The Second Princeton Consensus on Sexual Dysfunction and Cardiac Risk: New Guidelines for Sexual Medicine

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DOI: 10.1111/j.1743-6109.2005.00196.x

ABSTRACT

Introduction. Erectile dysfunction (ED) is a highly prevalent disorder associated with a significant burden of illness. The prevalence and incidence of ED are strongly age-related, affecting more than half of men >60 years. The first Princeton Consensus Conference (Princeton I) in 1999 developed guidelines for safe management of cardiac patients regarding sexual activity and the treatment of ED.

Aim. The second conference (Princeton II) was convened to update the recommendations based on the expanding knowledge base and new treatments available. This article reviews and expands on the Princeton II guidelines to address sexual dysfunction and cardiac risk.

Methods. A consensus panel of experts reviewed recent multinational studies in safety and drug interaction data for three phosphodiesterase type 5 (PDE5) inhibitors (sildenafil, tadalafil, vardenafil), with emphasis on the safety of these agents in men with ED and concomitant cardiovascular disease.

Results. Erectile dysfunction is an early symptom or harbinger of cardiovascular disease, due to the common risk factors and pathophysiology mediated through endothelial dysfunction. Major comorbidities include diabetes, hypertension, hyperlipidemia and heart disease. Any asymptomatic man who presents with ED that does not have an obvious cause (e.g., trauma) should be screened for vascular disease and have blood glucose, lipids, and blood pressure measurements. Ideally, all patients at risk but asymptomatic for coronary disease should undergo an elective exercise electrocardiogram to facilitate risk stratification. Lifestyle intervention in ED, specifically weight loss and increased physical activity, particularly in patients with ED and concomitant cardiovascular disease, is literature-supported.

Conclusions. The recognition of ED as a warning sign of silent vascular disease has led to the concept that a man with ED and no cardiac symptoms is a cardiac (or vascular) patient until proven otherwise. Men with ED and other cardiovascular risk factors (e.g., obesity, sedentary lifestyle) should be counseled in lifestyle modification. Jackson G, Rosen RC, Kloner RA, and Kostis JB. The second Princeton consensus on sexual dysfunction and cardiac risk: new guidelines for sexual medicine. J Sex Med 2006;3:28–36.

Key Words. Erectile Dysfunction; Cardiovascular Disease; Endothelial Health
endothelial dysfunction as a cause [3]. Vascular diseases are the most common cause of ED with coronary artery disease (CAD) and ED sharing the same risk factors (Figure 1). The first Princeton Consensus Conference (Princeton I) in 1999 addressed the evidence linking sexual activity and cardiac risk and developed guidelines for safe management of cardiac patients regarding sexual activity and the treatment of ED [4]. The second conference (Princeton II) was convened to update the recommendations based on the expanding knowledge base and new treatments available [5].

Princeton I has proved to be a useful clinical guide to evaluating ED and sexual activity in cardiac patients and those at cardiac risk. The importance of ED as a risk marker in men with no cardiac symptoms is highlighted in Princeton II as is the value of lifestyle changes. The concept of “physically fit = sexually fit” is emphasized. The categories of low-, intermediate- or indeterminate-, and high-risk patients are maintained but updated (Table 1) and the algorithm changed to emphasize the importance of CAD and general vascular disease risk assessment.

**Erectile Dysfunction as a Marker of Vascular Disease**

As ED and vascular disease share the same risk factors, the possibility arises that ED in otherwise asymptomatic men may be a marker of silent vascular disease, especially CAD [6]. This has now been established to be the case and represents an important new means of identifying those at risk of vascular disease.

Pritzker [7] studied 50 asymptomatic men (other than ED) aged 40–60 years who had cardiovascular risk factors (multiple in 80%). Exercise electrocardiogram (ECG) was abnormal in 28 men and subsequent coronary angiography in 20 men identified severe CAD in six, moderate two-vessel disease in seven, and significant single-vessel CAD in a further seven men. In a study of 132 men attending day case angiography, 40% had experienced ED before their CAD diagnosis had been made [8]. ED also correlates with the severity of CAD with single vessel disease patients having less difficulty in obtaining an erection [9].

It has been proposed that the smaller penile arteries (diameter 1–2 mm) suffer obstruction from plaque burden earlier than the larger coronary (3–4 mm), carotid (5–7 mm), or iliofemoral (6–8 mm) arteries, hence ED may be symptomatic before a coronary event [10]. Addressing cardiovascular risk early after the presentation of ED and aggressive intervention to reduce risk may have long-term symptomatic and prognostic cardiac benefits [11]. Most acute coronary syndromes follow from asymptomatic lipid-rich plaques rupturing and ED may therefore be a marker for reducing the risk of this happening [12].

Any asymptomatic man who presents with ED that does not have an obvious cause (e.g., trauma) should be screened for vascular disease and have blood glucose, lipids, and blood pressure measurements. Ideally, all at-risk but asymptomatic for coronary disease patients should undergo an elective exercise ECG to facilitate risk stratification [13].

**Treating ED in Patients with Cardiovascular Disease**

The Princeton II consensus guidelines recommend that all men with ED should undergo a full medical assessment (Figure 2). Baseline physical activity needs to be established and cardiovascular...
risk graded low, intermediate, or high. Most patients with low or intermediate cardiac risk can have their ED managed in the outpatient or primary care setting.

There is no evidence that treating ED or resuming sexual activity in patients with cardiovascular disease increases cardiac risk; however, this is with the proviso that the patient is properly assessed and the couple or individual (self-stimulation may be the only form of sexual activity) is appropriately counseled. Oral drug therapy is the most widely used because of its

Table 1  Risk from sexual activity in cardiovascular diseases: Second Princeton Consensus Conference

<table>
<thead>
<tr>
<th>Risk from Sexual Activity in Cardiovascular Diseases</th>
<th>Second Princeton Consensus Conference</th>
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<tbody>
<tr>
<td>Low risk: typically implied by the ability to perform exercise of modest intensity without symptoms</td>
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<tr>
<td>Asymptomatic and &lt;3 major risk factors (excluding gender)</td>
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<tr>
<td>Major cardiovascular disease risk factors include age, male gender, hypertension, diabetes mellitus, cigarette smoking, dyslipidemia, sedentary lifestyle, and family history of premature CAD</td>
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<tr>
<td>Controlled hypertension</td>
<td></td>
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<tr>
<td>Beta-blockers and thiazide diuretics may predispose to ED</td>
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<tr>
<td>Mild, stable angina pectoris</td>
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<tr>
<td>Noninvasive evaluation recommended</td>
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<tr>
<td>Antianginal drug regimen may require modification</td>
<td></td>
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<tr>
<td>Postrevascularization and without significant residual ischemia</td>
<td></td>
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<tr>
<td>ETT may be beneficial to assess risk</td>
<td></td>
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<tr>
<td>Post-myocardial infarction (MI) (&gt;6–8 weeks), but asymptomatic and without ETT-induced ischemia, or postrevascularization</td>
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<tr>
<td>If postrevascularization or no ETT-induced ischemia, intercourse may be resumed 3–4 weeks post-MI</td>
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<tr>
<td>Mild valvular disease</td>
<td></td>
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<tr>
<td>May include select patients with mild aortic stenosis</td>
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<tr>
<td>LVD (NYHA class I)</td>
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<tr>
<td>Most patients are low risk</td>
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<tr>
<td>Intermediate or indeterminate risk: evaluate to reclassify as high or low risk</td>
<td></td>
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<tr>
<td>Asymptomatic and ≥3 CAD risk factors (excluding gender)</td>
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<tr>
<td>Increased risk for acute MI and death</td>
<td></td>
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<tr>
<td>ETT may be appropriate, particularly in sedentary patients</td>
<td></td>
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<tr>
<td>Moderate, stable angina pectoris</td>
<td></td>
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<tr>
<td>ETT may clarify risk</td>
<td></td>
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<tr>
<td>MI &gt;2 weeks but &lt;6 weeks</td>
<td></td>
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<tr>
<td>Increased risk of ischemia, reinfarction, and malignant arrhythmias</td>
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<tr>
<td>ETT may clarify risk</td>
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<tr>
<td>LVD/congestive heart failure (CHF) (NYHA class II)</td>
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<tr>
<td>Moderate risk of increased symptoms</td>
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<tr>
<td>Cardiovascular evaluation and rehabilitation may permit reclassification as low risk</td>
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<tr>
<td>Noncardiac atherosclerotic sequelae (peripheral arterial disease, history of stroke, or transient ischemic attacks)</td>
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<tr>
<td>Increased risk of MI</td>
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<tr>
<td>Cardiological evaluation should be considered</td>
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<tr>
<td>High risk: defer resumption of sexual activity until cardiological assessment and treatment</td>
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<tr>
<td>Unstable or refractory angina</td>
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<tr>
<td>Increased risk of MI</td>
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<tr>
<td>Uncontrolled hypertension</td>
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<tr>
<td>Increased risk of acute cardiac and vascular events (i.e., stroke)</td>
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<tr>
<td>CHF (NYHA class III, IV)</td>
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<tr>
<td>Increased risk of cardiac decompensation</td>
<td></td>
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<tr>
<td>Recent MI (&lt;2 weeks)</td>
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<tr>
<td>Increased risk of reinfarction, cardiac rupture, or arrhythmias, but impact of complete revascularization on risk is unknown</td>
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<tr>
<td>High-risk arrhythmias</td>
<td></td>
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<tr>
<td>Rarely, malignant arrhythmias during sexual activity may cause sudden death</td>
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<tr>
<td>Risk is decreased by an implanted defibrillator or pacemaker</td>
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<tr>
<td>Obstructive hypertrophic cardiomyopathies</td>
<td></td>
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<tr>
<td>Cardiovascular risks of sexual activity are poorly defined</td>
<td></td>
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<tr>
<td>Cardiological evaluation (i.e., exercise stress testing and echocardiography) may guide patient management</td>
<td></td>
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<tr>
<td>Moderate to severe valve disease</td>
<td></td>
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<tr>
<td>Use vasoactive drugs with caution</td>
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</table>

Adapted from Kostis, et al. [5].

CAD = coronary artery disease; CHF = congestive heart failure; CV = cardiovascular; CVA = cerebrovascular accident; ED = erectile dysfunction; ETT = exercise tolerance test; LVD = left ventricular dysfunction; MI = myocardial infarction; NYHA = New York Heart Association.
acceptability and effectiveness, but all therapies have a place in management.

**Phosphodiesterase Type 5 Inhibitors**

Sildenafil, tadalafil, and vardenafil act by reducing the degradation of cyclic guanosine monophosphate (cGMP) by PDE5 promoting blood flow into the penis and the restoration of erectile function. Because their mechanism of action is the same, there is no reason to assume there will be any significant differences in ED effectiveness, but their half-life may be of cardiac clinical importance.

Sildenafil is the first oral treatment for ED and the most extensively evaluated [14]. Overall success rates in patients with cardiovascular disease of 80% or greater have been recorded with no evidence of tolerance. Patients with diabetes with or without additional risk factors, with their more complex and extensive pathophysiology, have an average success rate of 60%. In randomized trials to date, open-label, or outpatient monitoring studies the use of sildenafil is not associated with any excess risk of myocardial infarction, stroke, or mortality [15,16]. In patients with stable angina pectoris there is no evidence of an ischemic effect due to coronary steal, and in one large, double-blind, placebo-controlled, exercise study sildenafil 100 mg increased exercise time and diminished ischemia [17]. A study of the hemodynamic effects in men with severe CAD identified no adverse cardiovascular effects and a potentially beneficial effect on coronary blood flow reserve [18]. Sildenafil’s short half-life makes it the drug of choice in patients with the more severe cardiovascular disease, allowing early use of support therapy if an adverse clinical event occurs.

Tadalafil has also been extensively evaluated in patients with cardiovascular disease and has a similar safety and efficacy profile to sildenafil [19]. Studies have shown no adverse effects on cardiac contraction, ventricular repolarization, or ischemic threshold.

Because of its long half-life, tadalafil may not be the first choice for the patients with more severe cardiovascular disease. However, as 80% of patients with cardiovascular disease stratify into low risk it is an alternative for the majority.

Vardenafil has a very similar chemical structure to sildenafil so it is not surprising that it has a similar clinical profile. One study has reported no impairment of exercise ability in stable CAD patients receiving vardenafil 20 mg [20]. Similar clinical efficiency for all three agents has been observed in patients with diabetes.

**Other Therapies**

When oral agents are not effective, intracavernous injection therapy, transurethral alprostadil, and a vacuum pump are alternatives requiring specialized referral and advice [2,3]. There is no evidence of increased cardiovascular risk from using any of these therapeutic options. If surgical intervention with general anesthetic is being anticipated, a full cardiological risk evaluation is recommended.

**Interactions and Safety with Cardiovascular Drugs**

**Nitrates**

Organic nitrates, such as nitroglycerin, isosorbide dinitrate, and isosorbide mononitrate, are nitric oxide (NO) donors. NO stimulates the enzyme guanylyl cyclase to produce cGMP, the substance that ultimately causes relaxation of smooth muscle cells in the media of blood vessels. While NO donors increase the production of cGMP, PDE5 inhibitors prevent the breakdown of cGMP. When these two types of pharmacologic agents are coadministered, cGMP levels increase to the point that marked vasodilation of blood vessels may lead to symptomatic hypotension. Therefore, at the present time use of organic nitrates (including only occasional short-acting sublingual nitroglycerin) is an absolute contraindication to the prescription of the PDE5 inhibitors [21–23]. Some patients with a diagnosis of CAD who do not develop ischemia (often they have had revascularization procedures such as percutaneous coronary interventions or coronary artery bypass grafting) continue to carry nitroglycerin, when it may not be needed. If these patients have ED it may be worth evaluating their true need for nitrates, which may include an exercise stress test. If it is deemed that nitrates are not needed, these agents could be discontinued and a trial of PDE5 inhibition initiated.

Suppose a patient develops angina during sexual activity with a PDE5 inhibitor on board. The patient should stop sexual activity and if the pain does not resolve in a few minutes seek emergency care [24]. It is crucial that the patient communicate to his care providers that he has taken a PDE5 inhibitor, in which case nitroglycerin or other organic nitrates should not be given for anginal relief. If the chest pain is angina, then non-nitrate
antianginal/anti-ischemic agents (beta-blockers, calcium channel blockers, aspirin, oxygen, morphine, heparin, statin, others) may be safely administered in the setting of a PDE5 inhibitor [25].

If a PDE5 inhibitor has been taken, when is it safe to re-administer nitrates? If the patient has taken a short-acting PDE5 inhibitor (sildenafil or vardenafil—half-life about 4 hours) nitrates can be restarted 24 hours after the last PDE5 dose [25]. As tadalafil is a long half-life agent (17.5 hours) a study suggests that at least 48 hours should elapse between the last tadalafil dose and re-administration of a nitrate [26].

If a patient develops an acute myocardial infarction while taking PDE5 inhibitors, then the usual therapies such as aspirin, thrombolytics, percutaneous coronary intervention, and antiplatelet agents are fine; however, organic nitrates should not be given [25].

Alpha-Blockers

When PDE5 inhibitors are administered to patients on the antihypertensive agents, beta-blockers, diuretics, calcium blockers, angiotensin converting enzyme inhibitors, angiotensin receptor blockers, there is usually a small or no additional fall in arterial blood pressure [27–31]. In general, the PDE5 inhibitors are safe and effective in patients with ED on antihypertensives. There is one group of antihypertensive agents, however, that raises a precaution when given with PDE5 inhibitors—the alpha-blockers. Alpha-blockers alone can induce orthostatic hypotension. They also have varying degrees of interaction with PDE5 inhibitors that may result in added falls in blood pressure [32]. At the present time, all three of the alpha-blockers contain a precaution regarding the concomitant administration of PDE5 inhibitors. In general, it is prudent to start the PDE5 inhibitors at a low dose in patients who have already adjusted to a stable dose of an alpha-blocker.

Phosphodiesterase type 5 inhibitors have a small effect on the QTc interval of the ECG. While to date there have been no documented cases of Torsades des Pointe with the use of these agents, vardenafil, which increased the QTc by 6–9 milliseconds in one study, is not recommended for use in patients receiving type 1A antiarrhythmic agents (such as quinidine or procainamide) or type 3 antiarrhythmic agents (such as sotalol or admiodarone) or in patients with known congenital prolonged QT interval [21].

The Role of Lifestyle Factors

**Obesity and Sedentary Lifestyle**

Lifestyle factors have been associated with ED in both cross-sectional and longitudinal studies. In particular, obesity and sedentary lifestyle are clear-cut risk factors for ED, both in men with comorbid illnesses such as hypertension and diabetes, and especially in men without overt cardiovascular disease [33]. Other lifestyle factors, such as smoking and alcohol consumption, have been implicated in some, but not all, studies to date. Intervening on cardiovascular and lifestyle factors may have broader benefits beyond restoration of erectile function. This important concept needs careful consideration, as recent studies have implicated the role of the metabolic syndrome, obesity, insulin resistance, and lack of exercise as independent risk factors for both ED and cardiovascular disease [2,34,35].

The role of obesity in ED has been confirmed in large-scale, cross-sectional, and longitudinal studies [36,37]. In a study in The Netherlands, for example, 1,700 Dutch men between the ages of 50 and 75 were evaluated for the presence of ED and other health conditions [38]. Body mass index (BMI) was found to be a significant predictor of ED, both as a single factor and in combination with other risk factors (e.g., lower urinary tract symptoms [LUTS], hypertension, diabetes). Similar findings were reported in the Health Professionals Follow-Up Study in the United States [36], and in the recent Men in Australia, Telephone Survey (MATES) study in Australia [39]. In both of these large-scale, multinational studies, obesity was found to be a significant, independent risk factor for ED. The mechanisms remain to be established.

Physical activity is another lifestyle factor that has been strongly linked to the occurrence of ED in aging men. In the Health Professionals Follow-Up Study [36], for example, ED was associated with both BMI and level of physical activity. Participants were categorized according to their level of exercise or physical activity. Higher levels of sedentary behavior (less physical activity) was found to be a strong, independent predictor of ED in this study. Frequent vigorous exercise, it was shown, was associated with an approximately 30% reduction in the risk for ED. Similar results for the effects of exercise were reported in the Global Study of Sexual Attitudes and Behaviour (GSSAB) study [40].

In the cross-national sample from the GSSAB study, Nicolosi et al. [41] examined the associa-
tion between lifestyle factors and ED specifically in men without major medical comorbidities (e.g., prostate cancer, diabetes). In this sample of >1000 men, men with ED and without a diagnosis of cardiovascular or prostate diseases, diabetes, ulcer, or depression were investigated. The goal was to determine the significance of lifestyle factors in men with ED who do not have major medical illnesses or comorbidities. It is interesting to note that the association between erectile function and lifestyle factors, particularly obesity and sedentary behavior, was more clearly evident in this study compared with previous studies. Specifically, the authors found that 31.8% of men who reported less than average levels of physical activity had ED, compared with 13.9% of those who exercised more than average. The protective effects of exercise and lower BMI were strongly evident in this study, perhaps related to the fact that the direct effects of medical comorbidities (e.g., diabetes, heart disease) had not obscured the association.

The effects of weight loss and exercise were examined further in a recent, randomized intervention trial of lifestyle modification in men with obesity-related ED [42]. This study compared 2 years of exercise and weight loss with an educational control in 110 obese men (mean BMI = 36.4 kg/m^2) with moderate to severe ED. As in the cross-national study [41], men with overt diabetes or other cardiovascular diseases were excluded from the trial. Approximately one-third of men in the intervention group achieved normal levels of erectile function following treatment, compared with <5% of men in the control condition. Changes in weight loss and exercise were shown to affect endothelial function, as measured by forearm brachial Doppler assessment, and were highly correlated with improvements in erection.

Taken together, these studies strongly support the role of lifestyle factors in the development and maintenance of ED. Obesity and lack of exercise, in particular, have been strongly implicated in a number of cross-sectional and longitudinal studies. At least one long-term prospective study has shown that lifestyle intervention can effectively restore erectile function in a substantial number of men with obesity-related ED, at least among those without significant medical comorbidities. For clinicians, the implications are clear that men with ED and other cardiovascular risk factors (e.g., obesity, sedentary lifestyle) should be counseled in lifestyle modification.

Androgens

The use of testosterone replacement therapy for the treatment of hypogonadism and ED may assist PDE5 inhibitors if they have failed to be effective [43]. Testosterone levels within the normal range have neutral or potentially beneficial effects on the cardiovascular system [44]. Androgen replacement therapy should be offered to men with CAD and hypogonadism if symptomatically appropriate. The absence of long-term studies needs to be addressed in terms of possible preventive properties on the vascular wall, reduction in low-density lipoprotein levels, and the reduction of insulin resistance in contrast to the increase in hematocrit and risk of exacerbating prostate cancer.

Potential for PDE5 Inhibitors in Cardiovascular Disease

In studies of sildenafil on endothelial function as measured by forearm flow-mediated vasodilation in the acute setting, as well as following chronic oral dosing, a significant improvement was recorded in comparison with placebo in cardiac failure and diabetic patients [45,46]. In a study of 25 subjects with or without coronary atherosclerosis, the abnormal vasoconstrictor response to acetylcholine in diseased vessels was improved by sildenafil, while the normal vasodilator response in nondiseased vessels was unchanged [47]. These findings suggest that PDE5 inhibition improves the vasomotor aspect of endothelial function in the presence of diseased vessels.

The reported significant hemodynamic benefits as well as clinical benefits in pulmonary hypertension add weight to this concept [48]. Recent reports of clinical benefit in advanced heart failure have also excited interest [49]. There is little information on the therapeutic blood pressure response in hypertensive patients but falls of up to 24/8 mm Hg have been recorded—similar to hypotensive conventional agents [50].

Therefore, hypertension, pulmonary hypertension, cardiac failure, Raynaud's phenomenon, and the potential for prevention of disease or its progression in disease states such as diabetes are important areas for clinical study.

Conclusions

The Princeton II guidelines expand on the solid foundation of Princeton I. The recognition of ED as a warning sign of silent vascular disease has led to the concept that a man with ED and no cardiac
symptoms is a cardiac (or vascular) patient until proved otherwise. The next stage in our development and understanding of ED and the underlying mechanisms is to evaluate whether cardiac risk reduction will prevent subsequent events in this patient subset. ED stands for Erectile Dysfunction, Endothelial Dysfunction, Exercise and Diet in prevention, and Early Detection of risk factors with a view to preventing Early Death. Just as there is more to sex than an erect penis, there is more to ED than its symptomatic treatment, with or without PDE5 inhibitors.

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Conflict of Interest: None.

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Commentary

The Journal of Sexual Medicine is honored to have been chosen by these renowned authors for publication of the Princeton II Guidelines document. The Princeton I conference in 1999 had a profound impact on the public and the medical community, at a time when prescribing phosphodiesterase type 5 (PDE5) inhibitors in men with heart disease was considered dangerous and potentially lethal. The conference participants provided a well-needed strategic clinical guide that was evidence-based, rational, user-friendly, and practical. The algorithm, designed to aid the practitioner manage erectile dysfunction (ED) and sexual activity in men with cardiac risk, designated ED patients as low, intermediate, and high cardiac risk. Carefully defined logical patient management strategies for the important subpopulation of the cardiac patient followed, re-establishing order in the field of sexual medicine.

Why are the Princeton II Guidelines relevant to the healthcare provider? There are two key evidence-based and consensed statements in the new Princeton II Guidelines that will affect “Good Medical Practice” patterns.

Statement 1: “Any asymptomatic man who presents with ED that does not have an obvious cause (e.g., trauma or post-radical prostatectomy) should be screened for vascular disease and have blood glucose, lipids, and blood pressure measurements. Ideally, all at risk but asymptomatic for coronary disease should undergo elective exercise ECG to facilitate risk stratification.”

Statement 2: “Studies strongly support the role of lifestyle factors in the development and maintenance of ED. Obesity and lack of exercise have been strongly implicated in a number of cross-sectional and longitudinal studies. Men with ED and other cardiovascular risk factors, in particular, obesity and sedentary lifestyle, should be counseled in lifestyle modification.”

On a practical basis the Princeton II Guidelines will change how many practitioners care for their patients. Take the case of a 46-year-old man, obese and living a sedentary lifestyle, who seeks consultation for ED of 6-month duration. The clinician’s examination is, however, only cursory. A script for a PDE5 inhibitor and a follow-up visit in 1 year are the only management provided. Three months later the man suffers a fatal myocardial infarction. Upon the request of the family his medical records are reviewed and no notation of cardiac risk factors, lifestyle modification, or testing for cardiac disease is found. Did the clinician perform “Good Medical Practice” in this case?

The current practice guidelines for healthcare providers managing men with ED have been further clarified by the Princeton II Guidelines. Based on these new guidelines, the 46-year-old man (see above) should have had a more detailed history of vascular risk exposure obtained. In addition, the healthcare provider should have better screened the patient for vascular disease with a series of recommended blood tests. Should these blood tests have been abnormal, an elective electrocardiogram (ECG) may have been performed that may have identified the individual patient’s high risk, thereby potentially saving his life.

Kudos to the authors and their outstanding, innovative, and ground-breaking contributions from the Princeton I and II Conferences. The common denominator of endothelial dysfunction renders ED as an early symptom or harbinger of cardiovascular disease. The Princeton II Guidelines provide credibility and evidence to that association with the establishment of consensed guidelines.

Irwin Goldstein, MD
Editor-in-Chief, The Journal of Sexual Medicine