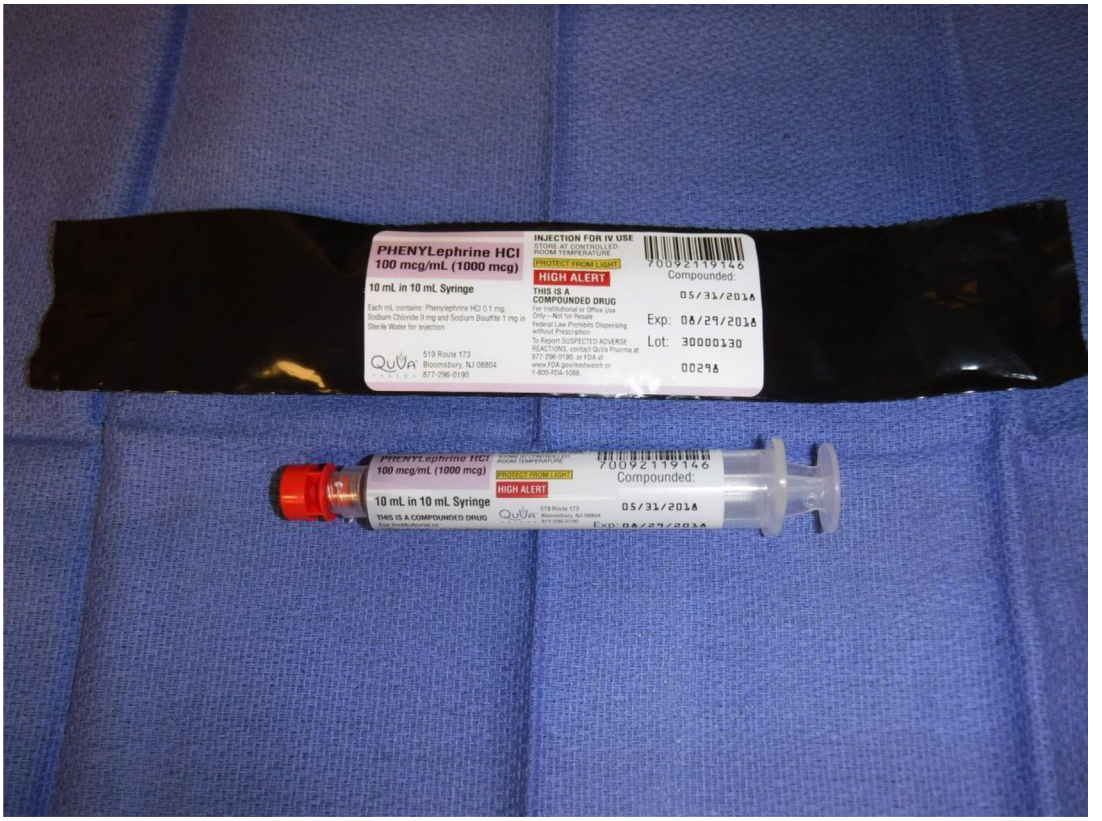

Fig. 1. At the author's institution, proprietary syringes of phenylephrine are available (10-mL syringe with 100 mcg/mL). This permits safe dosing. The authors typically use 2-mL aliquots with repeated aspirations followed by repeated injections. This avoids the dangers inherent with mixing solutions.

are recommended in children. This process of penoscrotal compression, aspiration, and injection generally can be repeated up to 1000 μg of phenylephrine without significant hypertension/reflex bradycardia in a healthy normotensive adult. At the authors' institution, proprietary syringes of phenylephrine are used (10 mL syringe with 200 μg/mL saline), allowing easy dosing in 1-mL aliquots. This avoids the dangers of mixing and sorting out diluent volumes. Priapism related to in office erectile function testing with ICIs easily may be reversed in most cases, with a single injection of 200 μg of phenylephrine; it is the authors' practice to reverse diagnostic erections at 1 hour.[4,7]

Methodical aspiration and irrigation with 0.9% normal saline as first-line therapy by AUA guideline.[1] A combination of corporal blood aspiration and saline irrigation effectively terminates priapism in 66% of cases compared with aspiration alone (reported success rate of 24%–36%).[33] Aspiration and saline irrigation continued throughout administration of phenylephrine is associated with increase in successful resolution in 81% of cases. In comparison the efficacy of injecting phenylephrine alone is lower 58%. These studies are consistent with the known physiology of ischemic priapism; the corporal smooth muscle will not contract and the ischemic priapism will not reverse so long as the environment is hypoxemic and hypercarbic. Aspiration followed by phenylephrine injection or irrigation with a diluted phenylephrine solution are more effective.[17]

Patients should be apprised of potential adverse events from phenylephrine administration, including headache, palpitations, and dizziness. In most clinic settings, automated blood pressure cuffs and heart rate monitoring are readily available; it is prudent for patients undergoing repeated aspiration/sympathomimetic injections or irrigations to be on sequential monitoring. Oral systemic therapy for sympathomimetics is not indicated in the treatment of acute ischemic priapism but has shown some efficacy in preventing stuttering priapism.[8]

SHUNTING PROCEDURES

Should initial management fail, second-line therapy for ischemic priapism is a distal shunting procedure. A variety of corporo-glanuar distal shunting procedures have been described, each with relatively similar rates of priapism resolution. The AUA (2003 and 2010) reviewed priapism resolution rates with their associated rates of postoperative ED in 3 common distal shunt procedures: Ebbehoj, Winter, and Al-Ghorab. These are detailed in **Table 4** and **Fig. 2**.[1]

Table 4
Distal Penile Shunting - Resolution and Outcomes

Distal Shunt	Resolution (%)	Erectile Dysfunction (%)
Ebbehoj	73	14
Winter	66	25
Al-Ghorab	74	25

Two modifications of distal shunt have been reported within the past decade. The T-shaped shunt involves placement of a no. 10 blade through the glans, 4 mm lateral to the meatus, into the corpora unilaterally; rotating the blade 90° away from the meatus; and then removing the blade.[34,35] Deoxygenated blood is milked out of the incision until there is flow of bright red oxygenated blood. The procedure can be repeated on the contralateral side if there is no resolution of the erection; if bilateral shunt creation fails, corporal tunneling with a smooth-tipped dilator through the incision can be considered.

The corpora snake maneuver is another recent innovation, consisting of bilateral distal corporoglanular shunting (Al-Ghorab) followed by passage of a 7/8 Hegar dilator down the length of the penis to the crura. The initial report indicated success in 8 of 10 patients, with a mean duration of priapism 75 hours.[36]

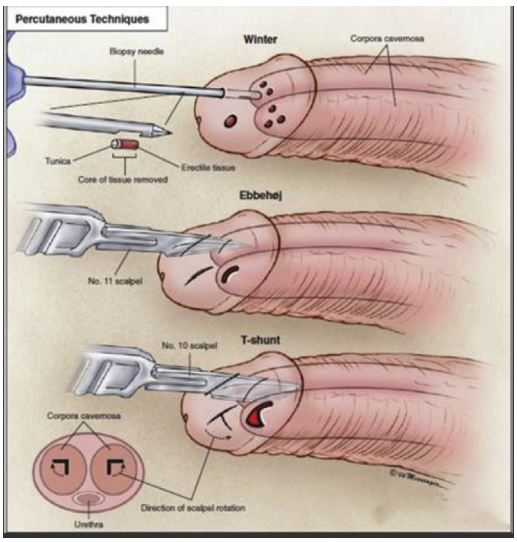

Fig. 2. Percutaneous shunting techniques. (*From* Tom F. Lue, Edoardo S. Pescatori,Surgical Techniques: Distal Cavernosum–Glans Shunts for Ischemic Priapism, The Journal of Sexual Medicine,Volume 3, Issue 4, 2006, Pages 749-752.)

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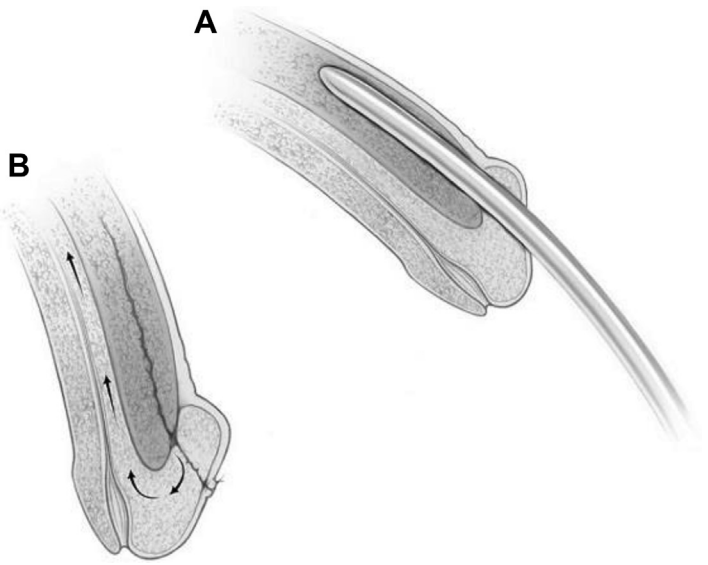

Fig. 3. (*A*) Corporal snake with Hegar dilator; (*B*) postdilation blood flow pathway. (*From* Burnett AL, Pierorazio PM. Corporal "snake" maneuver: corporoglanular shunt surgical modification for ischemic priapism. J Sex Med. 2009 Apr;6(4):1171-1176. doi: 10.1111/j.1743-6109.2008.01176.x. Epub 2009 Feb 4.)

As shown in **Figs. 3 and 4**A–D, Zacharakis and colleagues[37] describe 45 patients with priapism who underwent T-shunt with corporal snaking. For patients with priapism with duration less than 24 hours, 100% of patients had resolution of their erection. For erections of greater than 48 hours, only 30% had resolution. In the less than 24-hour duration group, approximately 50% of patients developed erectile dysfunction, whereas in the greater than 48-hour group, all patients had smooth muscle necrosis and long-term erectile dysfunction. Time to intervention was statically significant in its relationship to erectile function postoperatively.[37]

Despite high success rates of distal shunting to resolve ischemic priapism, approximately 50% of patients in 1 study became candidates for a penile implant.[38] Persistent ischemic priapism of greater than 36 hours uniformly results in erectile dysfunction. Informed consent prior to shunting procedures should clearly delineate for the patient that he will have some degree of ED; he may develop severe ED refractory to medical therapy. The goal of the successful shunt is to shorten the natural history of ischemic priapism, which is persistent painful erection of several weeks.

A recent novel surgical technique for refractory ischemic priapism is known as penoscrotal decompression (PSD). The surgical approach is identical to that of trans-scrotal placement of a penile prosthesis. One or both corporal bodies are isolated and opened sufficiently to permit advancing of a pediatric Yankauer suction distally and proximally to evacuate corporal thrombus. The technique, as first described, was used in patients failing distal corporo-glanuar shunting.[39] In a subsequent multi-institutional study, primary PSD was completely effective in 15 patients, with a mean ischemic duration of 71 hours.[40] The authors propose that primary PSD for ischemic priapism avoids trauma to the distal corpora and glans, which both is cosmetically deforming and makes the patient susceptible to future penile prosthetic erosion.

Conversion of ischemic to nonischemic hyperemic state may yield the appearance the day after surgery that ischemic erection persists. Before returning the patient to operating room for more interventions, it is prudent to perform penile blood gas and/or CDDU and determine whether cavernous flows have resumed.

Older literature described the creation of proximal shunts when distal shunting failed. Examples include shunts between the corpus cavernosum and corpus spongiosum (Quackles and Sacher). Proximal shunting of 1 or both corpora cavernosa directly to the saphenous or deep dorsal vein also has been described (known as Grayhack and Barry shunts, respectively). These procedures are associated with significant risks and unclear benefit; none appears in contemporary ischemic priapism management algorithms.

Early data suggest that there may be a role for antithrombotic therapy (ATT) in the management of ischemic priapism.[41] ATT may prevent thrombotic occlusion of shunts, which is thought to cause early recurrence of priapism.[41] One single-center study, in which 13 patients underwent shunting procedures without ATT therapy

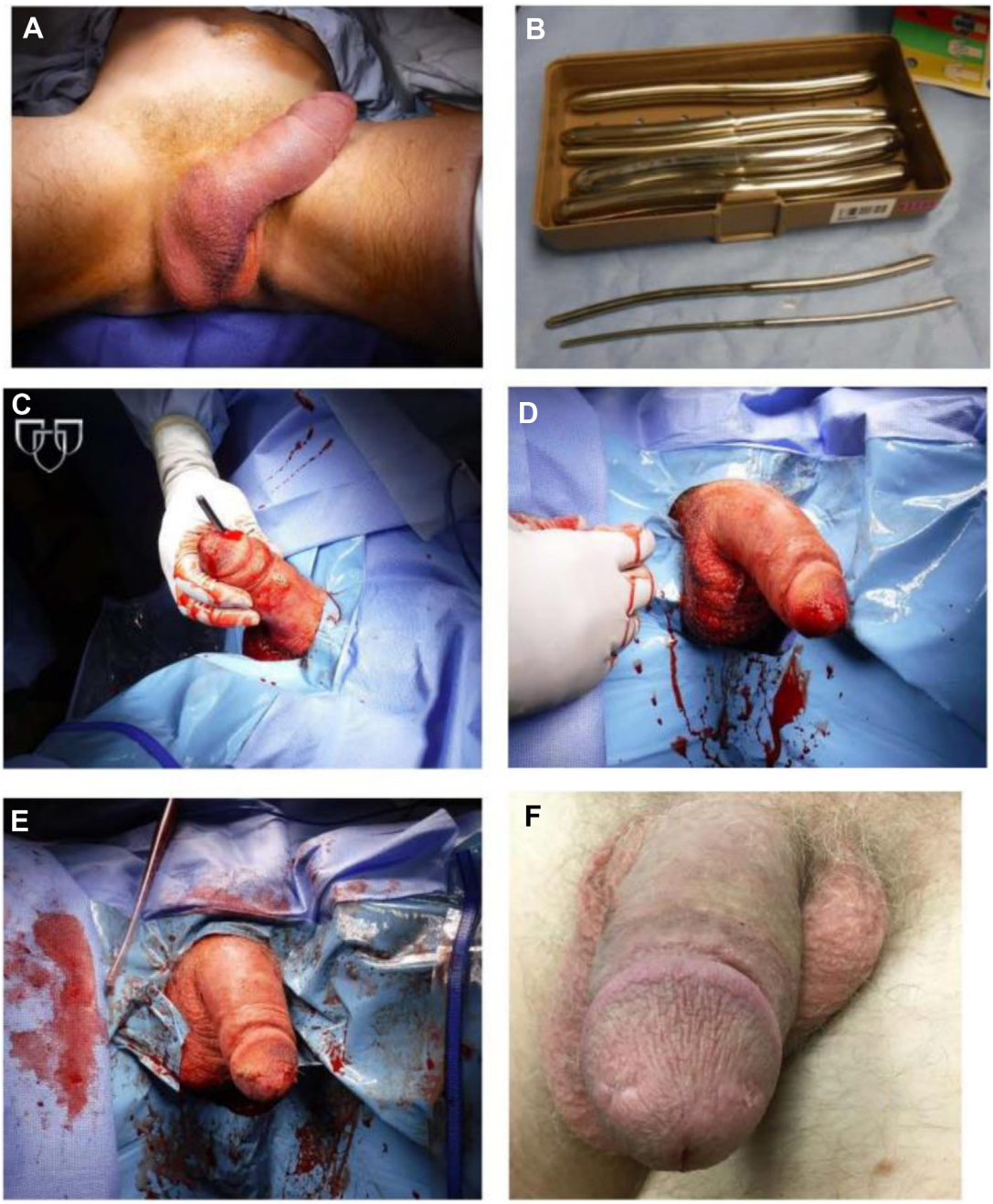

Fig. 4. (*A*) A 54-year-old white man with priapism following Trimix self-injection. He presented to outside hospital after 3 days of ischemic priapism. A Winter shunt failed to reverse priapism. (*B*, *C*, and *D*) On day 5, he was transferred and underwent bilateral T-shunts and corporal snake maneuver using Hegar dilators with successful detumescence. (*E*) Closed T-shunts. (*F*) Two-week postoperative wound check. (*Used with permission* of Mayo Foundation for Medical Education and Research, all rights reserved.)

compared with 9 patients who had both ATT and a shunting procedure, showed less recurrence of priapism, at rates of 11% versus 69%, respectively.[41] These preliminary findings from a single center should be investigated further, ideally in controlled and randomized studies.

EARLY VERSUS DELAYED PENILE PROSTHESIS PLACEMENT

Penile prosthesis placement is a useful management option for severe erectile dysfunction. In patients with a history of priapism, severe corporal

fibrosis can make placement of a prosthesis challenging for even the experienced implanter. For this reason, some experts advocate immediate or early (within several weeks of shunting procedures) penile prosthesis implantation in patients with ischemic priapism likely to result in severe ED.[42] Techniques, including extended corporotomies, excision of corporal fibrosis, and sharp dilation, are requisite.[43] Specialized dilators with a cutting edge may be utilized.[44] The EAU guidelines recommend consideration of penile implant in cases of ischemic priapism of greater than 48 hours' duration.[5] Immediate placement theoretically avoids the risk of penile foreshortening and narrowing that is common in postpriapism penile fibrosis. Unfortunately, immediate or early placement of penile prosthesis is in the setting of priapism carries some risks, including distal erosion, cavernositis, prosthetic infection, and significantly higher rates of reoperation.[45]

MANAGEMENT OF NONISCHEMIC PRIAPISM

Unlike ischemic priapism, nonischemic priapism is not an emergency. Symptoms can persist for years without adverse effects to sexual function.[4] Spontaneous resolution or response to conservative therapy has been reported in up to 62% of published series.[11] The mainstay of treatment remains selective arterial embolization.[1,5] Selective pudendal arteriography is invasive and requires significant skill set of an interventional radiologist familiar with pelvic anatomy. Pudendal arteriography should be reserved for those situations when the patient and urologist have elected to embolize nonischemic priapism and the risks and benefits have been discussed in detail.[4] The most common side effect after embolization is ED. The success rates with selective pudendal artery catheterization followed by embolization are reported between 89% and 100%, with normal erectile function postembolization seen in 75% to 86% of patients.[4] Recurrence has been identified to occur in up to 30% of patients, and retreatment with repeat embolization is an option in that setting.[7]

In patients who are not candidates for angioembolization or who do not have access to a center with the correct personnel or technology, surgical ligation of the cavernosal fistula is an alternate treatment option.[4] Even in these cases, transfer to a center with angiography capabilities is preferred. The surgical approach for this is transcorporal with corporal exploration and often requires intraoperative doppler examination. Care must be taken to not ligate the cavernosal artery in lieu of the true fistula.[46] Patients often must wait 1 months to 2 months after onset of nonischemic priapism secondary to perineal trauma in order for the fistula to fully mature prior to surgical intervention.[4]

SUMMARY

Priapism is a common urologic emergency that the urologist should be comfortable managing. Resolution of the acute event, preservation of the erectile function, and preventing recurrence are the goals of diagnosis and management. Treatment of priapism is diagnosis specific. Key Clinical Care Points of Priapism are:

1. Run the Priapism Checklist (**Table 2**)
2. Determine the etiology: ischemic vs. nonischemic
3. For ischemic priapism:
   a. Consider toxicology screening for substance abuse
   b. Consider Sickle Cell Disease supportive care: oxygenation, hydration, pain control, Hematology Consult
   c. Treat the ischemic compartment with penile aspiration and alpha adrenergic injection (phenylephrine)
   d. Surgical interventions are required for failure of pharmacologic reversal.
4. Nonischemic priapism is typically associated with perineal trauma.
   a. Injection of alpha adrenergics and surgical shunting are not appropriate.
   b. Spontaneous resolution has been reported.
   c. Supportive care with ice and compression on the perineum are reasonable.
   d. Selective pudendal arteriography for imaging and embolization can be considered for persistence and patient bother.

This article is a compilation of the evidence-based recommendations published by the American Urological Association in 2003 and the European Association of Urology published in 2014. The authors have updated those guidelines with current literature to facilitate best practices in the diagnosis and managemet of priapism.

DISCLOSURE

The authors have no disclosures.

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