The Cardiac Urologist or the Urologic Internist—Sexual Activity and the Heart in the Aging Male (Princeton III Guidelines)

Dr. Martin Miner
Providence, Rhode Island

Introduction

The third Princeton Consensus Conference took place in Miami Beach, Florida, November 8-10, 2010.[1] The group revisited and updated its 2005 recommendations regarding the cardiovascular risk associated with sexual activity in men with known cardiovascular disease (CVD). The third conference also focused on the predictive value of vasculogenic erectile dysfunction (ED) in assigning cardiovascular risk in men of all ages. The primary objective was to develop an approach to cardiovascular risk assessment in younger men with ED and no known CVD. The role of testosterone in erectile function and cardiovascular health, and the usefulness of testosterone replacement therapy were also examined.

Recommendations

Recommendations were developed by the same process used in the first and second conferences.[1,2] Briefly, an international panel of 22 experts provided state-of-the-art presentations on the epidemiological and physiological links among hypogonadism, ED and CVD; the benefits and risks of testosterone depletion and repletion; evaluation of traditional and emerging cardiometabolic risk factors and assessment tools; and available ED treatments. After the presentations a representative Consensus Panel met to review and, based on available scientific evidence, develop recommendations for further research and clinical practice. The recommendations were modified and finalized through electronic communication and approved by a final consensus of the panel.

The recommendations of the third Princeton Consensus Conference focus on 1) evaluation and management of cardiovascular risk in men with ED and no known CVD, and 2) reevaluation and modification of the second conference recommendations for evaluating the cardiac risk associated with sexual activity in men with known CVD.

Cardiovascular Risk in Patients with ED and No Known CVD

The figure shows panel recommendations for evaluating and managing cardiovascular risk in men with ED and no known CVD. The Consensus Panel defined cardiovascular risk as the risk of morbid events in a 3 to 5-year interval from ED onset (class 1b).[3]

The panel approach broadened the use of the 2010 American College of Cardiology Foundation/American Heart Association (ACCF/AHA) guideline for assessing cardiovascular risk in asymptomatic adults to address an at risk patient population that the guideline failed to mention, namely men with ED. The 2010 ACCF/AHA guideline provides recommendations and levels of evidence for some of the recommended assessments and the authors provided them for the other recommendations.[3]

ED provides an opportunity to decrease the CVD risk, as it shares risk factors with CVD and is itself a marker of increased risk of CVD

(class Ia).[4] In fact, incident ED has a similar or greater predictive value for cardiovascular events than traditional risk factors such as family history of myocardial infarction, smoking and hyperlipidemia.[5]

ED commonly occurs in the presence of silent coronary artery disease (CAD) with a 2 to 5-year interval between ED onset and a CAD event (class Ia).[6] Evidence suggests that ED predicts peripheral arterial disease (PAD) and stroke. In a population based study of 40 to 70-year-old men adding ED status to the Framingham Risk Score (FRS) in a multivariate statistical model resulted in reclassifying 5 men who were at low risk (less than 5%) to intermediate risk (5% to less than 10%) of CAD.[4]

Data from the Olmsted County study suggest that ED is far more predictive of CAD in 40 to 49-year-old men than in older men.[7] Also, the incidence of atherosclerotic cardiovascular events in men younger than 40 years with ED was more than 7 times the incidence in a reference population representative of the general male population of Western Australia.[8]

Thus, ED may be particularly useful to assess cardiovascular risk in younger and minority men whose risk may be underestimated by global risk assessments such as the FRS. ED assessment must include ED severity since more severe ED is associated with a greater risk of major cardiovascular events, CAD, the extent of CAD and the risk of PAD (class Ia).[9]

Consistent with other guidelines, the third Princeton Consensus Panel recommendations emphasize an approach to risk assessment that integrates multiple aspects of cardiometabolic health. Sexual function should be incorporated into CVD risk assessment for all men. ED may allow the identification of those at intermediate risk who require further cardiovascular evaluation incorporating an exercise stress test, imaging and biomarker assessment. The scientific evidence suggests that a comprehensive approach to cardiovascular risk reduction would improve overall vascular health, including sexual function.

Similar to the first and second Princeton Consensus Panels, the third panel also provides an approach to ensure that the cardiovascular health of each man is consistent with the physical demands of sexual activity before prescribing ED treatment. Finally, the panel encourages a collaborative approach to managing male sexual function and cardiovascular risk, incorporating general, urological, endocrine and cardiological expertise. The Executive Summary authored by the panel is pending publication. ◆

1. Kostis JB, Jackson G, Rosen R et al: Sexual dysfunction and cardiac risk (the Second Princeton Consensus Conference). Am J Cardiol 2005; **96**: 313.
2. DeBusk R, Drory Y, Goldstein I et al: Management of sexual dysfunction in patients with cardiovascular disease: recommendations of The Princeton Consensus Panel. Am J Cardiol 2000; **86**: 175.
3. Greenland P, Alpert JS, Beller GA et al: 2010 ACCF/AHA guideline for assessment of cardiovascular risk in asymptomatic adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 2010; **122**: e584.
4. Araujo AB, Hall SA, Ganz P et al: Does erectile dysfunction contribute to cardiovascular disease risk prediction beyond the Framingham Risk Score? J Am Coll Cardiol 2010; **55**: 350.
5. Thompson IM, Tangen CM, Goodman PJ et al: Erectile dysfunction and subsequent cardiovascular disease. JAMA 2005; **294**: 2996.
6. Montorsi P, Ravagnani PM, Galli S et al: Association between erectile dysfunction and coronary artery disease. Role of coronary clinical presentation and extent of coronary vessels. *Eur Heart J* 2006; **27**: 2613.
7. Inman BA, Sauver JL, Jacobson DJ et al: A population-based, longitudinal study of erectile dysfunction and future coronary artery disease. Mayo Clin Proc 2009; **84**: 108.
8. Chew KK, Finn J, Stuckey B et al: Erectile dysfunction as a predictor for subsequent atherosclerotic cardiovascular events: findings from a linked-data study. J Sex Med 2010; **7**: 192.

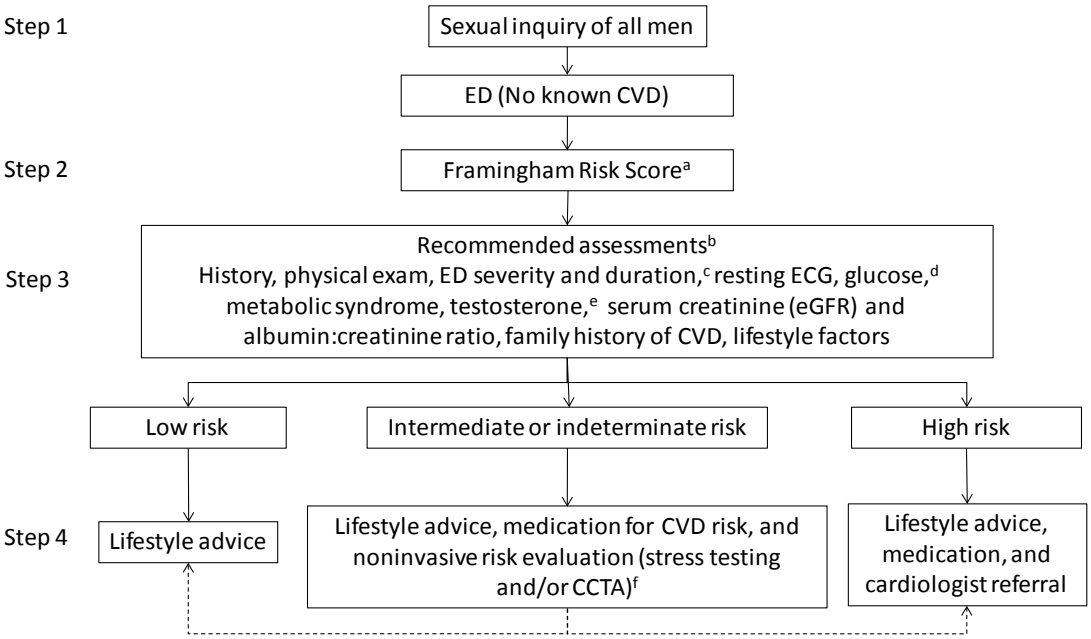

Figure. Cardiovascular risk assessment in men with ED and no known CVD. *ECG*, electrocardiogram. *eGFR*, estimated glomerular filtration rate. *CCTA*, coronary computerized tomography angiography.[a] Ten-year risk of myocardial infarction or coronary death incorporating age, sex, total cholesterol, high density lipoprotein cholesterol, smoking, systolic blood pressure and blood pressure therapy (http://hp2010.nhlbihin.net/atpiii/calculator.asp).[b] If man with ED has low, intermediate or indeterminate FRS, tests should be done to better clarify risk.[c] Determine severity based on International Index of Erectile Function, including mild—17 to 21, mild to moderate—12 to 16, moderate—8 to 11 or severe—1 to 7. Added risk if severe ED, longer than 3-year history and low testosterone.[d] Consider patients with diabetes at high risk.[e] In men diagnosed with organic ED and/or in whom phosphodiesterase type 5 inhibitors have failed.[f] Consider coronary computerized tomography angiography to determine obstructive/nonobstructive CAD, and biomarker assessment with coronary artery calcium scoring, carotid intima-media thickness, ankle-brachial index, pulse wave velocity, C-reactive protein, glycated hemoglobin, lipoprotein-associated phospholipase A2, urinary albumin excretion and endothelial function (ie EndoPAT™), when available.