Executive Summary

Purpose

Priapism is a persistent penile erection that continues hours beyond, or is unrelated to, sexual stimulation and results in a prolonged and uncontrolled erection. Given its time-dependent and progressive nature, priapism is a situation that both urologists and emergency medicine practitioners must be familiar with and comfortable managing. Although some forms of priapism are non-urgent in nature, prolonged (>4 hours) acute ischemic priapism, characterized by little or no cavernous blood flow and abnormal cavernous blood gases (i.e., hypoxic, hypercarbic, acidotic) represents a medical emergency and may lead to cavernosal fibrosis and subsequent erectile dysfunction. All patients with priapism should be evaluated emergently to identify the subtype of priapism (acute ischemic versus non-ischemic) and those with an acute ischemic event provided early intervention when indicated. This Guideline provides a clinical framework for the diagnosis, evaluation, and treatment (non-surgical and surgical) of acute ischemic priapism.

Methodology

A comprehensive search of the literature was performed by Emergency Care Research Institute for articles published between January 1, 1960 and May 1, 2020. Study designs included narrative reviews, systematic reviews, randomized controlled trials, controlled clinical trials, diagnostic accuracy studies, and observational studies (i.e., cohort studies, with and without comparison groups; case-control designs; case series). Searches identified 2948 potentially relevant articles, and 2516 of these were excluded at the title or abstract level for not meeting inclusion criteria for any key question. Full texts for the remaining 432 articles were ordered, and ultimately 137 unique articles were included in the report. These publications were used to create the majority of the clinical framework. When sufficient evidence existed, the body of evidence for a particular treatment was assigned a strength rating of A (high), B (moderate), or C (low), and evidence-based statements of Strong, Moderate, or Conditional Recommendation were developed. Additional information is provided as Clinical Principles and Expert Opinions when insufficient evidence existed.
American Urological Association (AUA) /Sexual Medicine Society of North America (SMSNA)

Guideline Statements

Diagnosis of Priapism

1. In patients presenting with priapism, clinicians should complete a medical, sexual, and surgical history, and perform a physical examination, including the genitalia and perineum. (Clinical Principle)

2. Clinicians should obtain a corporal blood gas at the initial presentation of priapism. (Clinical Principle)

3. Clinicians may utilize penile duplex Doppler ultrasound when the diagnosis of acute ischemic versus non-ischemic priapism is indeterminate. (Expert Opinion)

4. The clinician should order additional diagnostic testing to determine the etiology of diagnosed acute ischemic priapism; however, these tests should not delay, and should be performed simultaneously with, definitive treatment. (Expert Opinion)

Initial Management of Acute Ischemic Priapism

5. Clinicians should counsel all patients with persistent ischemic priapism that there is the chance of erectile dysfunction. (Moderate Recommendation; Evidence Level: Grade B)

6. Clinicians should counsel patients with a priapism event >36 hours that the likelihood of erectile function recovery is low. (Moderate Recommendation; Evidence Level: Grade B)

7. In patients presenting with a prolonged erection of four hours or less following intracavernosal injection pharmacotherapy for erectile dysfunction, clinicians should administer intracavernosal phenylephrine as the initial treatment option. (Expert Opinion)

8. In a patient with diagnosed acute ischemic priapism, conservative therapies (i.e., observation, oral medications, cold compresses, exercise) are unlikely to be successful and should not delay definitive therapies. (Expert Opinion)

Pre-Surgical Management of Acute Ischemic Priapism

9. Clinicians should manage acute ischemic priapism with intracavernosal phenylephrine and corporal aspiration, with or without irrigation, as first line therapy and prior to operative interventions. (Moderate Recommendation, Evidence Level: Grade C)

10. In patients receiving intracavernosal injections with phenylephrine to treat acute ischemic priapism, clinicians should monitor blood pressure and heart rate. (Clinical Principle)

Surgical Management of Acute Ischemic Priapism

11. Clinicians should perform a distal corporoglanular shunt, with or without tunneling, in patients with acute ischemic priapism who have failed pharmacologic intracavernosal reversal and corporal aspiration, with or without irrigation. (Moderate Recommendation, Evidence Level: Grade C)

12. In patients with acute ischemic priapism who failed a distal corporoglanular shunt, the clinician should consider corporal tunneling. (Moderate Recommendation, Evidence Level: Grade C)

13. Clinicians should counsel patients that there is inadequate evidence to quantify the benefit of performing a proximal shunt (of any kind) in a patient with persistent acute ischemic priapism after distal shunting. (Moderate Recommendation, Evidence Level: Grade C)
Post Shunting Management of Acute Ischemic Priapism

14. In an acute ischemic priapism patient with persistent erection following shunting, the clinician should perform corporal blood gas or color duplex Doppler ultrasound prior to repeat surgical intervention to determine cavernous oxygenation or arterial inflow. (Moderate Recommendation, Evidence Level: Grade C)

Penile Prosthesis

15. Clinicians may consider placement of a penile prosthesis in a patient with untreated acute ischemic priapism greater than 36 hours or in those who are refractory to shunting, with or without tunneling. (Expert Opinion)

16. In a patient with acute ischemic priapism who is being considered for placement of a penile prosthesis, clinicians should discuss the risks and benefits of early versus delayed placement. (Moderate Recommendation, Evidence Level: Grade C)

Introduction

Priapism is a condition resulting in a prolonged and uncontrolled erection. Although the incidence is relatively low, because of its time-dependent and progressive nature, priapism is a situation that both urologists and emergency medicine practitioners must be familiar with and comfortable managing. Although some forms of priapism are non-urgent in nature, prolonged (>4 hrs) acute ischemic priapism represents a medical emergency and may lead to cavernosal fibrosis and subsequent erectile dysfunction (ED).1, 2 Thus, all patients with priapism should be evaluated emergently to identify the sub-type of priapism (acute ischemic versus non-ischemic) and those with an acute ischemic event provided early intervention when indicated.

Given the significant heterogeneity of men presenting with acute ischemic priapism, the current Guideline emphasizes that specific interventions should be individualized based on clinical history and findings. While less-invasive, stepwise methods may be appropriate for most situations, others may be best managed using expedited surgical interventions. Decisions must also be based on patient objectives, available resources, and clinician experience. As such, a single pathway for managing the condition is oversimplified and no longer appropriate. Using this new, diversified approach, some men may be treated with intracavernosal injections (ICI) of phenylephrine alone, phenylephrine and aspiration, with or without irrigation, distal shunting, or non-emergent placement of a penile prosthesis.

Several other additions have been included in the guideline to address various diagnostic modalities. Specifically, the role of imaging (e.g., ultrasound, CT, MRI) is clarified during the initial diagnosis as well as post-treatment, such as with men exhibiting persistent pain or perceived rigidity post distal shunting.

New additions to the guideline also include greater detail on the role of:
- adjunctive laboratory testing,
- early involvement of urologists when presenting to the emergency room,
- discussion of conservative therapies,
- enhanced data for patient counseling on risks of ED and surgical complications,
- specific recommendations on intracavernosal phenylephrine with or without irrigation,
- inclusion of novel surgical techniques (e.g., tunneling), and
- early penile prosthesis placement.

Because priapism is rare and unpredictable, there is a dearth of high-level evidence-based data available from which strong evidence-based recommendations may be derived. Rather, most series represent small, single-site, retrospective, outcomes-based reports, with limited follow-up available and inconsistencies in reporting of outcomes. Similarly, as acute ischemic priapism is associated with ED (whether treated or untreated) and is progressive in nature, outcome reporting of various treatment strategies is inherently biased. These limitations preclude the ability to compare different treatment approaches or provide definitive recommendations in many cases. However, as with other American Urological Association (AUA) Guidelines, a thorough
contrive to the failure of the detumescence mechanism. Acute ischemic priapism is an acute, persistent erection characterized by little or no cavernous blood flow and abnormal cavernous blood gases (i.e., hypoxic, hypercarbic, acidotic). The corpora cavernosa are fully rigid and tender to palpation. Patients typically report pain. A variety of etiologic factors may contribute to the failure of the detumescence mechanism in this condition. Acute ischemic priapism is an emergency. As the natural history of untreated acute ischemic priapism includes days to weeks of painful erections followed by permanent loss of erectile function, the condition requires prompt evaluation and may require emergency management.

Non-ischemic (arterial, high flow): a persistent erection that may last hours to weeks and is frequently recurrent. Although the underlying physiology is incompletely understood, it likely results from unregulated control of arterial inflow and cavernous smooth muscle tone. Erections are nearly always non-painful, and cavernosal blood gas measurements are consistent with arterial blood. In contrast to acute ischemic priapism, the non-ischemic variant is not considered a medical emergency.

Resolution of acute ischemic priapism is characterized by the penis returning to a flaccid, nonpainful state, with restoration of penile blood flow. However, in many cases, persistent penile edema, ecchymosis, and partial erections can occur and mimic unresolved priapism. This often relates to the duration of priapism and may also signify segmental regions of cavernosal ischemia/necrosis.

Both acute ischemic and non-ischemic priapism may recur over time. The term stuttering priapism is used specifically with acute ischemic priapism, and signifies an intermittent, recurrent subtype, in which unwanted painful erections occur repeatedly with intervening periods of detumescence. Management of this condition requires not only treatment of acute episodes, but also focuses on future prevention and mitigation of an acute ischemic event necessitating surgical management.

Panel Formation
The Panel was created in 2018 by the American Urological Association Education and Research, Inc. This guideline was developed in collaboration with the Sexual Medicine Society of North America (SMSNA). The Practice Guidelines Committee (PGC) of the AUA selected the Panel Chairs who in turn appointed the additional panel members with specific expertise in this area, in conjunction with SMSNA. Additionally, the Panel included American College of Emergency Physicians and patient representation. Funding of the Panel was provided by the AUA; panel members received no remuneration for their work.

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The American Urological Association (AUA) /Sexual Medicine Society of North America (SMSNA)

Methods and Methodology

Literature Search

A comprehensive search of the literature was performed by staff in the Clinical Excellence and Safety Group at the Emergency Care Research Institute (ECRI). ECRI searched Medline and EMBASE for articles published between January 1, 1960 and May 1, 2020. Study designs included narrative reviews, systematic reviews, randomized controlled trials (RCTs), controlled clinical trials, diagnostic accuracy studies, and observational studies (i.e., cohort studies, with and without comparison groups; case-control designs; case series).

Study Screening and Selection

Relevant references retrieved by the literature searches were loaded into Distiller SR, systematic review software (Evidence Partners, Ottawa, Ontario, Canada). All screening through the abstract level was performed in Distiller SR. One analyst (Dr. Jeff Oristaglio) performed initial title screening and his list of excluded studies was reviewed by Dr. Stacey Uhl to confirm that no potentially relevant studies had been excluded. One analyst (Dr. Oristaglio) performed screening at the abstract level. References deemed with potential to satisfy the inclusion criteria (outlined below) and provide evidence for addressing one or more of the key questions specified by the panel were retrieved in full text for review by the team. Five analysts participated in full-text screening and approximately 10% of the studies at this level were reviewed by at least two analysts (double-screening). Conflicting decisions between analysts were tracked, reviewed, discussed, and resolved by consensus before individual analysts were allowed to screen full-text studies independently. This assured that a suitable sample of studies covering most of the key questions were assessed by all analysts and that decisions on inclusion or exclusion were understood. For all excluded studies, the reason for exclusion, and the level at which it was excluded (based on abstract or full text review) was recorded.

Inclusion Criteria

General Criteria

To focus the analysis on the most relevant evidence, only peer-reviewed journal articles published in English from January 1, 1960 to May 1, 2020, reporting data on human subjects with relevance to one or more of the key questions were considered. With regard to enrollment size, only individual case studies (n=1 subject) were systematically excluded, though some studies of this type were allowed when the quantity of evidence for a particular question was very low.

In summary, general inclusion criteria were as follows:

- published, peer-reviewed full-length individual studies or systematic reviews,
- individual studies limited to those not included in relevant systematic reviews (to avoid double-counting of evidence),
- published guidelines with systematic reviews and acceptable methodological details (including study quality assessment) and abstractable data,
- studies that enrolled or analyzed human male participants,
- studies that were published in the English language, and
- studies that had a patient enrollment of ≥2 per group at follow-up (except in instances of very limited evidence).

Exclusion criteria were as follows:

- studies not published in English,
- case reports (n=1 studies), except in instances of very limited evidence,
- narrative reviews,
- guidelines or reviews with no systematic literature search or methodological details (e.g., risk of bias assessment),
- opinions/editorials/commentaries,
- conference abstracts, and
- in vitro studies or animal studies.

Assessment of Study Quality

Ideally, different key questions required different types of evidence in terms of trial design and study type. However, realizing that the evidence base for this topic would be limited, very liberal inclusion criteria was adopted. The vast majority of studies were observational in design and most of these were retrospective. The criteria set for assessing the quality of different study designs, prior to
formal assessments, are listed below. Note that there were not any RCTs with comparisons that addressed any of the specified key questions. Because of this, while RCTs with relevant data were accepted, they were typically graded as observational studies.

For assessing RCTs, an adaptation of the Cochrane risk-of-bias instrument was used, which assessed five of its seven domains:

- random sequence generation,
- allocation concealment,
- incomplete outcome data,
- selective outcome reporting, and
- other potential sources of bias (e.g., lack of balance in group baseline characteristics).

The Cochrane domains concerning blinding, which is not practically or ethically feasible for surgical interventions, were not considered.

For non-randomized comparative trials, the following domains were assessed:

- prospective versus retrospective design,
- consecutive enrollment,
- baseline comparability of groups,
- use of statistical controls for confounding,
- incomplete outcome data,
- selective outcome reporting, and
- other potential threats to validity.

For diagnostic accuracy studies, appropriate items from the QUADAS (Quality Assessment of Diagnostic Accuracy Studies) instrument were used:

- Was a consecutive or random sample of patients enrolled?
- Was a case-control design avoided (when the true status of patients was known prior to inclusion in the study)?
- Did the study avoid inappropriate exclusions (i.e., spectrum bias)?
- Were the index test results interpreted without knowledge of the results of the reference standard?
- Was the reference standard likely to classify the target condition correctly?

Finally, and most importantly, for this evidence base, observational and single-arm studies were assessed with the following domains:

- prospective versus retrospective design,
- consecutive enrollment,
- methodological detail (e.g., specification of follow-up time),
- incomplete outcome data,
- selective outcome reporting, and
- other potential threats to validity (e.g., lacking measures of dispersion; failure to use appropriate statistical techniques).

**Determination of Evidence Strength**

The AUA employs a three-tiered strength of evidence system to underpin evidence-based Guideline statements. In short, high certainty by GRADE (Grading of Recommendations Assessment, Development and Evaluation) translates to AUA A-category strength of evidence, moderate to B, and both low and very low to C (Table 1).

The AUA categorizes body of evidence strength as Grade A (well-conducted and highly-generalizable RCTs or exceptionally strong observational studies with consistent findings), Grade B (RCTs with some weaknesses of procedure or generalizability or moderately strong observational studies with consistent findings), or Grade C (RCTs with serious deficiencies of procedure or generalizability or extremely small sample sizes or observational studies that are inconsistent, have small sample sizes, or have other problems that potentially confound interpretation of data). By definition, Grade A evidence is evidence about which the Panel has a high level of certainty, Grade B evidence is evidence about which the Panel has a moderate level of certainty, and Grade C evidence is evidence about which the Panel has a low level of certainty.

Level B evidence may include observational studies rated as low quality if findings are consistent and of a strong treatment effect. Panelists can therefore make a stronger statement based on this evidence. In instances where evidence for a given question is rated as level C, this does not mean that the panel cannot make a statement based on the evidence, particularly if findings from included studies are not substantially different. Further-
more, in cases where studies show conflicting evidence or evidence is sparse, panelists may still use clinical judgment to inform a guideline statement. Note that the worst possible rating for RCTs is Level B. Therefore, evidence comprised of RCTs and systematic reviews that included only RCTs would be judged as either Level A or Level B.

**AUA Nomenclature: Linking Statement Type to Evidence Strength**

The AUA nomenclature system explicitly links statement type to body of evidence strength, level of certainty, magnitude of benefit or risk/burdens, and the Panel’s judgment regarding the balance between benefits and risks/burdens (Table 2). Strong Recommendations are directive statements that an action should (benefits outweigh risks/burdens) or should not (risks/burdens outweigh benefits) be undertaken because net benefit or net harm is substantial. Moderate Recommendations are directive statements that an action should (benefits outweigh risks/burdens) or should not (risks/burdens outweigh benefits) be undertaken because net benefit or net harm is moderate. Conditional Recommendations are non-directive statements used when the evidence indicates that there is no apparent net benefit or harm, when benefits and harms are finely balanced, or when the balance between benefits and risks/burden is unclear.

All three statement types may be supported by any body of evidence strength grade. Body of evidence strength Grade A in support of a Strong or Moderate Recommendation indicates that the statement can be applied to most patients in most circumstances and that future research is unlikely to change confidence. Body of evidence strength Grade B in support of a Strong or Moderate Recommendation indicates that the statement can be applied to most patients in most circumstances, but that better evidence could change confidence. Body of evidence strength Grade C in support of a Strong or Moderate Recommendation indicates that the statement can be applied to most patients in most circumstances, but that better evidence is likely to change confidence. Conditional Recommendations also can be supported by any evidence strength. When body of evidence strength is Grade A, the statement indicates that benefits and risks/burdens appear balanced, the best action depends on patient circumstances, and future research is unlikely to change confidence. When body of evidence strength Grade B is used, benefits and risks/burdens appear balanced, the best action also depends on individual patient circumstances and better evidence could change confidence. When body of evidence strength Grade C is used, there is uncertainty regarding the balance between benefits and risks/burdens; therefore, alternative strategies may be equally reasonable, and better evidence is likely to change confidence.

Where gaps in the evidence existed, the Panel provides guidance in the form of Clinical Principles or Expert Opinions with consensus achieved using a modified Delphi technique if differences of opinion emerged. A Clinical Principle is a statement about a component of clinical care that is widely agreed upon by urologists or other clinicians for which there may or may not be evi-

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**Table 1: Strength of Evidence Definitions**

<table>
<thead>
<tr>
<th>AUA Strength of Evidence Category</th>
<th>GRADE Certainty Rating</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A</strong></td>
<td>High</td>
<td>We are very confident that the true effect lies close to that of the estimate of the effect</td>
</tr>
<tr>
<td><strong>B</strong></td>
<td>Moderate</td>
<td>We are moderately confident in the effect estimate. The true effect is likely to be close to the estimate of the effect, but there is a possibility that it is substantially different</td>
</tr>
<tr>
<td><strong>C</strong></td>
<td>Low</td>
<td>Our confidence in the effect estimate is limited. The true effect may be substantially different from the estimate of the effect</td>
</tr>
<tr>
<td></td>
<td>Very Low</td>
<td>We have very little confidence in the effect estimate. The true effect is likely to be substantially different from the estimate of the effect</td>
</tr>
</tbody>
</table>
### TABLE 2: AUA Nomenclature Linking Statement Type to Level of Certainty, Magnitude of Benefit or Risk/Burden, and Body of Evidence Strength

<table>
<thead>
<tr>
<th>Evidence Strength A</th>
<th>Evidence Strength B</th>
<th>Evidence Strength C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>(High Certainty)</strong></td>
<td><strong>(Moderate Certainty)</strong></td>
<td><strong>(Low Certainty)</strong></td>
</tr>
<tr>
<td><strong>Strong Recommendation</strong></td>
<td>Benefits &gt; Risks/Burdens (or vice versa)</td>
<td>Benefits &gt; Risks/Burdens (or vice versa)</td>
</tr>
<tr>
<td>(Net benefit or harm substantial)</td>
<td>Net benefit (or net harm) is substantial</td>
<td>Net benefit (or net harm) is substantial</td>
</tr>
<tr>
<td>Applies to most patients in most circumstances and future research is unlikely to change confidence</td>
<td>Applies to most patients in most circumstances but better evidence could change confidence</td>
<td>Applies to most patients in most circumstances but better evidence is likely to change confidence (rarely used to support a Strong Recommendation)</td>
</tr>
<tr>
<td><strong>Moderate Recommendation</strong></td>
<td>Benefits &gt; Risks/Burdens (or vice versa)</td>
<td>Benefits &gt; Risks/Burdens (or vice versa)</td>
</tr>
<tr>
<td>(Net benefit or harm moderate)</td>
<td>Net benefit (or net harm) is moderate</td>
<td>Net benefit (or net harm) is moderate</td>
</tr>
<tr>
<td>Applies to most patients in most circumstances and future research is unlikely to change confidence</td>
<td>Applies to most patients in most circumstances but better evidence could change confidence</td>
<td>Applies to most patients in most circumstances but better evidence is likely to change confidence</td>
</tr>
<tr>
<td><strong>Conditional Recommendation</strong></td>
<td>Benefits = Risks/Burdens</td>
<td>Benefits = Risks/Burdens</td>
</tr>
<tr>
<td>(No apparent net benefit or harm)</td>
<td>Best action depends on individual patient circumstances</td>
<td>Best action appears to depend on individual patient circumstances</td>
</tr>
<tr>
<td>Future research unlikely to change confidence</td>
<td>Better evidence could change confidence</td>
<td>Better evidence likely to change confidence</td>
</tr>
<tr>
<td><strong>Clinical Principle</strong></td>
<td>A statement about a component of clinical care that is widely agreed upon by urologists or other clinicians for which there may or may not be evidence in the medical literature</td>
<td></td>
</tr>
<tr>
<td><strong>Expert Opinion</strong></td>
<td>A statement, achieved by consensus of the Panel, that is based on members' clinical training, experience, knowledge, and judgment for which there is no evidence</td>
<td></td>
</tr>
</tbody>
</table>
Evidence in the medical literature. Expert Opinion refers to a statement, achieved by consensus of the Panel, that is based on members’ clinical training, experience, knowledge, and judgment for which there may or may not be evidence.

Results

Searches identified 2948 potentially relevant articles, and 2516 of these were excluded at the title or abstract level for not meeting inclusion criteria for any key question. Full text publications for the remaining 432 articles were ordered, and ultimately 137 unique articles were included for this report.

Peer Review and Document Approval

An integral part of the guideline development process at the AUA is external peer review. The AUA conducted a thorough peer review process to ensure that the document was reviewed by experts in the diagnosis and management of priapism. In addition to reviewers from the AUA PGC, Science and Quality Council, and Board of Directors, the document was reviewed by representatives from SMSNA, American College of Emergency Physicians, and external content experts. A call for reviewers was placed on the AUA website from April 14 - May 3, 2021 to allow any additional interested parties to request a copy of the document for review. The guideline was also sent to the Urology Care Foundation to open the document further to the patient perspective. The draft guideline document was distributed to 55 peer reviewers, including 9 external reviewers. All peer review comments were blinded and sent to the Panel for review. In total, 41 reviewers provided comments. At the end of the peer review process, a total of 519 comments were received. Following comment discussion, the Panel revised the draft as needed. Once finalized, the guideline was submitted for approval to the AUA PGC, Science and Quality Council, Board of Directors, and the governing bodies of SMSNA.

Guideline Statements

Diagnosis of Priapism

1. In patients presenting with priapism, clinicians should complete a medical, sexual, and surgical history, and perform a physical examination, including the genitalia and perineum. (Clinical Principle)

2. Clinicians should obtain a corporal blood gas at the initial presentation of priapism. (Clinical Principle)

3. Clinicians may utilize penile duplex Doppler ultrasound when the diagnosis of acute ischemic versus non-ischemic priapism is indeterminate. (Expert Opinion)

4. The clinician should order additional diagnostic testing to determine the etiology of diagnosed acute ischemic priapism; however, these tests should not delay, and should be performed simultaneously with, definitive treatment. (Expert Opinion)

The optimal method for diagnosing priapism and differentiating acute ischemic versus non-ischemic subtypes has not been defined. Specifically, no studies have di-
rectly compared various diagnostic algorithms or provided positive and negative predictive values for one form of testing over another. In the majority of cases of priapism, the differentiation of acute ischemic versus non-ischemic may be made using only the history and physical exam. However, in cases where the subtype is indeterminate, additional testing may be warranted.

The initial presentation of priapism often happens acutely and in the setting of an emergency department. Early identification of this diagnosis, as well as the subtype of priapism, allows for rapid initiation of indicated treatments. Thus, collaboration between emergency medicine physicians and urologic specialists is imperative to the provision of appropriate, timely care.

**History**
Understanding the history of the episode of priapism is important because the history and etiology may determine the most effective treatment. Historical features that should be identified are:

- baseline erectile function,
- duration of erection,
- degree of pain,
- previous history of priapism and its treatment,
- use of drugs that might have precipitated the episode (Table 3),
- history of pelvic, genital, or perineal trauma, especially a perineal straddle injury,
- personal or family history of sickle cell disease or other hematologic abnormality, and
- personal history of malignancies, particularly genitourinary malignancies.

**Examination**
The genitalia, perineum, and abdomen should be carefully examined. In patients with priapism, the corpora cavernosa are typically affected while the corpus spongiosum and the glans penis are not. In patients with acute ischemic priapism, the corpora cavernosa are often fully rigid and tender, while men with non-ischemic exhibit partial corporal tumescence (Table 4). Abdominal, pelvic, and perineal examination may reveal evidence of trauma or malignancy.

**Corporal Blood Gas**
Blood gas testing is the most common diagnostic methods of distinguishing acute ischemic from non-ischemic priapism when the diagnosis cannot be made by history alone. Blood aspirated from the corpus cavernosum in patients with acute ischemic priapism is hypoxic (dark red), while corporal blood in non-ischemic priapism is normally oxygenated (bright red). Corporal blood gases in men with acute ischemic priapism typically have a PO2 of < 30 mm Hg, a PCO2 of > 60 mm Hg, and a pH < 7.25. cavernous blood gases in men with non-ischemic priapism are similar to the blood gases of arterial blood, while normal flaccid penis cavernous blood gas levels are approximately equal to those of mixed venous blood. Typical blood gas values are shown in Table 5.

Obtaining a corporal blood gas during the initial diagnosis of ischemic priapism should be performed in the majority of cases. However, there are certain clinical situations where a blood gas may be omitted at the clinician’s discretion. Examples include priapism induced by in-office or at home ICI therapies, cases of recurrent ischemic priapism (i.e., sickle cell disease), or when the diagnosis is abundantly clear by history and examination alone. In the majority of cases presenting acutely to the emergency department, however, corporal blood gas should be obtained during initial evaluation to diagnosis the priapism subtype.

**Radiologic Evaluation**
Penile duplex Doppler ultrasonography (PDUS) is not the primary way to diagnose priapism. While radiologic imaging studies have demonstrated utility in the evaluation and management of priapism, this is largely outside of the acute phase of presentation. As such, imaging studies should not be incorporated into the acute evaluation and management of priapism in the emergency department by non-urologist specialists.

However, imaging may be utilized in less clearly delineated cases to differentiate between acute ischemic and non-ischemic priapism. PDUS findings that are consistent with acute ischemic priapism include bilateral absence of flow through the cavernosal arteries, peak systolic flows <50 cm/sec, mean velocity <6.5 cm/sec, and diastolic reversal (i.e., negative end diastolic velocities). In contrast, non-ischemic priapism is associated with peak systolic velocities of >50 cm/sec in a study of 52 men with priapism. In the non-acute setting, PDUS may also identify anatomical abnormalities, such as a cavernous artery fistula or pseudoaneurysm in patients who already have the diagnosis of non-ischemic priapism. These abnormalities may occur following a strad-
dle injury or direct scrotal trauma and are, therefore, most often found in the perineal portions of the corpora cavernosa.

Pelvic MRIs have also been described as another potential imaging modality to assist in acute ischemic priapism management. In one notable study, T2-weighted gadolinium-enhanced MRI demonstrated 100% sensitivity in identifying non-viable corporal smooth muscle and which predicted future erectile dysfunction. However, given the time sensitivity of ischemic priapism diagnosis and management, MRI likely does not have a role in the initial diagnostic and treatment phase of priapism.

**Laboratory Evaluation**

The optimal blood tests to identify the etiology of ischemic priapism have not been defined and should be selectively ordered based on specific patient risk factors and clinical suspicion. A complete blood count is a routine test that may identify elevated white blood cell counts, potentially identifying cases where priapism is due to underlying malignancy (e.g., leukemia). Among men with sickle cell disease, acute ischemic priapism is associated with lower hemoglobin and elevated lactate dehydrogenase, bilirubin, aspartate aminotransferase, reticulocyte count, white blood cells, and platelet counts. Platelet and eosinophil counts may also be elevated in men with acute ischemic priapism. While these laboratory values may contribute to the identification of underlying cause, they often will not be used to guide treatment of the acute presentation.

Hemoglobin electrophoresis, and other sickle cell testing, may be appropriate in select clinical scenarios and based on underlying risk factors (e.g., patient race). In most cases, men with sickle cell disease will have been diagnosed previously. The yield of identifying men with previously undiagnosed sickle cell trait/disease among a cohort of men presenting with priapism is not well established. As such, electrophoresis and other sickle cell testing should be reserved for select clinical scenarios. A reticulocyte count is often used in the evaluation and management in patients carrying a diagnosis of sickle cell disease during presentations of acute vaso-occlusive crisis and may thus be incorporated into the workup of these patients, along with a complete blood count.

Screening for psychoactive drugs and urine toxicology may also be performed. Priapism has been associated with certain medications and substances, including drugs of abuse, psychoactive medications, and other classes of medication, both in therapeutic and overdose levels. Despite the role these substances play in the development of priapism, it is notable that testing for potential substances may have a high rate of false negativity, particularly with synthetic and otherwise altered versions of common illicit substances. Additionally, patient history alone may provide much of this information without needing to perform additional testing. Given these associated risks, a thorough medication and social history may provide enough information for the examining practitioner to determine the underlying cause of the priapism presentation without collection of these studies.

**Initial Management of Acute Ischemic Priapism**

5. Clinicians should counsel all patients with persistent ischemic priapism that there is the chance of erectile dysfunction. (Moderate Recommendation; Evidence Level: Grade B)

6. Clinicians should counsel patients with a priapism event >36 hours that the likelihood of erectile function recovery is low. (Moderate Recommendation; Evidence Level: Grade B)

The patient with diagnosed acute ischemic priapism should be informed that the natural history of untreated acute ischemic priapism is possible permanent loss of erectile function and corporal fibrosis leading to penile shortening. ED is the most significant complication in patients with prolonged acute ischemic priapism and the likelihood of developing ED is related to the length of an acute ischemic priapism event. Bennett and Mulhall demonstrated that sickle cell patients with priapism of >36 hours had permanent erectile dysfunction with no men recovering erectile function. As the duration of acute ischemic priapism increases, so too does necrosis of the smooth muscle tissue, resulting in fibrosis and ED. While the exact time point of irreversible smooth muscle loss is undetermined, it is recognized that smooth muscle edema and atrophy occur as early as six hours. In a study by Zacharakis et al., patients who presented with unresolved acute ischemic
Table 3: Drugs/Medications Associated with Priapism

<table>
<thead>
<tr>
<th>Drug class</th>
<th>Documented examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attention deficit hyperactivity disorder medications</td>
<td>Atomoxetine</td>
</tr>
<tr>
<td>Alpha-Adrenergic Blockers</td>
<td>Doxazosin, prazosin, tamsulosin, terazosin</td>
</tr>
<tr>
<td>Anticoagulants</td>
<td>Heparin, warfarin</td>
</tr>
<tr>
<td>Antidepressants/Antipsychotics</td>
<td>Bupropion, chlorpromazine, clozapine, fluoxetine, lithium, olanzapine, phenothiazines, risperidone,</td>
</tr>
<tr>
<td>Antihypertensives</td>
<td>Guanethidine, hydralazine, propranolol</td>
</tr>
<tr>
<td>Hormone therapy</td>
<td>Gonadotropin-releasing hormone, testosterone</td>
</tr>
<tr>
<td>Recreational drugs</td>
<td>Alcohol, cocaine, marijuana</td>
</tr>
<tr>
<td>Vasoactive erectile agents</td>
<td>Alprostadil, papaverine, phentolamine, prostaglandin E1, combination agents</td>
</tr>
</tbody>
</table>

Table 4: Key Findings in the Evaluation of Priapism

<table>
<thead>
<tr>
<th>Finding</th>
<th>Ischemic Priapism</th>
<th>Non-ischemic Priapism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corpora cavernosa fully rigid</td>
<td>U</td>
<td>O</td>
</tr>
<tr>
<td>Penile pain</td>
<td>U</td>
<td>O</td>
</tr>
<tr>
<td>Abnormal cavernous blood gases</td>
<td>U</td>
<td>O</td>
</tr>
<tr>
<td>Blood abnormalities and hematologic malignancy</td>
<td>S</td>
<td>O</td>
</tr>
<tr>
<td>Recent intracavernous vasoactive drug injections</td>
<td>S</td>
<td>O</td>
</tr>
<tr>
<td>Chronic, well-tolerated tumescence without full rigidity</td>
<td>O</td>
<td>U</td>
</tr>
<tr>
<td>Perineal trauma</td>
<td>O</td>
<td>S</td>
</tr>
</tbody>
</table>

O: Seldom present; S: Sometimes present; U: Usually present

Table 5: Typical Blood Gas Values

<table>
<thead>
<tr>
<th>Source</th>
<th>PO2 (mm Hg)</th>
<th>PCO2 (mm Hg)</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute ischemic priapism (cavernous blood)</td>
<td>&lt;30</td>
<td>&gt;60</td>
<td>&lt;7.25</td>
</tr>
<tr>
<td>Normal arterial blood (room air)</td>
<td>&gt;90</td>
<td>&lt;40</td>
<td>7.40</td>
</tr>
<tr>
<td>Normal mixed venous blood (room air)</td>
<td>40</td>
<td>50</td>
<td>7.35</td>
</tr>
</tbody>
</table>
Priapism >48 hours had extensive necrosis of the cavernous smooth muscle, which resulted in severe ED. They also found that >50% of patients with priapism lasting between 24-48 hours had permanent ED.

Managing patients who present with acute ischemic priapism is considered an urologic emergency and the clinician should not treat the patient conservatively. As the duration of the priapism increases, patients may be refractory to first-line treatments, such as aspiration, with or without irrigation, and ICI with phenylephrine. In a patient with acute ischemic priapism >36 hours, surgical interventions, such as distal shunting with or without tunneling may be required to achieve detumescence; it is unlikely the acute ischemic event will resolve with aspiration and ICI therapy with phenylephrine. While these measures may resolve the symptoms of priapism, patients will develop post-operative ED. Clinical judgement and patient-specific factors will dictate the interventions necessary to resolve the priapic event. In recent years, immediate placement of a penile prosthesis has been advocated and will be addressed in later Guideline statements.

Studies included in the evidence base for this Guideline (one observational and four retrospective chart reviews) reported on erectile function following distal shunt procedures with or without tunneling. In most cases, distal shunts with tunneling had a deleterious effect on erectile function recovery; the use of tunneling can cause destruction of the corporal tissue due to shear tearing and injury to the penile vasculature. While the exact cause of permanent ED is unknown, contributing factors include baseline erectile function, duration of ischemic priapism, and surgical procedures (distal shunt with tunneling technique) utilized to reverse the acute ischemic priapic episode.

A retrospective chart review of 19 acute ischemic priapism patients by Ortac et al. evaluated detumescence and ED outcomes in patients who failed conservative measures (i.e., aspiration and injection of an intracavernosal alpha-adrenergic agent) and subsequently underwent shunting with or without tunneling. Patients were divided into four groups by duration of priapism (<36 hours, 36-48 hours, 48-72 hours, >72 hours). While all patients experienced detumescence, statistical analysis showed that duration of priapism (median: 58 hours) was negatively correlated with post-operative IIEF-5 scores (p=0.046). Sixteen (84.21%) patients experienced post-operative ED; 46.35% (n=9) were unable to achieve any spontaneous erections. The mean post-operative IIEF-5 score across all time durations was 12.68 hours (range 5-23 hours); patients with priapism <48 hours had higher IIEF-5 (16.4) scores than patients with priapism >48 hours (10; p<0.05). However, all patients had some degree of ED post distal shunting, with or without tunneling.

In another retrospective chart review of patients with prolonged acute ischemic priapism (n=45; median duration: 96 hours), Zacharakis et al. likewise found a negative correlation between the duration of priapism and developing post-operative ED. Patients were divided into four groups by duration of priapism: <12 hours, 12-24 hours, 24-36 hours, 36-48 hours, >48 hours. All patients, regardless of duration, were refractory to aspiration and ICI and subsequently underwent distal shunting with tunneling. In those with acute ischemic priapism lasting 36 hours, 50% had severe ED and 25% had mild to moderate ED; in patients with priapism events lasting 48 hours, 60% had severe ED and 20% had mild to moderate ED; severe ED developed in 100% of patients who had priapism >48 hrs. Across all patient groups, post-operative IIEF-5 scores were reduced to a mean of 7.7 (from a pre-operative mean of 24), which was related to the duration of the priapism event (p<0.0005). Histopathological results corroborate these findings. Each patient had a distal and proximal smooth muscle biopsy taken from the corpora cavernosa; histology results showed that the percentage of viable tissue decreased, and the percentage of fibrosis and necrosis increased, with the duration of the priapism, such that at 36 hours no patients had viable tissue left and necrosis and fibrosis started as early as 12-24 hours.

Similar results were found in other retrospective case series. Pal et al. prospectively observed 19 patients who presented with acute ischemic priapism (mean duration: 96.7 hours), all of whom failed aspiration and ICI and subsequently underwent distal shunting. Only five patients (26.3%) preserved normal erectile function at follow-up. Of the eight patients in the Segal et al. study who were successfully treated with distal shunting (mean duration: 75 hours) none reported return of intact spontaneous erectile function and only two reported partial recovery of erectile function.
All patients (n=12; mean duration: 2.8 days) in the study by Lian et al.\textsuperscript{13} developed ED following distal shunts plus tunneling; the mean pre-surgical IIEF score was 23.7; the follow-up score was 11.7, indicating a significant decrease in post-surgical erectile function (p<0.01).

It is difficult to ascertain if the duration of acute ischemic priapism itself or the surgical procedures to relieve it are primarily responsible for the development of post-operative ED. Age and pre-operative ED may also be contributing factors. Nonetheless, a priapism event >36 hours in duration is considered an emergency and requires immediate intervention for detumescence and pain relief, although it is unlikely that this patient population will have any meaningful spontaneous erections.\textsuperscript{11} The clinician should counsel the patient that surgical interventions, while effective at achieving detumescence, are likely to result in post-operative ED especially in men with acute ischemic priapism of >36 hours.

7. In patients presenting with a prolonged erection of four hours or less following intracavernosal injection pharmacotherapy for erectile dysfunction, clinicians should administer intracavernosal phenylephrine as the initial treatment option. (Expert Opinion)

In contrast to true acute ischemic priapism, prolonged erections, which are <4 hours in duration and occur following ICI pharmacotherapy for ED, are arguably much more common and may be managed differently than acute ischemic priapism. The physiology of prolonged erections versus acute ischemic priapism is also distinct, as the latter often represents conditions where cloting has occurred and true tissue ischemia (with impaired smooth muscle function and impaired oxygenation) has begun. Prolonged erections frequently occur following deformity assessments, following PDUS for ED, following ICI training for ED therapy, or following one of several intracavernosal therapies. It is important to recognize that there are very few studies which have been published on this topic, with no high level (i.e., RCTs) studies available to inform recommendations or guidelines. As such, the following commentary should be considered Expert Opinion.

Prior to initiating treatment, it is important to differentiate conditions which require therapy versus those which may be reasonably observed. Men with prolonged erections that are not fully rigid are less likely to later progress to acute ischemic priapism compared to those with fully rigid erections. As such, partial erections should likely not be counted towards the four-hour time criteria. Similarly, the specific medication used to achieve the erection is an important factor to consider. Men treated with alprostadil alone are less prone to progress to ischemic priapism compared to those treated with papaverine and phentolamine, which may counteract normal pathways of detumescence. Pain is also not likely a helpful indicator, as many men may experience pain relating to the injection medication or pain from full engorgement. Ultimately, clinical judgment is required to determine if any specific therapy is warranted versus additional observation.

Men with prolonged erections <4 hours who are deemed candidates for treatment should be considered for an injection of intracavernosal phenylephrine as a primary treatment option. See Appendix A for guidance on dosing and administration of phenylephrine. Rationale for the use of phenylephrine over other sympathomimetic agents and specific dosing are discussed in Statement 9. Intracavernosal aspiration and irrigation likely represents too aggressive of a therapy for this specific clinical scenario to be used as a first-line therapy. Additionally, the physiologic rationale for aspiration and irrigation is to remove intracavernosal clots and permit entry of fresh blood in an attempt to restore smooth muscle function and vascular drainage. As the pathologic state of intracavernosal clotting and ischemia likely is not present with prolonged erections <4 hours, aspiration and irrigation is rarely warranted. However, persistent, prolonged erections may be considered for aspiration and irrigation if phenylephrine alone is unsuccessful. See Appendix B for guidance on aspiration and irrigation.

Other therapies are commonly used to treat prolonged erection, including ice compresses, laying supine, ejaculation, and oral medications such as pseudoephedrine. There may be some basis for these therapies, however, in the absence of any clinical data demonstrating efficacy, the Panel is unable to endorse their routine use (see Statement 8). As such, these treatments may be discussed but should not be used in lieu of more established therapies.
In contemporary practice, prolonged erections often present in distinct ‘virtual’ clinical settings, including during telephone conversations, text messages, and other similar scenarios. It is the Panel’s opinion that these must be managed using the clinician’s best judgment and may lead to recommendations of observation with status updates, oral or topical therapies (e.g., pseudoephedrine, ice), urgent return to clinic with anticipated phenylephrine injection, or referral to the emergency department.

8. In a patient with diagnosed acute ischemic priapism, conservative therapies (i.e., observation, oral medications, cold compresses, exercise) are unlikely to be successful and should not delay definitive therapies. (Expert Opinion)

As acute ischemic priapism represents a true time-sensitive emergency, ineffective therapies that delay resolution are ill-advised. This remains true for events secondary to sickle-cell disease, pharmacotherapy, or other etiologies. No evidence-based recommendations can be made on self-help strategies involving exercise, cool or warm compresses, oral hydration, or masturbation. Likewise, oral pharmacotherapy is not recommended for management of acute ischemic priapism. Experts have highlighted that the minimal corporal blood flow characteristic of this condition would preclude efficacy of oral agents. Additionally, these drugs may place patients at risk, as seen with the numerous reports of toxicity from oral pseudoephedrine when used to treat priapism.

Prior work has shown that for erections induced by intracavernosal alprostadil, oral pseudoephedrine was not better than placebo for achieving resolution. Although terbutaline appeared more effective than placebo, it was not significantly better than pseudoephedrine. Subsequent work disputed any value of various doses of terbutaline relative to placebo and noted that this drug has actually been shown to induce erections. The lack of efficacy for achieving a prompt response is expected based on bioavailability studies. At 30 minutes following a 10 mg dose of oral terbutaline, serum concentration is zero. It reaches 1 ng/mL at one hour, and peak concentration at six hours. Additionally, peak levels will be much lower in non-fasting subjects.

Pre-Surgical Management of Acute Ischemic Priapism

9. Clinicians should manage acute ischemic priapism with intracavernosal phenylephrine and corporal aspiration, with or without irrigation, as first line therapy and prior to operative interventions. (Moderate Recommendation, Evidence Level: Grade C)

Given the emergent nature of acute ischemic priapism, ICI with phenylephrine should begin as rapidly as possible following diagnosis. Specifically, intracavernosal treatments should not be delayed due to other systemic therapies (e.g., hydration, exchange transfusion), but may be administered concomitantly in most cases. When a decision must be made between systemic and intracavernosal treatments, intracavernosal therapy should take precedence in the majority of cases.

While efficacy has been reported for epinephrine and ethylephrine, the most frequently used agent is phenylephrine. As no other injectable agent has a comparable sample size within the literature, phenylephrine was compared to all other agents combined and found to have a 28% higher rate of detumescence, while other agents appeared comparable to aspiration alone. Although use in this context is off-label, phenylephrine is recognized as the preferred agent of choice. It offers rapid onset, short duration of action, and alpha-1 selectivity is attractive for reducing the potential for adverse cardiovascular events. See Appendix A for guidance on dosing and administration of phenylephrine.

Corporal aspiration refers to the intracavernosal placement of a needle followed by withdrawal of corporal blood. Irrigation indicates subsequent instillation of fluid (typically saline) into the corpora. These two procedures are often combined to remove clotted, deoxygenated blood and restore arterial flow and smooth muscle and endothelial function. They may be performed alone or combined with instillations of phenylephrine. See Appendix B for guidance on aspiration and irrigation.

Although a modest amount of data exists regarding various ICI therapies, the Panel was unable to identify any studies that specifically compared aspiration and irrigation with saline to alpha adrenergic injections alone. Rather, several studies reported outcomes on
the combination of aspiration, irrigation, and alpha adrenergics. Overall results demonstrate successful detumescence in 71–93% of cases, with durations of priapism ranging from 5 to 104 hours (mean durations 10–22 hours). Two studies reported post-treatment erectile function and noted overall preservation in 70–92% of patients, with longer durations of priapism associated with worsened long-term function.

In comparing outcomes data between combination therapy of aspiration, irrigation, and intracavernosal alpha adrenergics to alpha adrenergics alone, results appear to suggest greater resolution rates with combination therapy. Specifically, the need for subsequent shunt surgery was required in 15–28% of patients who received combination therapy compared to 43–63% of patients who received intracavernosal phenylephrine without aspiration and saline irrigation.

In evaluating aspiration and saline irrigation as solitary therapy, an RCT was performed to compare varying temperatures (10–37°C) of irrigation in men with iatrogenic priapism. Patients were treated with 25 mL instillations every 20 minutes until resolution or a maximum of 125 mL was administered. Men who received the coldest saline (10°C) experienced the highest rates of resolution (96% versus 60% in men with saline at 37°C). Those failing to detumescence were subsequently treated with ephedrine and achieved a complete response. Although the study population likely represents an easier to treat group (i.e., shorter duration, iatrogenic) compared to the typical emergency department patient, results suggest the potential benefits of using colder irrigation solutions and further support the additive benefits of combination therapy over aspiration and saline irrigation alone. It is noteworthy, however, that cold saline should not be used in men with sickle cell disease so as to avoid worsening / precipitating additional complications.

Based on the above data, clinicians treating ischemic priapism may elect to proceed with alpha adrenergics, or aspiration and saline irrigation, or a combination of both therapies based on their clinical judgment. However, given the relatively high-resolution rates, surgical shunting should not be performed until both alpha adrenergics and aspiration and saline irrigation have been attempted. Even in cases where preserved erectile function is unlikely, clinicians may elect to perform combined treatments to improve penile pain, if present. Intracavernosal therapies may be deferred when ED is anticipated, and expedited placement of a penile prosthesis is planned.

10. In patients receiving intracavernosal injections with phenylephrine to treat acute ischemic priapism, clinicians should monitor blood pressure and heart rate. (Clinical Principle)

Given the alpha-adrenergic effect of phenylephrine, systemic absorption following intracavernosal administration raises concerns for adverse cardiovascular effects, possibly through coronary vasospasm. Additionally, dosages are often calculated based on bedside preparations that may lack precision. Monitoring patients during and following treatment allows for detection of elevation in blood pressure, tachycardia, or reflex bradycardia.

The number of studies specifically reporting use of continuous monitoring are few, with even fewer commenting on numerical values. In most cases, there was no change in heart rate or blood pressure, but even when mild changes were detected, they were not found to be clinically relevant.

Although few in number, case reports have described adverse events such as myocardial infarction and intracranial bleeding following intracavernosal phenylephrine. However, some instances were questionable for causation based on the low dose of administered medication (i.e., 100 mcg) or excessive use of pseudoephedrine prior to presentation.

It is possible that phenylephrine doses higher than those suggested in prior guidelines may better facilitate prompt detumescence, especially in an acidic corporal environment. A need for less injections seems advantageous for patients and earlier resolution may also mean less physician fatigue factoring into a decision to proceed to shunting. One series featuring a median dose of 1000 mcg (500-2000 mcg) noted absence of adverse effects in all patients; however, none suffered from baseline coronary artery disease or peripheral artery disease, and no patient had a history of using monoamine oxidase inhibitors (MAOI). Another study featuring a median dose of 1500 mcg noted a decline in diastolic blood pressure and heart rate between admission and
discharge, but this was clinically insignificant and possibly confounded by change in pain level and overall clinical condition. Additionally, cumulative doses of 40-50 mg over 1-2 days have been reported without adverse outcomes.

Blood pressure and heart rate monitoring seems especially prudent in patients with a history of cardiovascular disease, hypertension, prior stroke, and those using medications such as MAOIs. Phenylephrine is a direct-acting sympathomimetic (alpha-1 selective) with end organ selectivity, and there are no reports of toxicity when used for priapism in men using MAOI. Potentiation of phenylephrine effects by prior administration of MAOI is most significant with use of oral phenylephrine, which is dissimilar from intracavernosal administration. When parenteral use of phenylephrine has been deemed necessary in patients on MAOI, recommendations have included use of low starting doses; as such, gradual dose escalation may be reasonable when treating priapism in men using these medications. Should blood pressure spike, this would be detected by monitoring and appropriate medical intervention could be performed.

Surgical Management of Acute Ischemic Priapism

11. Clinicians should perform a distal corporoglanular shunt, with or without tunneling, in patients with acute ischemic priapism who have failed pharmacologic intracavernosal reversal and aspiration, with or without irrigation. (Moderate Recommendation, Evidence Level: Grade C)

A surgical shunt should not be considered as first-line therapy. The decision to initiate surgery requires the failure of nonsurgical interventions. However, deciding when to end nonsurgical procedures and proceed with surgery will depend on the duration of the priapism. For acute ischemic priapism of extended duration, response to ICI of sympathomimetics becomes increasingly unlikely. Phenylephrine is less effective in priapism of more than 48-hour duration because ischemia and acidosis impair the intracavernous smooth muscle response to sympathomimetics. Under such anoxic conditions, phenylephrine produces poorly sustained phasic contractile responses. In particular, injection of sympathomimetics after 72 hours offers a lower chance of successful resolution and a surgical shunting procedure often is required to re-establish circulation of the corpora cavernosa.

Accordingly, when non-surgical interventions fail, a distal corporoglanular shunt should be considered. The optimal type of distal corporoglanular shunt (e.g., Winter’s, Al Gorab, Ebbehoj, T-Shunt) for the treatment of acute ischemic priapism has not been defined. Specifically, no studies have directly compared the various surgical approaches. The overwhelming majority of studies include small patient cohorts and are retrospective in nature, except for one prospective study that included 19 patients.

Similarly, there are no studies comparing shunting alone versus shunting with tunneling. Four studies reporting on various distal shunts with corporal tunneling, including the Burnett snake maneuver, demonstrate generally high rates of immediate success at relieving priapism. In five studies with pre- and post-treatment erectile function information, distal shunts, both with and without tunneling, demonstrate deleterious effects on erectile function. Use of tunneling, however, is associated with greater degradation of post-procedure erectile function compared to distal shunting alone.

Potential non-erectile complications of distal shunting and tunneling procedures include urethral injury, cavernositis, persistence of fistula, infection, and penile skin necrosis.

12. In patients with acute ischemic priapism who failed a distal corporoglanular shunt, clinicians should consider corporal tunneling. (Moderate Recommendation, Evidence Level: Grade C)

Distal corporoglanular shunts allow to relieve a compartment syndrome through evacuation of blood trapped within the corpora. As an adjunct to needle or scalpel-based opening of the distal end(s) of the corpora, instrument passage (typically a dilator) into the corporal tissue has been used to further facilitate drainage and detumescence. This is referred to as 'tunneling' or 'snaking'. This concept using surgical dilators to evacuate ischemic clotted blood from the proximal crus of the penis through a distal shunt aims to re-establish blood flow. The data to evaluate the utility of tunneling is very limited and of low quality. There are no RCTs or comparative studies, and observational studies preclude...
unbiased comparisons between distal shunts with and without tunneling.

Pooled data suggest that the addition of tunneling may afford slightly higher rates of successful detumescence. However, the success rates of studies without tunneling are driven lower by the poor results seen with Winter's shunts. Analysis of the literature has shown that scalpel-based shunts (e.g., Ebbehoj, Al Ghorab, Lue T Shunt) provide higher success than needle-based (i.e., Winter's) shunts. Another potential factor relevant to comparative success rates is duration of priapism prior to the intervention of interest. In one study of patients managed with tunneling, detumescence was achieved in 100%, 34%, and 0% of cases treated before 24 hours, at or beyond 48 hours, and at or beyond 96 hours, respectively.8

While all distal shunts may be contributory to future erectile function, the limited data suggests the insult of the dilator to the corporal tissue may be greater with tunneling.10, 12, 13 However, patient-related factors and duration of ischemia are confounders. Thus, it is unclear whether tunneling produces an insult detrimental to future ED that exceeds the risk of ischemic priapism itself. Complications including wound infections, fistula, skin necrosis, and gangrene have been reported for distal shunts, with and without tunneling, so it is unclear if the additional corporal disruption imparts greater risk.48, 59, 60

13. Clinicians should counsel patients that there is inadequate evidence to quantify the benefit of performing a proximal shunt (of any kind) in a patient with persistent acute ischemic priapism after distal shunting. (Moderate Recommendation, Evidence Level: Grade C)

Proximal shunts are optional for the surgeon, based on clinical judgment and comfort level. In general, it is the Panel’s opinion that proximal shunting represents a historical procedure and has largely been replaced by distal shunts with tunneling procedures. Several proximal shunting procedures have been described to address persistent priapism after failure or suspected failure of distal shunts, including Quackels (corpus cavernosum to spongiosum), Grayhack (corpus cavernosum to saphenous vein), and Barry (corpus cavernosum to deep dorsal vein) procedures. To evaluate the role and efficacy of these procedures, a systematic review was performed of all published literature from 1960 to 2020 where proximal shunts were performed after suspected failed distal shunts. A total of 17 observational studies were included (n=62 patients in total), of which two were moderate and 15 were low quality. Specific protocols for managing priapism varied among the studies, including different utilizations of aspiration, irrigation, and ICI therapy; specific distal shunt performed; and number of prior attempted shunts. Similarly, the study cohorts were very heterogeneous and included priapism durations ranging from 6-180 hours and sickle cell and nonsickle cell populations. With few exceptions, outcomes were not measured in a rigorous manner, with detumescence defined clinically and few studies utilizing the standardized IIEF to characterize erectile function postoperatively.

Results demonstrated an overall rate of successful priapism resolution in 76.6% of cases with similar rates among the various procedures. The majority of studies included outcomes of Grayhack and Quackel procedures (n=13 studies), one study utilized the Barry technique, and the remainder failed to report details of the specific procedure. With limited data, the duration of priapism did not appear to meaningfully impact the ability to achieve detumescence, with successful resolution achieved in 50%, 55.6%, and 60% of men who had priapism for 5-30 hours, 36-72 hours, and >72 hours, respectively. Older men were more likely to experience successful detumescence after the proximal shunt (63.6%, 60%, and 90% for 13 to 29 years, 30 to 44 years, and over 45 years of age, respectively).

Arguably, the two key objectives in achieving detumescence in men with priapism are to preserve erectile function and to reduce post-procedure pain. Using combined data from 12 studies (n=30 patients), and assuming best case scenarios in cases where the data were ambiguous (i.e., considering an ambiguous outcome as successful), only 27.5% of patients experienced preserved erectile function after proximal shunting.10, 48, 53, 54, 61-68 As with distal shunting, the duration since onset of priapism was a strong predictor of preserved erectile function. Limited data from 5 studies (n=12 patients), demonstrated a strong correlation between the time since onset of priapism and ultimate erectile function outcome (r=0.78, p<0.01, with one
outlier excluded). Using a 72-hour cut-point, all men with successful detumescence prior to this time experienced some degree of preserved erectile function compared to 40% with minimally preserved function beyond that time. These data would argue for more aggressive measures during the first 2-3 days of priapism, with declining benefits when performed beyond that time period.

Beyond the data presented, there are several important clinical considerations in deciding on whether a proximal shunt is appropriate and should be performed. One key issue is the ability to determine if detumescence has been adequately achieved following distal shunting. Particularly in men with more prolonged cases of priapism (>24 hours), edema, ecchymoses, and induration are often indistinguishable from persistent priapism. In this setting, and recognizing an absence of data, is the Panel recommends that a vascular study (such as a PDUS) or cavernosal blood gas should be performed prior to performing additional interventions (repeat distal or proceeding to proximal shunting). See Statement 14 for a detailed discussion on imaging.

The Panel also recognizes the significant lack of data on proximal shunts. As noted previously, the entirety of published literature available over the past 60 years includes only 62 patients. This paucity of data suggest that proximal shunting procedures are likely rarely performed in contemporary and historical clinical practice. As such, there are likely no surgeons who have extensive experience in this area, and broader training and education on methods of optimizing outcomes are therefore not possible. Additionally, the extent and rate of complications from proximal shunting is understudied and could potentially lead to significant comorbidities such as urethrocutaneous fistulae, urethral strictures, or other similar issues. From a practical standpoint, such limited data would typically relegate a procedure to experimental status. Additionally, some of the described procedures require distinct skillsets outside of a general urologist’s training, including performing vascular anastomoses to the saphenous or dorsal penile vein.

With the above recognitions, the Panel suggests that the decision to proceed with a proximal shunt should be based on several factors, including the surgeon’s comfort level with the procedure, patient age and pre-operative erectile function, and duration since onset of priapism. Additionally, a proximal shunt should only be considered after failure of more established, conservative procedures, including distal shunting with tunneling. Using these criteria, in situations when surgeons are uncomfortable performing proximal shunts, in the case of older patients, those with poor erectile function at baseline, and men with priapism duration >72 hours, observation or placement of a penile prosthesis may be preferred in lieu of a proximal shunt. Additionally, because of the above-mentioned limitations, the Panel consensus is that proximal shunting should not be considered a mandatory procedure for men who have been confirmed to have failed distal shunting but rather one of several treatment options which may be considered.

**Post-Shunting Management of Acute Ischemic Priapism**

14. In an acute ischemic priapism patient with persistent erection following shunting, the clinician should perform corporal blood gas or color duplex Doppler ultrasound prior to repeat surgical intervention to determine cavernous oxygenation or arterial inflow. (Moderate Recommendation, Evidence Level: Grade C)

In cases where a patient is refractory to shunting, subsequent intervention may be necessary. In this scenario, the clinician must perform a confirmatory test to assess penile hemodynamic characteristics and extent of necrosis/fibrosis to inform secondary treatment decisions and should not base further surgical decisions based on exam alone. The Panel acknowledges this is a complex scenario; therefore, corporal blood gas or imaging should be utilized following shunt procedure to differentiate persistent ischemic priapism from reactive hyperemia or conversion to non-ischemic priapism. Evaluating the status of a patient with refractory priapism is particularly important in the event that a patient is referred from another institution and/or the clinician is seeing a patient who had been previously treated elsewhere and a complete patient history may not be available.

Penile corporal blood gas is easily performed and should be utilized in patients when the clinician must establish cavernosal oxygenation status post-shunting. This can help with decision making in proceeding to additional surgical procedures including placement of an
immediate penile prosthesis.

The role of imaging is a diagnostic intervention in the management of ischemic priapism, particularly in patients who require assessment of arterial inflow during an acute ischemic event. In the diagnostic phase, imaging ascertains the nature of blood flow to the corpora cavernosa and determines whether the patient has either acute ischemic or non-ischemic priapism. In a diagnosed acute ischemic priapism patient who has undergone a distal shunt, with or without tunneling, post-procedural imaging can determine shunt patency by showing restoration of cavernosal arterial inflow.

Much of the data that examines the use and accuracy of different imaging techniques on priapism patients is indirect (i.e., assessing pre-procedure integrity and viability of penile tissue, and is not powered to study the accuracy of imaging techniques in patients who have failed shunting surgery and are therefore candidates for further intervention. However, one study by Chiou et al. retrospectively reviewed charts of 24 patients who presented with priapism, 11 of whom were referred from other institutions and were refractory to previous aspiration and ICI therapy (n=2), distal (n=8), or proximal (n=1) shunts. PDUS at presentation showed no detectable cavernosal arterial flow in any of the patients, verifying earlier interventions had failed. Distal shunts were placed in all 11 patients; 12 post-operative PDUS studies in 8 patients were performed, revealing patency in all patients was achieved.

In a retrospective chart review of 52 priapism patients, von Stemple et al. used PDUS of acute ischemic (n=42) and non-ischemic (n=10) priapism patients and compared the results against each other and against tissue biopsy to assess the accuracy of imaging. The acute ischemic priapism patients had either failed aspiration and irrigation but had not yet undergone shunt surgery (n=14) or had failed a previous shunt (n=22), or had not yet undergone intervention (n=6). Most of the acute ischemic patients (n=37) had biopsy samples taken at the time of surgical intervention and were analyzed for fibrosis or necrosis and provided a measure of PDUS diagnostic accuracy. PDUS results in the non-ischemic and acute ischemic patients who either failed conservative therapies, or had not had any interven-

Acute Ischemic Priapism

tions, appeared to be predictive and accurate, however the results were mixed in ischemic patients who failed shunt placement. Of this latter group, PDUS results were accurate and showed classic ischemic patterns in nine patients; however, in 13 patients, results overlapped between ischemic and non-ischemic parameters and could not reliably predict clinical outcome. Histologically, only three patients showed normal tissue with the remaining showing varying degrees of fibrosis. When compared against PDUS results, there was poor correlation between blood flow and histological outcomes, leading the authors to conclude that MRI might be a better alternative than PDUS to assess for smooth muscle viability/necrosis (see below) prior to repeat surgical interventions.

Ralph et al. examined the use of MRI to assess penile tissue integrity in 23 patients and compared these results against histological analysis of corpora cavernosa biopsy, which were used as the gold standard comparator. Fifteen patients had an MRI of the penis but did not undergo biopsy. In the 23 patients who also underwent biopsy of the corpora cavernosa, results indicated that MRI was 100% sensitive to predict nonviable tissue. Of the 15 patients who chose not to undergo biopsy or further treatment, use of MRI showed nonviable tissue in 10 patients. Of these 10 patients, seven ultimately developed ED. MRI showed viable corporal tissue in the remaining five patients whose priapism resolved and who ultimately maintained erectile function. The accuracy of MRI has yet to be broadly established as a diagnostic tool in priapism patients; however, these data show that MRI is reliable in predicting nonviable smooth muscle tissue and may be used by the clinician to counsel patients about subsequent corporal fibrosis and ED.

PDUS has traditionally been used to assess blood flow, however the accuracy is limited, particularly in patients who have undergone previous procedures. It can be difficult to interpret, illustrated by von Stemple, and may be inaccurate for acute ischemic priapism patients, especially in the acute setting when qualified personnel with appropriate expertise are lacking. However, PDUS been shown to be effective in assessing blood flow in many clinical conditions and is an option in a diagnostic setting to differentiate between acute ischemic and non-ischemic priapism. Unfortunately, its use is limited by the number of specialists who can currently perform the procedure. Furthermore, in the emergency depart-
While the role of MRI has yet to be definitively established to assess corporal blood flow, it is an alternative imaging modality with diagnostic utility which can be used in the acute setting. MRI has excellent accuracy to assess corporal tissue necrosis and fibrosis and can be used as an adjunct to counsel patients about subsequent erectile function recovery following acute ischemic priapism. The data presented in this guideline suggest MRI findings can be used to counsel patients about preceding to immediate penile prosthesis (see Statements 15 and 16) due to destruction of corporal smooth muscle which correlates with erection recovery.

Penile Prosthesis

15. Clinicians may consider placement of a penile prosthesis in a patient with untreated acute ischemic priapism greater than 36 hours or in those who are refractory to shunting, with or without tunneling. (Expert Opinion)

Although most reported cases of acute ischemic priapism resolve with bedside management, some will require surgical intervention. Shunting, with or without tunneling, may provide detumescence for many patients, but some will be refractory. Even when patients respond to shunting, they will often experience impotence secondary to ischemia and resultant fibrosis, and potentially from the surgical intervention(s) as well. Men in need of detumescence for pain relief and in those hoping to optimize erectile performance in the future can be considered for placement of a penile prosthesis.

Decisions regarding placement of a penile prosthesis in a patient with acute ischemic priapism must be made after weighing multiple factors. These include, but are not limited to, the quality of the history provided relative to duration of persistent priapism, overall condition of the patient, health literacy and comprehension, and physician experience. Perhaps due to the complex nature of such decision-making, there are no RCTs relevant to this pathway. The available data suggests that prostheses placed in the setting of acute ischemic priapism are highly effective in providing detumescence, relief of pain, preservation of penile length, return to sexual activity, and overall satisfaction. Infection rates were below 10% for all studies reviewed.

In theory, avoiding disruption of the distal tunica when the chance of priapism resolution is extremely low may prove advantageous for subsequent penile prosthesis placement. In the work by Zacharakis et al, less than half of the men who received a penile implant within 17 days of priapism onset had undergone prior distal shunting. However, infection (7%) and erosion (3%) were unique to this cohort. The authors noted that distal perforation can occur in up to 6% of patients who have undergone previous shunt surgery. Of the men who received inflatable devices in delayed fashion (median: 5 months), 80% required narrow base cylinders. In a separate multicenter study with less patients, 40% of men with prior distal shunts undergoing penile implant placement required narrow base cylinders, and 20% needed subsequent explantation for distal erosion.

One center has shown complete concordance between radiologist-based determination of non-viable corporal tissue on pre-operative penile MRI and the presence of smooth muscle necrosis on intraoperative biopsy. The same group has also reported that ischemic priapism in excess of 36 hours is invariably associated with corporal fibrosis and ED. Thus, results of imaging in those with prolonged priapism may assist patient counseling. Likewise, if the prospects of functional recovery are dramatically low, clinicians may wish to weigh and consider the potential detriment of distal shunting for patients who may elect subsequent implant placement.

16. In a patient with acute ischemic priapism who is being considered for placement of a penile prosthesis, clinicians should discuss the risks and benefits of early versus delayed placement. (Moderate Recommendation, Evidence Level: Grade C)

Once it has been established that a patient suffering from acute ischemic priapism is a candidate for a penile prosthesis, either because other interventions have failed or the timeline suggests function is not otherwise salvageable, they should be counseled about factors relevant to the timing of device placement.

The Panel identified eight primary non-comparative studies addressing immediate insertion.
and eight which addressed delayed insertion. Most involved small patient populations. Definitions of ‘early’ and ‘late’ varied by reporting institutions, but those undergoing placement after failed shunting were generally deemed ‘late’. For immediate or early placement, duration of priapism ranged from 2 to 720 hours, whereas mean duration in delayed studies ranged from 33 hours to 10.5 months. Early placements more often involved malleable devices, whereas malleable and inflatable versions were more evenly distributed in delayed placement studies. Etiologies varied and were similarly distributed across the grouped studies.

Only one study provided comparative data of early versus delayed penile prosthesis placement. Results demonstrated that patients undergoing delayed placement were significantly more likely to report penile shortening and to undergo revision surgery. Similarly, the delayed group had a higher rate of infection (19% versus 7% for early placement). All cases of erosion and device malfunction were unique to the delayed group and satisfaction was higher for the early placement group (96% versus 60% for delayed placement).

When all data were considered, the reoperation rate was similar for early and delayed placement, and rates of erosion, malfunction or failure, and penile curvature were low for all patients. However, infection rates and penile shortening were higher for delayed placement, and length was related to patient satisfaction.

Clinicians should consider all items of relevance before proceeding with a penile prosthesis in a patient with priapism. Repetitive bedside irrigation procedures may, in theory, increase the chances for bacterial entry into the corpora that could threaten an implant with infection. Distal shunts may have compromised the integrity of the tunica albuginea that would surround an implant, possibly predisposing to erosion. Patients may not be in optimal condition for an implant due to status of comorbid conditions (e.g., diabetes) or use of problematic medications (e.g., anticoagulants, immunosuppressants). The urologist involved for management of priapism may lack the experience, comfort level, or materials to render device placement practical and/or possible.

Conversely, allowing fibrosis to mature within the corporal bodies may render them difficult or impossible to dilate, possibly necessitating use of shorter and/or narrower devices than what may have been feasible earlier in the disease process. The need for aggressive maneuvers may also increase the likelihood for inadvertent corporal and/or urethral perforation.

Future Directions

Priapism remains an understudied area of sexual medicine, with several areas of future research required:

- Basic translational science of the pathophysiology of priapism to identify the most effective therapeutic targets.
- Preventative medical and interventional strategies for stuttering priapism, especially in the sickle cell population.
- Identifying the timeline of acute ischemic priapism and permanent corporal fibrosis with subsequent erectile dysfunction in various clinical and etiologic settings.
- Defining risks and benefits of penile prosthetics placement in acute ischemic priapism, including patient reported outcomes, complications, prosthesis durability, and role of malleable versus inflatable devices.
- Methods of controlling thrombosis, including preserving shunt patency.
- Comparisons of surgical techniques: distal versus penoscrotal approaches to distal shunts; distal shunting with or without tunneling.
- Comparison of embolization techniques and materials, including short- and long-term outcomes including patient reported outcomes.
- Comparative, prospective protocols for both acute ischemic and non-ischemic priapism management to better identify optimal management strategies.
- Identifying a role of sexual health counselor in patients with acute ischemic priapism undergoing surgery and how this affects short- and long-term mental health.

As noted above, there are numerous areas where additional research is warranted to improve our understanding and treatment of priapism. Fundamental basic science investigations are necessary to identify pathophysiologic mechanisms and potential treatment targets. The enhanced understanding of mechanisms and pathways of priapism would allow for new pharmaco-
logic treatment strategies to prevent and terminate priapism early in its course. Although a base-level understanding of disease mechanisms currently exists with priapism in general, more nuanced evaluations and research separating subtypes of priapism (e.g., ICI-induced, oral medication-induced, sickle-cell, idiopathic) may provide for a more customized treatment approach. This is particularly relevant with cases of stuttering priapism, where management includes not only the acute phase but also long-term prevention strategies. Research in this area may expand to include the study of the sleep cycle, neurologic perturbations, and ‘backward engineering’ from medications which have shown some efficacy, including baclofen, antiandrogens or anxiolytics, among others.

Another critical question which remains outstanding relates to the timeline and progression of irreversible corporal damage related to priapism. The issue is further challenged by inaccuracies of estimated duration, possibility of intermittent periods of complete or partial priapism, underlying health of the corporal tissue (i.e., patient age, prior ED, comorbid conditions), prior episodes of priapism, various subtypes (e.g., sickle cell), and interventions performed. For example, a 50-year-old diabetic patient with persistent, untreated priapism lasting 72 hours likely will have a different outcome compared to an 18-year-old sickle cell patient with episodic priapism of a similar duration. This is particularly relevant as providers consider earlier definitive interventions such as placement of a penile prosthesis, wherein confidence is required that spontaneous recovery of erectile function is not possible. Future research into imaging studies, biopsies, adjunctive laboratory testing, or other modalities may help to better inform these decisions. However, at the present time, data are clearly lacking to quantify the true risks and benefits of early, definitive surgical interventions including distal shunting and prosthesis placement in men with acute ischemic priapism.

A third area where future research may benefit outcomes is with anti-thrombotic therapies. As prolonged priapism is associated with cavernosal thrombosis, these therapies may have roles in both the early and late phases of treatment. Specifically, further research is required to determine if anti-thrombotics reduce the frequency of stuttering priapism, minimize the extent of ischemia in active priapism, and/or prevent closure of surgical shunts. Currently, there are very limited data on these topics, however, given the pathophysiology of priapism, the ability to control or regulate corporal thrombosis has inherent appeal.

Finally, significantly more research is required comparing various treatment strategies. Because priapism is an unpredictable and rare event, nearly all research reports are retrospective in nature and do not include comparison groups. Prospective, comparative protocols are warranted to better define optimal treatment approaches. These may include differing surgical techniques (e.g., proximal versus distal approaches, tunneling versus no tunneling, specific methods of shunting); preventative medications; agents and protocols for embolization; imaging modalities; customized algorithms based on etiology and clinical factors; and efficacy of conservative therapies. Outcomes-based assessments and longer-term follow-ups are also merited, as it is not uncommon to see restoration of excellent erection post priapism management in one setting, while another results in clustered recurrence of priapic episodes in another. Although the ideal research protocol would include development of a national priapism registry, in its absence, ambitious clinicians and scientists should consider beginning an institutional database tracking priapism patients and outcomes with pre-defined protocols and standardized follow-up assessments. The development of such protocols would be expected to greatly enhance our understanding of priapism and help provide the data necessary to further refine the next set of guidelines.
### Abbreviations

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<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>AUA</td>
<td>American Urological Association</td>
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<tr>
<td>CT</td>
<td>Computerized tomography</td>
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<tr>
<td>ECRI</td>
<td>Emergency Care Research Institute</td>
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<td>ED</td>
<td>Erectile dysfunction</td>
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<tr>
<td>GRADE</td>
<td>Grading of Recommendations Assessment, Development and Evaluation</td>
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<tr>
<td>ICI</td>
<td>Intracavernosal injection</td>
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<td>IIEF</td>
<td>International index of erectile function</td>
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<td>MAOI</td>
<td>Monoamine oxidase inhibitors</td>
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<td>MRI</td>
<td>Magnetic resonance imaging</td>
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<td>PDUS</td>
<td>Penile duplex Doppler ultrasonography</td>
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<td>PGC</td>
<td>Practice guidelines committee</td>
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<td>QUADAS</td>
<td>Quality assessment of diagnostic accuracy studies</td>
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<tr>
<td>RCT</td>
<td>Randomized controlled trial</td>
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<tr>
<td>SMSNA</td>
<td>Sexual Medicine Society of North America</td>
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References


25. Fuselier HA, Jr., Allen JM, Annaloro A et al: Incidence and simple management of priapism fol-


American Urological Association (AUA) / Sexual Medicine Society of North America (SMSNA)


Appendix A

Dosing and Administration of Phenylephrine

The optimal regimen for phenylephrine dosing, frequency, and method of administration has not been clearly defined in the scientific literature. As such, the recommendations which follow are all based on expert opinion and recommendations. Clinicians should consider blood pressure monitoring in men undergoing repeated injections and in those with underlying, relevant comorbid conditions (e.g., hypertension). Monitoring seems especially prudent in patients with a history of cardiovascular disease, hypertension, prior stroke, and those using medications such as monoamine oxidase inhibitors (MAOI). Phenylephrine is a direct-acting sympathomimetic (alpha-1 selective) with end organ selectivity, and there are no reports of toxicity when used for priapism in men using MAOI. Potentiation of phenylephrine effects by prior administration of MAOI is most significant with use of oral phenylephrine, which is dissimilar from intracavernosal administration. When parental use of phenylephrine has been deemed necessary in patients on MAOI, recommendations have included use of low starting doses, thus gradual dose escalation may be reasonable when treating priapism in men using these medications. Should blood pressure spike, this would be detected by monitoring and appropriate medical intervention could be performed.

Although there is no upper limit to the number of injections which may be performed, injections should be stopped if blood pressure changes are detected. Similarly, if the erection persists despite repeated attempts with injections and aspiration/irrigation over a period of one hour or more, the panel recommends proceeding with more definitive therapy (i.e., shunting procedure). Indeed, some clinical scenarios may be more appropriate for a more rapid transition to surgical procedures, without prolonged attempts at phenylephrine and aspiration/irrigation (e.g., priapism >36 hours).

**Dosing and instructions:**

- Phenylephrine 100-500 mcg doses suspended in 1 ml of normal saline (optimally premixed by pharmacy to minimize risks of miscalculation/overdose)
- Doses administered ≥5 minutes apart'
- Administered intracavernosally (not subcutaneously)
- Administered laterally (3 or 9 o’clock position) near the base of the penile shaft
- May be continued for up to 1 hour (see commentary above)
- Small needles may be used (e.g., 27G)
- Consider performing a penile block with local anesthetic prior to beginning
- In cases where the combination of phenylephrine and aspiration/irrigation are performed, aspiration should precede phenylephrine administration to permit fresh, oxygenated blood to fill the corpora and potentially improve the yield of phenylephrine administration
Appendix B

Sample Protocol for Aspiration and Irrigation:

The following protocol is one potential example of aspiration/irrigation with instillation of phenylephrine. However, this should not be considered the gold-standard approach, as there are currently no publications which have identified any method which is superior to another. Similarly, the decision as to when to stop performing aspiration/irrigation with phenylephrine will depend on clinical factors, including response to aspiration/irrigation and time since priapism onset, among others.

Steps for aspiration/irrigation with phenylephrine administration:

- Perform a penile block with local numbing medication (if not previously performed).
- Place a 16-18 gauge butterfly needle in the 3 or 9 o’clock position on the penis near the base.
- Connect the butterfly needle to a 30-60 cc Luer Lock syringe.
- Alternate between aspiration of blood clots and instillation of saline (chilled if available and if the patient does not have sickle cell disease) until some degree of detumescence can be achieved.
- Instill phenylephrine.
- Allow 3-5 minutes of time to pass.
- Repeat steps 4-6 until detumescence is achieved or until the decision has been made to proceed with surgical shunting.
- If temporary detumescence is achieved with aspiration followed by a rapid refilling of blood despite multiple attempts of phenylephrine instillation, consideration may be given to placement of a firm penile wrap at the time of aspiration to maintain detumescence.
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Acute Ischemic Priapism

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Disclaimer
This document was written by the Acute Ischemic Priapism Panel of the American Urological Association Education and Research, Inc., which was created in 2018. The Practice Guidelines Committee (PGC) of the AUA selected the commit-tee chair. Panel members were selected by the chair. Membership of the Panel included specialists in urology and emergency medicine with specific expertise on this disorder. The mission of the panel was to develop recommendations that are analysis based or consensus-based, depending on panel processes and available data, for optimal clinical practices in the treatment of acute ischemic priapism. Funding of the panel was provided by the AUA. Panel members received no remuneration for their work. Each member of the panel provides an ongoing conflict of interest disclosure to the AUA, and the Panel Chair, with the support of AUA Guidelines staff and the PGC, reviews all disclosures and addresses any potential conflicts per AUA’s Principles, Policies and Procedures for Managing Conflicts of Interest. While these guidelines do not necessarily establish the standard of care, AUA seeks to recommend and to encourage compliance by practitioners with current best practices related to the condition being treated. As medical knowledge expands and technology advances, the guidelines will change. Today these evidence-based guidelines statements represent not absolute mandates but provisional proposals for treatment under the specific conditions described in each document. For all these reasons, the guidelines do not pre-empt physician judgment in individual cases. Treating physicians must take into account variations in resources, and patient tolerances, needs, and preferences. Conformance with any clinical guideline does not guarantee a successful outcome. The guideline text may include information or recommendations about certain drug uses (‘off label’) that are not approved by the Food and Drug Administration (FDA), or about medications or sub-stances not subject to the FDA approval process. AUA urges strict compliance with all government regulations and protocols for prescription and use of these substances. The physician is encouraged to carefully follow all available prescribing information about indications, contraindications, precautions and warnings. These guidelines and best practice statements are not intended to provide legal advice about use and misuse of these substances. Although guidelines are intended to encourage best practices and potentially encompass available technologies with sufficient data as of close of the literature review, they are necessarily time-limited. Guidelines cannot include evaluation of all data on emerging technologies or management, including those that are FDA-approved, which may immediately come to represent accepted clinical practices. For this reason, the AUA does not regard technologies or management which are too new to be addressed by this guideline as necessarily experimental or investigational.