REVIEW ARTICLE

Erectile Dysfunction for the Family Physician

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Abstract: Erectile dysfunction (ED) is a relatively important issue in men's health that warrants further discussion and consideration amongst primary care physicians. While it was originally believed that the underlying mechanism of ED was more psychogenic in nature, over the last 40 years such thinking has been abandoned in favor of investigating underlying organic causes as the primary contributor. The causes of ED are varied and it is clear that there are a multitude of medical comorbidities that contribute to it, including diabetes, hypertension, vascular disease, chronic kidney disease, etc. More candid conversations need to be had between primary care physicians and their male patients regarding symptoms of ED, especially after the age of 40.

INTRODUCTION

Erectile dysfunction, more commonly referred to as ED and previously termed impotence, is a medical condition defined as the persistent inability to achieve and/or maintain an erection that is satisfactory and sufficient for sexual performance.[1,2,3,4] In 1992, the NIH Consensus Development Panel on Impotence defined erectile dysfunction in very similar terms.[5,6] In most cases, ED in patients presenting to the family medicine physician is of an acquired nature – i.e. the ED began after a period of normal erectile function the remainder, those who suffer ED from the outset of sexual desire, is beyond the scope of this article.[2] While these definitions seem to allow for considerable subjectivity from person-to-person, it is clear from the research on this subject that ED has a significantly higher prevalence among males aged 40 to 90 years old 70% of men greater than 70 years of age suffer from ED while only 5% of men younger than 40 years of age express such difficulty.[2] Family medicine physicians should feel comfortable enough distinguishing ED from other male sexual dysfunctions that may include libido issues, ejaculatory disorders, or infertility.[5] This review article focuses primarily on the issues involving male ED, as it applies to family medicine physicians.

STATISTICS

While many men in the United States 43% will report erectile difficulty as a sexual problem at some point during their lifetime, it is evident that ED becomes more of an issue in the aging male population. Notably, statistics indicate that by the year 2025, nearly 322 million men worldwide will report some degree of ED.[3,7] The Massachusetts Male Aging Study MMAS, a cross-sectional survey of a randomly selected group of nearly 1,300 males in the Boston region initiated in 1987 and ending in 1989, helped shed further light on the subject of ED.[6] This study discovered an overall occurrence of 52% for any degree of ED in men aged 40 to 70 years old. Incidence was found to increase nearly 12.5% in the decade of life 40-49 years of age, and an even higher incidence 46.4% was discovered in men between the ages of 60-69 years of age.[5] This statistical analysis revealed a directly proportional increase in the incidence of ED with advancing age. Similarly, the Health Professionals Follow-Up Study, a study that began in 1986 with follow-up surveys mailed to participants every two years thereafter, surveyed approximately 31,000 health care professionals and found the prevalence of ED to be 33% for those men between the ages of 53 and 90 years of age.[5,8] Perhaps surprisingly, most of these statistics suggest that ED is not as uncommon as many might think, and therefore, this condition warrants further clinical consideration by primary care physicians in the outpatient setting. Yet, knowing all this, ED can still go undetected by the physician as a result of male patients not actively pursuing a dialogue with their physicians when such issues arise. Table 1 *(page 26)* highlights some of the more commonly reported reasons as to why men do not seek out medical attention regarding ED.

PATHOPHYSIOLOGY

In order to understand the pathological process that encompasses ED, it is primarily essential that one understands the normal processes regarding male sexual function, more specifically, the physiology of male penile erection.

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TABLE 1:

Some Commonly Reported Reasons Why Men Do Not Seek Medical Attention Regarding ED

Perception that lack of complete erection is a part of normal aging processes	Sexual inactivity as a result of widowhood
Not perceiving ED as a medical disorder	Perception of a lack of effective treatment options
Ashamed to discuss sexual issues with doctor	

TABLE 2:

Risk Factors Associated with ED

Age	Cardiovascular Conditions
Metabolic Conditions	Lifestyle Habits
Chronic Kidney Disease	Medication Induced Side-effects
Hormonal (low testosterone)	Obesity
Psychological	

Male erection is a complex process that entails vascular, hormonal, neurological, and psychological components.[2] In the simplest context, it is external stimuli via autonomic and somatic pathways that help provoke penile erection. From a neuroanatomy perspective, we recognize the autonomic nervous system as being comprised of two divisions, the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). However, in consideration of normal male erectile function, it is the pathway of the PNS and its activation that we are most concerned. From an osteopathic viewpoint, the autonomic pathways involved in normal penile erection are localized predominantly to sacral spine regions S2-S4 and are aligned with the parasympathetic nervous system. Nerve fibers of the inferior hypogastric plexus, comprised of parasympathetic fibers from the pelvic splanchnic nerves S2-S4 region and sympathetic fibers from the lower thoracic and upper lumbar region T12-L1, can be further subdivided into what is termed the prostatic plexus. The prostatic plexus itself contains both sympathetic nerve fibers responsible predominantly for ejaculation and parasympathetic nerve fibers formed predominantly from the pelvic splanchnic nerves that are responsible for normal penile erection.[9]

It is upon activation of PNS pathways that nitric oxide (NO) is released from penile cavernous nerves and endothelial cells. This release of NO is what leads to penile cavernosal smooth muscle relaxation, a decreased peripheral arteriolar resistance, and, subsequently permits influx of blood into the penile cavernosum with concomitant decrease in venous drainage resulting in penile erection.[2] It is when these normal processes are disrupted, that ED is eventually perceived and then reported by the patient.

RISK FACTORS

Several studies have suggested possible associations between other comorbid health conditions and ED see Table 2. Some of the more considerable risk factors and comorbid conditions will be discussed here.

Age

As previously mentioned in this article, statistics reveal a modest increase in acquired ED beginning in the fourth decade of life and beyond.[2] This relationship seems to be directly proportional in nature –i.e. the older the male individual is, the more likely he is to suffer from ED. This relationship has been discussed more than once in literature.[4,6,7,8] It is likely that these results are mostly attributable to the development of other comorbid disease states as the individual ages. Some of the more commonly observed comorbidities will be discussed in further detail below. In addition, aging itself has been attributed to natural declines in circulating testosterone levels that can have a negative effect on normal erectile function.[10] See also section on Hormonal.

Cardiovascular

Medical literature provides ample documentation regarding traditional cardiovascular risk factors, specifically hypertension and dyslipidemia, as they relate to acquired ED. The more pivotal question that may arise is that of which came first. More recently, evidence suggests that acquired ED, in the right clinical scenario, may actually be an early indicator of ASCVD. Indeed, ED may precede the onset of identifiable cardiovascular events by as many as 3 to 5 years.[11] Clinical trials have demonstrated the presence of vascular disease in men suffering from ED even without the more traditional risk factors of hypertension, dyslipidemia, diabetes, etc.[12] Other studies also point to a relationship between ED and coexisting cardiovascular disease.[12] It has also been discovered that the increase in prevalence of acquired ED with age is followed by the development of atherosclerotic plaque lesions in the systemic vascular beds.[12] Prior prospective studies have indicated that in men without known CVD, those with ED have a higher risk of grouped CVD outcomes including CHD, stroke, PVD, and all-cause mortality as compared to those men without ED.[13]

Such evidence suggests that family medicine physicians should consider routinely screening their male patients for ED, especially those males 40 years of age or older, in light of assessing future cardiovascular risk.

Metabolic

Just recently, the United States Preventative Services Task Force has recommended grade B recommendation that overweight or obese adults between the ages of 40 and 70 years of age be screened for abnormal elevations in blood glucose levels in an attempt to detect metabolic syndrome and diabetes earlier. Diabetes is a condition that if left untreated or undertreated, can lead to catastrophic outcomes for patients. Some of the complications

attributed to diabetes include retinopathy, nephropathy, neuropathies, and limb infections with potential limb amputation, as well as myocardial infarction. While we certainly recognize these as the more notable complications of diabetes, diabetes has also has been implicated in the development of acquired ED. Further investigation reveals that ED is more common in men with diabetes than in men without diabetes.[14] Statistics indicate a prevalence of anywhere between 35-75% in diabetic males as compared to 26% in the general population.[14] Men with diabetes are also more likely to experience problems with acquired ED 10 to 15 years earlier than their male counterparts without diabetes.[14] Given the rather epidemic number of newly diagnosed diabetics annually, it may be prudent to screen adult diabetic males earlier on for signs of ED. Since diabetic males are more likely to experience ED than their non-diabetic counterparts, it is befitting for clinicians to screen males for ED, especially after age 40, as this also may be an early indicator of diabetic disease.[15] The main mechanisms behind development of ED in diabetic males seem to be multifactorial and these changes do not occur suddenly, but rather, over the course of time. The factors that most often contribute to the development of ED in diabetic males are mainly neurologic usually comprised of autonomic neuropathy and vascular atherogenic in nature.[14]

Hormonal

Another topic that bears mentioning in the discussion of ED is male hormonal androgen deficiency or low testosterone (LT). The extent and breadth of this male-related problem is beyond the scope of this article, however, it is worth mentioning in the context of ED. More information has been gathered in recent years suggesting the direct effects of testosterone on penile tissues as they relate to erectile function. Testosterone deficiency results in decreased action of nitric oxide synthase and its production that has significant negative implications on normal erectile function.[3] Moreover, when phosphodiesterate-5 inhibitor PDE-5 pharmacotherapy is suboptimal in resultant effect for treatment of ED, testosterone replacement therapy has been found to positively augment the effectiveness of PDE-5 treatment.[3,16] While testosterone replacement therapy may play a role in the treatment of these individuals, one must also consider the potential for increased cardiovascular disease risk, heart attack and stroke, that is now known to be associated with testosterone replacement therapy. As it stands, approximately one-third of men with ED have some degree of hypogonadism, or LT.[10] It has been understood from an assortment of clinical studies that serum concentrations of testosterone decline with age in the male patient for a variety of reasons.[10,16,17] Synchronous elevation in the levels of circulating sex-hormone binding globulin (SHBG) with aging also contributes to the development of ED through a decreased bioavailability of free circulating testosterone.[17]

Lifestyle

Lifestyle choices can also play a significant role in the development of ED. Lifestyle and nutrition have a significant influence on the production of NO in vascular beds and subsequently, can affect normal male erection leading to the development of ED.[7] It is now widely known that a sedentary lifestyle combined with a poor diet often lead to deleterious cardiovascular effects including secondary obesity (see Obesity section). Limited physical activity attributes to the evolution of ED by diminishing individual cardiovascular fitness, contributing to an increase in endothelial dysfunction, increasing oxidative stress on the body, and may contribute to poor self-esteem and mental outlook.[7] Smoking, whether via direct use or via second-hand smoke exposure, also has deleterious effects on the body with known contributions to the development of ED. The mechanisms by which smoking promote ED are most likely related to its devastating negative effects on the vasculature, increased oxidative stress/damages, as well as decreased NO release.[18] The statistics regarding the relationship between smoking and ED are alarming. When non-smoking men were compared to men who smoked up to 10 cigarettes per day, the smoking group had a 27 percent greater chance of developing ED. Men who smoked greater than 20 cigarettes per day had at least a 65 percent greater likelihood of suffering from ED.[18] These numbers indicate a directly proportional link between the amount of cigarettes smoked and the development of ED.

Obesity

Excess adipocity also poses a risk for development of ED. The mechanisms by which adipocytes contribute to ED are probably related to hormonal effects more than anything else. It is known that adipocytes possess the capacity to peripherally convert testosterone to estrogen by aromatase, thereby reducing the free circulating amount of testosterone. Thus, the lowered circulating amount of testosterone can negatively effect normal erectile function contributing to development of ED. Since adipocytes function as endocrine cells, they also secrete adipocytokines and adipokines, with leptin being a primary constituent of these. Leptin receptors found in Leydig cells appear to have an inhibitory effect on the generation of testosterone.[16] Also, noted in obese males is a decrease in lutenizing hormone (LH) pulse that occurs, hence, reducing the magnitude of downstream production of testosterone from the testicles.[16]

Medication Induced

Ironically, some of the medications used to treat co-morbid conditions often associated with ED, more specifically, hypertension, can also contribute to the development of ED. This issue should be of concern to physicians due to the fact that some male patients may reduce or even discontinue their anti-hypertensive regimen without first notifying their physician because of the undesired side-effect of ED. Such behaviors can have adverse effects on a patient's blood pressure control, potentially leading to undesired cardiovascular outcomes such as chronic renal disease, myocardial infarction, or even stroke.

While there remains a lack of absolute evidence on the matter, it has been suggested that the two classes of anti-hypertensive medication that contribute the most to the development of symptoms of ED are earlier generation beta-blockers and thiazide diuretics.[4] Knowing this, it may be more prudent to periodically screen patients on such medications for ED in the appropriate clinical context. If ED is detected, it may be acceptable to modify anti-hypertensive therapy by using newer generation beta-blockers, such as nebivolol, ACE inhibitors, or ARBs in place of the earlier generation beta-blockers or thiazide diuretic, if deemed clinically appropriate and safe for the patient.

The use of chronic opioids in male patients suffering from ED is also a subset of patients worth mentioning here. Previous in-

vestigations of patients on long-term opioid therapy for chronic pain, specifically low back pain, have indicated increased need for awareness of co-morbid sexual dysfunction; including ED.[19] Other studies have described a link between male hypogonadism and chronic opioid use due to the suppressive effects of opioids on the Hypothalmic-Pituitary-Gonadal HPG axis.[19-25] The lower circulating bioavailable levels of testosterone subsequently contribute to development of ED (see section "Hormonal" under Risk Factors).

Co-morbid Psychological/Psychiatric Disorders

In the 1970's, Masters and Johnson described male impotence, what we now know is ED, as a predominantly psychogenic problem with less than 10% of cases having an organic root cause.[6] Much has changed in our understanding of ED since that time. Yet, while much of the attention focuses on organic causes, once these causes are ruled out, the clinician must also consider underlying psychogenic causes of ED. There have been several investigatory studies that have suggested an existing relationship between co-existing depression and anxiety in male patients who suffer from ED.[26] It is therefore important for the clinician to keep this in mind when evaluating a male patient for ED.

DIAGNOSTIC WORK-UP

Much of the diagnostic work-up for ED focuses on ruling out underlying comorbid organic disease. The 5-item abridged version of the International Index of Erectile Function IIEF-5 is an in-office questionnaire used to screen for the presence and severity of ED. Men's responses are scored and tallied for a total sum score that can then be interpreted. Refer to IIEF-5 questionnaire in Figure 1. Aside from this useful screening tool, it is also appropriate and essential to do a thorough physical exam, including examination of the male genitalia, as well as asking about previous sexual, psychological, and family history, recent or past recreational use of illicit drugs, and determination of present comorbid medical problems. Initial laboratory evaluation should at the very least include: blood glucose levels preferably fasting, HbA1c level, biochemical assessment of kidney function, as nearly 70 percent of males with chronic kidney disease report some degree of ED,[27] liver function tests, fasting cholesterol panel, as well as consideration of a work-up for acquired male hypogonadism including morning serum total testosterone level, TSH level, LH levels, and SHBG levels, if deemed appropriate *(see Table 3)*. Evaluation for lower urinary tract symptoms including UA, DRE for prostate evaluation, and consideration of PSA in the appropriate clinical setting, are also prudent in the diagnostic evaluation process. Vascular studies to assess blood flow penile duplex ultrasonography and arteriography as well as psychiatric evaluation can also be considered, but usually not until after a thorough work-up as mentioned above has been performed.[2]

TABLE 3:

Suggested Laboratory Studies to Consider in the Work-up of ED

HbA1c	Fasting Glucose	CMP	Fasting Lipid Profile
TSH	LH levels	Morning Total Testosterone Level	Sex-Hormone Binding Globulin (SHBG) level
	Urinalysis	Prostate Specific Antigen (PSA) level	

TREATMENT STRATEGIES

Oral Medicinal Approaches

The most widely accepted first line of oral treatments for ED remains the phosphodiesterase-5 (PDE-5) inhibitors. The medications available by prescription only in the U.S. that fall into this particular class are listed in Table 4. The American Urological Association endorses the use of sildenafil, tadalafil, vardenafil, and avanafil as first-line oral therapy in this class.[3] These therapeutic agents inhibit the high concentrations of PDE-5 found in the corpora cavernosa and, through a cascade of chemical processes, help promote erection.[2] In an ideal patient i.e. those without comorbid contraindications, see "Contraindications and Cautions," Table 3, they are generally effective, convenient, and well tolerated. One important point to stress when utilizing these medications is that in order for them to work appropriately, sexual stimulation is required. Many couples fail to understand this important point, and as such, may report an inadequate response to therapy or even perceived ineffectiveness of the medication. As a point of anticipatory guidance, the physician should attempt to explain this point to both partners. It is also important to note that this class of medication is contraindicated in patients who are currently taking nitrates as this combination can potentially lead to severe and life-threatening hypotension.[11] The physician should obtain a thorough sexual history in order to facilitate appropriate treatment dosing and/or strategies. Sildenafil, tadalafil, and vardenafil all share as needed dosing schedules usually taken anywhere from 30 minutes to 1 hour prior to sexual intercourse. Tadalafil is unique, however, in that it is the only one of the three that also carries a once daily dosing regimen without regard to timing of sexual activity. On follow-up visits to the office, the physician should inquire about the effectiveness of the medication, as failing to do so may often miss any potential for drug titration and/or further consideration/investigation in the matter. It is important to review these matters in follow-up visits with patients as there is as high as a 33 percent discontinuation rate of successful therapy with PDE-5 inhibitors for a variety of reasons *(see Table 5)* including unacceptability of planned sexual activity.[28]

Non-Oral Medicinal Approaches

Other medicinal strategies include prostaglandin E1 (PGE1), also known as alprostadil, either via transurethral use or intracavernosal penile injections. While transurethral alprostadil can be considered an acceptable first line treatment option for ED alone or in combination with PDE-5 inhibitor therapy, intracavernosal injection of prostaglandin E1 therapy is considered more of a second line treatment strategy for ED.[2] Use of PGE1 can be helpful in those suffering from neurological conditions that contribute to ED as this approach often bypasses the need for intact neurological architecture as it applies to erection.[2] Potential adverse reactions to PGE1 therapy include headache, back pain, urethral pain and bleeding with intraurethral insertion, testicular pain, pro-

TABLE 4:

FDA Approved PDE-5 Inhibitor Therapies

Drug	Dosing	Half-Life	Commonly Reported Side-Effects	Contraindications & Cautions	Relative Cost
Sildenafil (Viagra®)	25,50,100mg PRN dosing (Max: 100mg/dose, 1 dose/24 hrs	4 hours	Headache, Flushing, Visual Disturbances, Dyspepsia	CAD or H/O MI w/in 6 mo, HTN, Hypotension, Renal or Hepatic Impairment, Nitrate use within last 24 hrs	$$$
Vardenafil (Levitra®)	2.5,5,10,20mg PRN dosing (Max: 20mg/dose, 1 dose/24 hrs)	4 hours	Headache, Rhinitis, CK elevations, Flushing, Abnormal LFT's, Back Pain, Lengthening of QT interval	Prolonged QT interval, Hepatic or Renal Impairment, HTN, CAD or H/O MI w/in 6 months, Nitrate use with last 24 hrs	$$$$
Tadalafil (Cialis®)	2.5,5,10,20mg PRN dosing 2.5-5mg daily regimen dosing	17.5 hours	Headache, Back Pain, Myalgia, Flushing, Nasal Congestion, Dyspepsia	HTN, CAD or H/O MI w/in 6 mo, Hypotension, Renal or Hepatic Impairment, Nitrate use within last 48 hrs	$$$$
Avanafil (Stendra®)	50,100,200mg PRN dosing (Max: 200mg/dose, 1 dose/24 hrs)	1.5 hours	Headache, Flushing, Nasal Congestion, Nasopharyngitis & Back Pain	CAD or H/O MI w/in 6 mo, HTN or Hypotension, Hepatic or Renal Impairment, History of hereditary retinal disorders	$$$$

Reference: *ePocrates Drug Reference and adapted from Hakky, Tariq Said, and Lakshay Jain. "Current Use of Phosphodiesterase Inhibitors in Urology." Türk Üroloji Dergisi/Turkish Journal of Urology Turkish Journal of Urology 41.2 (2015): 88-92. PubMed Central. Web. 22 Sept. 2015.*

longed erection, penile ecchymosis or fibrosis with intracavernosal injection, and influenza-like symptoms. PGE1 therapy should be avoided in patients who have sickle cell anemia or trait, penile deformities, or penile implants.[29,30] Other options for intracavernosal injection other than PGE1 monotherapy include the addition of papaverine and/or phentolamine, although these additional agents may be considered more controversial.[2] Obvious deterrents to these methods are patient reported discomfort associated with penile injections or intraurethral insertion of PGE1.

Non-Medicinal Approaches

Vacuum assist devices or, vacuum erection devices VED, can be used effectively in most cases as a first-line treatment strategy for ED.[2] Although effective, there is a reported unfavorable acceptance amongst male patients and high long-term rate of discontinuation, mostly attributable to the peculiar feeling of the erection achieved with such device and the cumbersome nature of the entire process, in general.[2]

Other approaches, generally considered third-line strategies, would include penile prosthesis pumps and revascularization techniques that are beyond the scope of this particular article. In such cases, these patients are likely being evaluated and managed by urological specialists.

Osteopathic Manipulative Treatment OMT may also be utilized in male patients suffering from ED. Since somatic inputs from the pudendal nerve S2-S4 and parasympathetic reflexes S2-S4 are involved in male erection, it would be beneficial to identify and target any potential somatic dysfunctions involving the S2-S4 nerve root distribution pelvic splanchnics. Possible somatic dysfunctions that may involve the sacroiliac SI joint include sacroiliac strains, sacral shears, as well as sacral torsions. These somatic dysfunctions should be considered in male patients suffering from ED when other organic causes have been ruled out.[31] A variety of osteopathic treatments may be enacted for sacral dysfunctions including sacral rocking and various muscle energy techniques.

TABLE 5:

Reported Reasons for Discontinuation of PDE-5 Therapy Despite Effectiveness

Emotional unreadiness after an extended period of abstinence	Concerns regarding adverse effects of medication
Return of spontaneous erection	Refusal to accept 'drug-dependent' erections
Lack of sexual interest	Unacceptability of planned sexual intercourse

SUMMARY

Erectile dysfunction is a relatively important issue in men's health that warrants further discussion and consideration amongst primary care physicians. While it was originally believed that the underlying mechanism of ED was more psychogenic in nature, over the last 40 years such thinking has been abandoned in favor of investigating underlying organic causes as the primary contributor. The causes of ED are varied and it is clear that there are multitudes of medical comorbidities that contribute to it, including diabetes, hypertension, vascular disease, chronic kidney disease, etc. Conversations that are more candid need to be had between primary care physicians and their male patients regarding symptoms of ED, especially after the age of 40. While the topic may be one of considerable anxiety for many males in the context of a routine office visit, screening male patients for ED may help family physicians earlier discern undiagnosed cardiovascular health concerns as well as metabolic complications commonly associated with occult/undiagnosed diabetes. Diagnostic work-up includes routine chemistry studies, hemoglobin A1c HbA1c determination, UA, assessment of fasting cholesterol levels, and, in some cases, further analysis of laboratory studies for male hypogonadism, along with a thorough physical exam and medical history. Oral medicinal treatment

IIEF - 5 SCREENING QUESTIONNAIRE FOR ERECTILE DYSFUNCTION

Please Circle the Response That Best Describes You for the Following 5 Questions					
Over the Past 6 Months:					
1. How do you rate the confidence that you could get and keep an erection?	Very low 1	Low 2	Moderate 3	High 4	Very High 5
2. When you had erections with sexual stimulation, how often were your erections hard enough for penetration?	Almost never or never 1	A few times *(much less than half the time)* 2	Sometimes *(about half the time)* 3	Most times *(much more than half the time)* 4	Almost always or always 5
3. During sexual intercourse, how often were you able to maintain your erection after you had penetrated your partner?	Almost never or never 1	A few times *(much less than half the time)* 2	Sometimes *(about half the time)* 3	Most times *(much more than half the time)* 4	Almost always or always 5
4. During sexual intercourse, how difficult was it to maintain your erection to completion of intercourse?	Extremely difficult 1	Very difficult 2	Difficult 3	Slightly difficult 4	Not difficult 5
5. When you attempted sexual intercourse, how often was it satisfactory for you?	Almost never or never 1	A few times 2	Sometimes 3	Most times 4	Almost always or always 5

TOTAL SCORE: _____

INTERPRETATIONS: 1 - 7: Severe ED 8 - 11: Moderate ED 12 - 16: Mild-Moderate ED 17 - 21: Mild ED 22 - 25: No ED

strategies in the form of PDE-5 inhibitors are now widely available in the United States, allow for relative patient convenience, and, generally speaking, can be safely prescribed by most primary care physicians under the appropriate circumstances. If first line therapies such as the oral PDE-5 inhibitors, injection therapies, intraurethral therapies, or VEDs do not prove effective, then further work-up and more invasive strategies may be considered necessary under the guidance of a trained urological specialist. Identifying sacral somatic dysfunctions and providing corrective treatment with osteopathic manipulative therapy may also be a helpful adjunct to other conventional strategies.

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