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Sexual Activity and Cardiovascular Disease : A Scientific Statement From the American Heart Association

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AHA Scientific Statement

Sexual Activity and Cardiovascular Disease

A Scientific Statement From the American Heart Association

Endorsed by the American Urological Association, Society for Cardiovascular Angiography and Interventions, Society of Thoracic Surgeons, American Association of Cardiovascular and Pulmonary Rehabilitation, International Society of Sexual Medicine, American College of Cardiology Foundation, Heart Rhythm Society, and Heart Failure Society of America

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Sexual activity is an important component of patient and partner quality of life for men and women with cardiovascular disease (CVD), including many elderly patients.¹ Decreased sexual activity and function are common in patients with CVD and are often interrelated to anxiety and depression.^{2,3} The intent of this American Heart Association Scientific Statement is to synthesize and summarize data relevant to sexual activity and heart disease in order to provide recommendations and foster physician and other healthcare professional communication with patients about sexual activity. Recommendations in this document are based on published studies, the Princeton Consensus Panel,^{4,5} the 36th Bethesda Conference,^{6–10} European Society of Cardiology recommendations on physical activity and sports participation for patients with CVD,^{11–13} practice guidelines from the American College of Cardiology/American Heart Association^{14–16} and other organizations,¹⁷ and the multidisciplinary expertise of the writing group. The classification of recommendations in this document are based on established ACCF/AHA criteria (Table).

Acute Cardiovascular Effects of Sexual Activity

Numerous studies have examined the cardiovascular and neuroendocrine response to sexual arousal and intercourse, with most assessing male physiological responses during heterosexual vaginal intercourse.^{18–24} During foreplay, systolic and diastolic systemic arterial blood pressure and heart rate increase mildly, with more modest increases occurring transiently during sexual arousal. The greatest increases occur during the 10 to 15 seconds of orgasm, with a rapid return to baseline systemic blood pressure and heart rate thereafter. Men and women have similar neuroendocrine, blood pressure, and heart rate responses to sexual activity.^{24,25}

Studies conducted primarily in young married men showed that sexual activity with a person's usual partner is comparable to mild to moderate physical activity in the range of 3 to 4 metabolic equivalents (METs; ie, the equivalent of climbing 2 flights of stairs or walking briskly²⁶) for a short duration. Heart rate rarely exceeds 130 bpm and systolic blood pressure rarely exceeds 170 mm Hg^{4,18,27} in normoten-

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Table. Applying Classification of Recommendation and Level of Evidence

| | | SIZE OF TREATMENT EFFECT | | | | | | | | | | | | |
|---|--|--|---|--|--|--|----------------|-----------|---------------------|-------------|-------------------|---------------|------------------------------------|---------------------|
| | | CLASS I <i>Benefit >>> Risk</i> Procedure/Treatment SHOULD be performed/administered | CLASS IIa <i>Benefit >> Risk</i> <i>Additional studies with focused objectives needed</i> IT IS REASONABLE to perform procedure/administer treatment | CLASS IIb <i>Benefit ≥ Risk</i> <i>Additional studies with broad objectives needed; additional registry data would be helpful</i> Procedure/Treatment MAY BE CONSIDERED | CLASS III <i>No Benefit or CLASS III Harm</i> | | | | | | | | | |
| | | | | | <table border="1"> <thead> <tr> <th></th> <th>Procedure/Test</th> <th>Treatment</th> </tr> </thead> <tbody> <tr> <td>COR III: No benefit</td> <td>Not Helpful</td> <td>No Proven Benefit</td> </tr> <tr> <td>COR III: Harm</td> <td>Excess Cost w/o Benefit or Harmful</td> <td>Harmful to Patients</td> </tr> </tbody> </table> | | Procedure/Test | Treatment | COR III: No benefit | Not Helpful | No Proven Benefit | COR III: Harm | Excess Cost w/o Benefit or Harmful | Harmful to Patients |
| | Procedure/Test | Treatment | | | | | | | | | | | | |
| COR III: No benefit | Not Helpful | No Proven Benefit | | | | | | | | | | | | |
| COR III: Harm | Excess Cost w/o Benefit or Harmful | Harmful to Patients | | | | | | | | | | | | |
| ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT | LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses | <ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Sufficient evidence from multiple randomized trials or meta-analyses | <ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Some conflicting evidence from multiple randomized trials or meta-analyses | <ul style="list-style-type: none"> Recommendation's usefulness/efficacy less well established Greater conflicting evidence from multiple randomized trials or meta-analyses | <ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Sufficient evidence from multiple randomized trials or meta-analyses | | | | | | | | | |
| | LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies | <ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Evidence from single randomized trial or nonrandomized studies | <ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Some conflicting evidence from single randomized trial or nonrandomized studies | <ul style="list-style-type: none"> Recommendation's usefulness/efficacy less well established Greater conflicting evidence from single randomized trial or nonrandomized studies | <ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Evidence from single randomized trial or nonrandomized studies | | | | | | | | | |
| | LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care | <ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Only expert opinion, case studies, or standard of care | <ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Only diverging expert opinion, case studies, or standard of care | <ul style="list-style-type: none"> Recommendation's usefulness/efficacy less well established Only diverging expert opinion, case studies, or standard of care | <ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Only expert opinion, case studies, or standard of care | | | | | | | | | |
| Suggested phrases for writing recommendations | | should is recommended is indicated is useful/effective/beneficial | is reasonable can be useful/effective/beneficial is probably recommended or indicated | may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established | COR III: No Benefit is not recommended is not indicated should not be performed/administered/other is not useful/beneficial/effective | COR III: Harm potentially harmful causes harm associated with excess morbidity/mortality should not be performed/administered/other | | | | | | | | |
| Comparative effectiveness phrases† | | treatment/strategy A is recommended/indicated in preference to treatment B treatment A should be chosen over treatment B | treatment/strategy A is probably recommended/indicated in preference to treatment B it is reasonable to choose treatment A over treatment B | | | | | | | | | | | |

A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Although randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as sex, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use.

†For comparative effectiveness recommendations (Class I and IIa; Level of Evidence A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

sive individuals. However, one study of normotensive men demonstrated substantial variations in peak heart rate and systemic blood pressure during orgasm.²³ Because most of the studies that assessed the cardiovascular effects of sexual activity were conducted in healthy men who were young to middle-aged, equating the myocardial oxygen demand of intercourse to climbing 2 flights of stairs is a generalization that may not characterize all individuals, especially those who are older, are less physically fit, or have CVD.¹⁸ Therefore, it is probably more reasonable to state that sexual activity is equivalent to mild to moderate physical activity in the range of 3 to 5 METS, taking into account the individual's capacity

to perform physical activity. Some patients, particularly older people,¹ may have difficulty reaching an orgasm for medical or emotional reasons. In attempting to achieve a climax, it is possible that such individuals may exert themselves to a greater degree of exhaustion with relatively greater demand on their cardiovascular system (although specific data on this are lacking).

Sexual Activity and Cardiovascular Risk Sexual Activity and Angina

Coital angina ("angina d'amour"), angina that occurs during the minutes or hours after sexual activity, represents <5% of

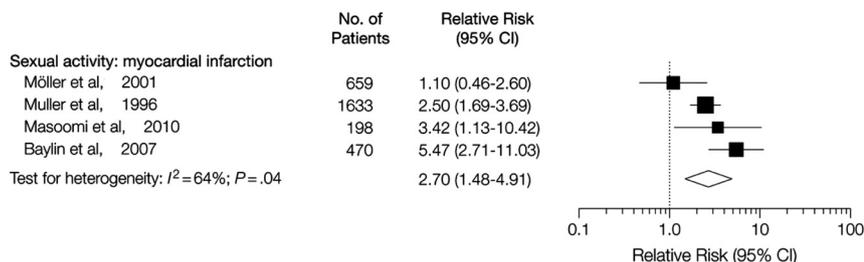


Figure. Forest plot of case-crossover studies assessing the association of sexual activity with myocardial infarction. CI indicates confidence interval. Modified from Dahabreh et al³⁰ with permission of the publisher. Copyright © 2011, American Medical Association. All rights reserved.³⁰

all anginal attacks.²⁸ It is rare in patients who do not have angina during strenuous physical exertion and more prevalent in sedentary individuals with severe coronary artery disease (CAD) who experience angina with minimal physical activity. If a patient can achieve an energy expenditure of ≥ 3 to 5 METs without demonstrating ischemia during exercise testing, then the risk for ischemia during sexual activity is very low.²⁹

Sexual Activity and Myocardial Infarction

Meta-analysis of 4 case-crossover studies, which consisted of 50% to 74% males predominantly in their 50s and 60s, showed that sexual activity was associated with a 2.70 increased relative risk of myocardial infarction (MI) compared with periods of time when the subjects were not engaged in sexual activity (Figure).³⁰ The relative risk of MI does not appear to be higher in subjects with a history of MI than in those without prior known CAD.³¹ Sedentary individuals have a relative risk of coital MI of 3.0, whereas physically active individuals have a relative risk of 1.2.³¹ The Stockholm Heart Epidemiology Programme (SHEEP) study of post-MI patients (50% women) similarly found that those who were sedentary had a higher risk of MI with sexual activity (relative risk 4.4) than did those who were physically active (relative risk 0.7).³²

Although sexual activity is associated with an increased risk of cardiovascular events, the absolute rate of events is miniscule because exposure to sexual activity is of short duration and constitutes a very small percentage of the total time at risk for myocardial ischemia or MI. Sexual activity is the cause of $<1\%$ of all acute MIs.³¹ The absolute risk increase for MI associated with 1 hour of sexual activity per week is estimated to be 2 to 3 per 10 000 person-years.³⁰ Individuals with higher habitual sexual activity levels experience smaller increases in risk than individuals with low activity levels. For the individual with a previous MI, the annual risk of reinfarction or death is estimated to be 10% (or as low as 3% if the individual has good exercise tolerance).³³ In such individuals, engaging in sexual activity transiently increases the risk of reinfarction or death from 10 chances in 1 million per hour to 20 to 30 chances in 1 million per hour.³¹

Sexual Activity and Ventricular Arrhythmias/Sudden Death

In an autopsy report of 5559 instances of sudden death, 34 (0.6%) reportedly occurred during sexual intercourse.³⁴ Two other autopsy studies reported similarly low rates (0.6%–1.7%) of sudden death related to sexual activity.^{35,36} Of the subjects who died during coitus, 82% to 93% were men, and

the majority (75%) were having extramarital sexual activity, in most cases with a younger partner in an unfamiliar setting and/or after excessive food and alcohol consumption. The increase in absolute risk of sudden death associated with 1 hour of additional sexual activity per week is estimated to be <1 per 10 000 person-years.³⁰

There are minimal data on the effect of sexual activity in patients with or at risk for ventricular arrhythmias. In a study of post-MI patients, sexual activity did not elicit an increase in ventricular ectopic activity compared with other activities.³⁷ In another report, the frequency of ventricular ectopy and other dysrhythmias was less during sexual activity than during standard exercise testing in male post-MI patients.³⁸ In a small study of 43 patients (8 females) with an internal cardioverter-defibrillator (ICD), the relative risk of tachyarrhythmic events was comparable during physical exertion, mental stress, and sexual activity.³⁹

Sexual Activity and CVD: General Recommendations

1. Women with CVD should be counseled regarding the safety and advisability of contraceptive methods and pregnancy when appropriate (*Class I; Level of Evidence C*).
2. It is reasonable that patients with CVD wishing to initiate or resume sexual activity be evaluated with a thorough medical history and physical examination (*Class IIa; Level of Evidence C*).
3. Sexual activity is reasonable for patients with CVD who, on clinical evaluation, are determined to be at low risk of cardiovascular complications (*Class IIa; Level of Evidence B*).^{30–32,40}
4. Exercise stress testing is reasonable for patients who are not at low cardiovascular risk or have unknown cardiovascular risk to assess exercise capacity and development of symptoms, ischemia, or arrhythmias (*Class IIa; Level of Evidence C*).
5. Sexual activity is reasonable for patients who can exercise ≥ 3 to 5 METs without angina, excessive dyspnea, ischemic ST-segment changes, cyanosis, hypotension, or arrhythmia (*Class IIa; Level of Evidence C*).⁵
6. Cardiac rehabilitation and regular exercise can be useful to reduce the risk of cardiovascular complications with sexual activity for patients with CVD (*Class IIa; Level of Evidence B*).^{31,41}
7. Patients with unstable, decompensated, and/or severe symptomatic CVD should defer sexual activity until their condition is stabilized and optimally managed (*Class III; Level of Evidence C*).

8. Patients with CVD who experience cardiovascular symptoms precipitated by sexual activity should defer sexual activity until their condition is stabilized and optimally managed (Class III; Level of Evidence C).

Men and women with stable CVD who have no or minimal symptoms during routine activities can engage in sexual activity. This includes patients with (1) Canadian Classification System class I or 2 angina; (2) New York Heart Association (NYHA) class I or II heart failure; (3) mild to moderate valvular disease; (4) no symptoms after MI; (5) successful coronary revascularization; (6) most types of congenital heart disease (CHD); and (7) ability to achieve ≥ 3 to 5 METS during exercise stress testing without angina, ischemic electrocardiographic changes, hypotension, cyanosis, arrhythmia, or excessive dyspnea. In patients with unstable or decompensated heart disease (ie, unstable angina, decompensated heart failure, uncontrolled arrhythmia, or significantly symptomatic and/or severe valvular disease), sexual activity should be deferred until the patient is stabilized and optimally managed. In patients whose exercise capacity or cardiovascular risk is unknown, exercise stress testing can be useful to assess exercise capacity and development of symptoms, ischemia, cyanosis, hypotension, or arrhythmias.

Exercise training during cardiac rehabilitation has been shown to increase maximum exercise capacity and decrease peak coital heart rate.⁴¹ Regular exercise is associated with a decreased risk of sexual activity–triggered MI.³¹ Thus, cardiac rehabilitation and regular exercise are reasonable strategies in patients with stable CVD who plan to engage in sexual activity.

In addition to the physical demands of sexual activity, the safety and advisability of contraceptive methods and pregnancy should be considered in women with CVD, especially those with CHD, valvular heart disease, or dilated cardiomyopathy.⁴² Combination hormonal oral contraceptives increase the risk of thromboembolic complications, and recommendations for their use in various cardiovascular conditions have been published.⁴² Pregnancy is associated with physiological changes that may adversely affect women with certain cardiac conditions and is of particular concern for those undergoing anticoagulation therapy with warfarin because it poses a risk to the fetus (ie, teratogenicity) and mother (ie, bleeding). Conversely, inadequate anticoagulation may lead to complications such as acute prosthetic valve thrombosis and thromboembolism.

Sexual Activity and Specific Cardiovascular Conditions

Coronary Artery Disease

Recommendations

- 1. Sexual activity is reasonable for patients with no or mild angina (Class IIa; Level of Evidence B).^{30–32,40}**
- 2. Sexual activity is reasonable 1 or more weeks after uncomplicated MI if the patient is without cardiac**

symptoms during mild to moderate physical activity (Class IIa; Level of Evidence C).

- 3. Sexual activity is reasonable for patients who have undergone complete coronary revascularization (Class IIa; Level of Evidence B)^{30–32,40} and may be resumed (a) several days after percutaneous coronary intervention (PCI) if the vascular access site is without complications (Class IIa; Level of Evidence C) or (b) 6 to 8 weeks after standard coronary artery bypass graft surgery (CABG), provided the sternotomy is well healed (Class IIa; Level of Evidence B).^{43,44}**
- 4. Sexual activity is reasonable for patients who have undergone noncoronary open heart surgery and may be resumed 6 to 8 weeks after the procedure, provided the sternotomy is well healed (Class IIa; Level of Evidence C).**
- 5. For patients with incomplete coronary revascularization, exercise stress testing can be considered to assess the extent and severity of residual ischemia (Class IIb; Level of Evidence C).**
- 6. Sexual activity should be deferred for patients with unstable or refractory angina until their condition is stabilized and optimally managed (Class III; Level of Evidence C).**

Stable Ischemic Heart Disease

For patients with stable ischemic heart disease, evaluation of their cardiovascular risk before they initiate or resume sexual activity is reasonable. Patients with mild, stable angina are considered to be at low risk for cardiovascular events, whereas those with unstable or refractory angina are considered to be at high risk.^{4,5} For patients whose symptoms are intermediate or whose risk cannot be determined during initial evaluation, exercise testing may (1) provide an objective assessment of exercise tolerance and capacity; (2) determine whether angina occurs with exertion (and at what level of exertion); and (3) assess the severity of ischemia with physical activity.

Previous MI

Patients with previous MI who are asymptomatic or have no ischemia with stress testing or who have undergone complete coronary revascularization are at low risk for coital MI. Before the routine use of reperfusion therapy, it was recommended that sexual activity be avoided for 6 to 8 weeks after MI. In 2005, the Princeton Conference suggested that post-MI patients who had undergone successful coronary revascularization or had a treadmill test without ischemia could resume sexual activity 3 to 4 weeks after MI.⁵ In contrast, the 2004 “ACC/AHA Guidelines for the Management of Patients with ST-elevation Myocardial Infarction” condoned sexual activity as early as 1 week after MI in the stable patient.¹⁵ Because participation of stable patients in cardiac rehabilitation exercise programs 1 week after MI has proved safe,⁴⁵ resumption of sexual activity soon after uncomplicated MI seems reasonable in the stable patient who is asymptomatic with mild to moderate physical activity (eg, 3–5 METS).

Post-PCI

The cardiovascular risk of sexual activity after PCI is likely related to the adequacy of coronary revascularization. Patients with complete revascularization should be able to

resume sexual activity within days of PCI, provided there are no complications related to femoral vascular access. Patients in whom there is reason to suspect a vascular complication should undergo appropriate evaluation before resuming sexual activity. Patients who undergo PCI via radial access should be able to resume sexual activity as early as if not earlier than those who undergo PCI via the femoral access. In patients with incomplete coronary revascularization, exercise stress testing may be of benefit in assessing the extent and severity of residual ischemia.

Post-CABG and Noncoronary Open Heart Procedures

CABG and most other heart surgeries (eg, valve repair/replacement) are commonly performed through a median sternotomy, with sternal healing typically complete, or nearly so, 8 weeks after surgery. Because sexual activity may involve considerable stress on the chest and breathing patterns that generate high intrathoracic pressures that could compromise sternal wound healing, it is generally recommended that sexual activity be delayed for 6 to 8 weeks after CABG and noncoronary open heart procedures. Patients who have undergone surgery should be counseled to avoid positions that cause discomfort or put undue stress on the surgical site, particularly in the early postoperative months. Physical vigor in such patients is best reintroduced in a gradual fashion. After successful recovery after CABG, sexual activity is usually resumed and sexual satisfaction is usually maintained for many patients.^{43,44}

Minimal access cardiac surgery that involves no or a limited sternotomy may allow earlier resumption of sexual activity. Robot-assisted surgery avoids a sternotomy incision and is an iteration of a less-invasive surgical procedure; patients treated with this procedure may similarly be able to resume sexual activity earlier than those undergoing median sternotomy.

CABG usually achieves complete or near-complete revascularization. In those in whom there is reason to believe there is significant incomplete revascularization (or graft failure), stress testing may be of benefit in assessing the extent and severity of residual ischemia.

Heart Failure

Recommendations

- 1. Sexual activity is reasonable for patients with compensated and/or mild (NYHA class I or II) heart failure (Class IIa; Level of Evidence B).**^{46–49}
- 2. Sexual activity is not advised for patients with decompensated or advanced (NYHA class III or IV) heart failure until their condition is stabilized and optimally managed (Class III; Level of Evidence C).**

Hemodynamic, vascular, hormonal, and neurohormonal abnormalities may contribute to the sexual dysfunction that commonly occurs in heart failure patients.⁵⁰ Approximately 60% to 87% of heart failure patients report sexual problems, including a marked decrease in sexual interest and activity, with one quarter reporting cessation of sexual activity altogether.^{51–53} Sexual function correlates with symptomatic status (ie, NYHA functional class and 6-minute walk test) but

not with ejection fraction.⁵² Interestingly, many heart failure patients place greater importance on improving quality of life (including sexual activity) than on improving survival.^{54,55}

Optimal medical treatment of heart failure patients increases the likelihood of safe and satisfactory sexual activity. Exercise training improves quality of life⁵⁶ in heart failure patients and may favorably impact their sexual activity.⁵⁷ Heart failure patients who experience shortness of breath or fatigue during sexual activity can be advised to use a semireclining or “on-bottom” position during coitus, which decreases the level of physical exertion, and to rest if dyspnea occurs.⁵⁸

The safety of sexual activity can reasonably be assumed to be related to the symptomatic severity of heart failure (ie, NYHA class) and whether or not the patient is decompensated (eg, volume overloaded). Studies involving stable heart failure patients have shown that it is safe for such patients to engage in sexual activity.^{46–49}

Valvular Heart Disease

Recommendations

- 1. Sexual activity is reasonable for patients with mild or moderate valvular heart disease and no or mild symptoms (Class IIa; Level of Evidence C).**
- 2. Sexual activity is reasonable for patients with normally functioning prosthetic valves, successfully repaired valves, and successful transcatheter valve interventions (Class IIa; Level of Evidence C).**
- 3. Sexual activity is not advised for patients with severe or significantly symptomatic valvular disease until their condition is stabilized and optimally managed (Class III; Level of Evidence C).**

Although recommendations on physical activity in patients with valvular heart disease are available,^{6,11,14} there are no published studies that specifically address the issue of sexual activity in such patients. Because patients with mild or moderate valve disease can safely participate in physical activities involving light or moderate exertion, it is also reasonable for such patients to engage in sexual activity, presuming that such activity does not precipitate significant cardiovascular symptoms. In patients with severe valvular disease with significant symptoms (or even mild symptoms with severe valvular aortic stenosis), it is prudent to defer sexual activity until medical or surgical treatment addresses these conditions. There is no reason to preclude sexual activity in patients with normally functioning prosthetic valves. The timing of return to sexual activity after surgical valve repair or replacement is discussed above in the section on Post-CABG and Noncoronary Open Heart Procedures.

In patients whose symptoms or valve disease severity are indeterminate and in those with asymptomatic severe valvular disease, exercise stress testing may provide an assessment of symptomatic and hemodynamic response to physical activity, as well as the possible precipitation of arrhythmias. It can be particularly helpful in assessing the individual with asymptomatic moderate or severe aortic stenosis and patients with severe valve dysfunction of other types who are asymptomatic. Exercise echocardiography can provide additional infor-

mation on the physiological response to exercise, including ventricular function, inducible increases in valve gradients, and inducible pulmonary hypertension.

The physiological effects of pregnancy are of particular concern in female patients with moderate to severe mitral or aortic stenosis and in those whose valvular lesions have caused symptoms, arrhythmias, pulmonary hypertension, ascending aortic dilation, or significant left ventricular dysfunction or dilation.⁵⁹ In addition, females with a mechanical prosthetic valve on warfarin therapy should be informed that warfarin poses a risk to the fetus (ie, teratogenicity) and mother (ie, bleeding), whereas inadequate anticoagulation may lead to acute valve thrombosis and thromboembolism.

Arrhythmias, Pacemakers, and ICDs

Recommendations

1. Sexual activity is reasonable for patients with atrial fibrillation or atrial flutter and well-controlled ventricular rate (*Class IIa; Level of Evidence C*).
2. Sexual activity is reasonable for patients with a history of atrioventricular nodal reentry tachycardia, atrioventricular reentry tachycardia, or atrial tachycardia with controlled arrhythmias (*Class IIa; Level of Evidence C*).
3. Sexual activity is reasonable for patients with pacemakers (*Class IIa; Level of Evidence C*).
4. Sexual activity is reasonable for patients with an ICD implanted for primary prevention (*Class IIa; Level of Evidence C*).
5. Sexual activity is reasonable for patients with an ICD used for secondary prevention in whom moderate physical activity (≥ 3 –5 METS) does not precipitate ventricular tachycardia or fibrillation and who do not receive frequent multiple appropriate shocks (*Level of Evidence C*).
6. Sexual activity should be deferred for patients with atrial fibrillation and poorly controlled ventricular rate, uncontrolled or symptomatic supraventricular arrhythmias, and spontaneous or exercise-induced ventricular tachycardia until the condition is optimally managed (*Class III; Level of Evidence C*).
7. Sexual activity should be deferred in patients with an ICD who have received multiple shocks until the causative arrhythmia is stabilized and optimally controlled (*Class III; Level of Evidence C*).

As discussed above, sudden death is an extremely rare occurrence during sexual activity in the general population. There are limited data on the incidence of arrhythmias induced with sexual activity in patients with a known history of arrhythmia. The risk of ventricular arrhythmia during sexual activity in patients with CVD, including those with an ICD, does not appear to be greater than during comparable physical exertion or exercise testing.^{38,39} Thus, it is reasonable to recommend that patients with arrhythmias who are considered safe to participate in leisure (or more active) sporting activities are able to participate in sexual activity. These would include patients with (1) atrial fibrillation or atrial flutter and a well-controlled ventricular response; (2) a history of atrioventricular nodal reentrant tachycardia, atrio-

ventricular reentry tachycardia, or atrial tachycardia with controlled arrhythmias; (3) a pacemaker; (4) an ICD implanted for primary prevention who have not received multiple shocks appropriate to the patient's arrhythmia; and (5) an ICD implanted for secondary prevention in whom comparable levels of physical activity do not precipitate ventricular tachycardia or ventricular fibrillation and who do not receive frequent appropriate shocks.^{4,12,13,60} In patients who have received multiple ICD shocks, it is prudent to first stabilize and optimally control the arrhythmia (and underlying cause) before the patient engages in sexual activity. A history of multiple shocks per se is not necessarily a contraindication to the patient ever engaging in sexual activity.

As noted above, the presence of an ICD is not a contraindication for sexual activity, and it is reasonable for most patients with an ICD to continue sexual activity. In the patient with an ICD, partner overprotectiveness and the fear of shock with sexual activity are important concerns for the patient and his or her partner.^{61,62} Accordingly, sexual activity often decreases after ICD implantation.^{62–64} The sexual partner is not believed to be at risk from defibrillation if the ICD discharges during sexual activity.^{61,64} Stress testing may provide reassurance to the patient and spouse or partner that sexual activity is unlikely to precipitate or exacerbate arrhythmia.⁶⁵ Strategies are available for healthcare specialists to use in counseling ICD patients and their partners,^{61,64} and an excellent "Cardiology Patient Page" (<http://circ.ahajournals.org/content/122/13/e465.long>) addresses the concerns of patients and partners.⁶¹

Congenital Heart Disease

Recommendation

1. Sexual activity is reasonable for most CHD patients who do not have decompensated or advanced heart failure, severe and/or significantly symptomatic valvular disease, or uncontrolled arrhythmias (*Class IIa; Level of Evidence C*).

There are estimated to be more than 1 million CHD patients >21 years of age in the United States. Patients with simple, as well as more complex, disease are at risk for atrial and ventricular arrhythmias, stroke, and rarely coronary ischemia. To date, however, there are only rare reported deaths or strokes during sexual activity in this population. In 1 study, 9% of women with CHD reported symptoms during sexual activity, which included dyspnea, perceived arrhythmia, increased fatigue, or syncope.⁶⁶ Symptoms were more common in those with severe lesions, worse functional status, or cyanosis. In a survey of men with CHD, 9% reported dyspnea, 9% reported subjective arrhythmias, and 5% reported chest pain with sexual activity, with symptoms more common in patients with greater functional impairment (NYHA class III).⁶⁷

Published guidelines allow for unlimited physical activity in asymptomatic CHD patients with closed or small atrial or ventricular septal defects, mild coarctation of the aorta, closed patent ductus arteriosus, and other mild congenital defects with normal right-sided heart volume, no pulmonary

hypertension, and no significant outflow obstruction on the right or left side of the heart.^{7,11} On the basis of these recommendations, sexual activity appears to be reasonable in most patients with CHD. Patients in whom the safety of sexual activity is less certain or unclear include those with significant pulmonary hypertension, cyanotic heart disease, severe left-sided heart outflow obstruction, uncontrolled arrhythmias, and anomalous coronary artery passing between the pulmonary artery and aorta.

Issues regarding contraception and pregnancy are particularly important in women with CHD. A study of women with CHD lesions associated with a high risk of pregnancy-related cardiovascular complications showed that 28% were not using adequate birth control methods, 20% were using methods considered contraindicated for their condition, 43% had not been counseled about contraception, and 48% had not been informed of pregnancy-related risks.⁶⁶

Hypertrophic Cardiomyopathy

Recommendations

1. **Sexual activity is reasonable for most patients with hypertrophic cardiomyopathy (HCM) (Class IIa; Level of Evidence C).**
2. **Sexual activity should be deferred for patients with HCM who are severely symptomatic until their condition is stabilized (Class III; Level of Evidence C).**

HCM is a heterogeneous genetic cardiac disease and the most common cause of arrhythmia-related sudden cardiac death in the young,⁶⁸ including competitive athletes.⁶⁹ Approximately 70% of HCM patients may have left ventricular outflow obstruction either at rest⁷⁰ or with physiological provocation,⁷¹ independent of whether limiting symptoms are present. The underlying arrhythmogenic substrate in HCM is unpredictable,^{72,73} but a linkage between physical activity and sudden death events attributable to ventricular tachycardia/ventricular fibrillation⁶⁹ raises concern that vigorous sexual activity might heighten risk in patients with this disease. However, there are no documented cases of cardiac arrest related to sexual activity in HCM patients. This is consistent with the physical activity recommendations afforded HCM patients that only prohibit participation in intense competitive sports or activities that mimic such forms of exercise.⁶⁰

Cardiovascular Drugs and Sexual Function

Recommendation

1. **Cardiovascular drugs that can improve symptoms and survival should not be withheld because of concerns about the potential impact on sexual function (Class III: Harm; Level of Evidence C).**

Numerous classes of cardiovascular drugs, particularly diuretics and β -blockers, have been implicated in causing erectile dysfunction (ED)^{74–79}; however, recent studies and reviews have not found clear relationships between many contemporary cardiovascular drugs and ED.^{76–78,80–83} An

analysis of 6 studies involving almost 15 000 people found β -blocker therapy increased the annual reported rate of sexual dysfunction by only 5 reports per 1000 patients and the annual reported rate of impotence by only 3 per 1000 patients.⁸⁴ In addition, a nocebo effect, in which a patient's knowledge that a drug has been associated with ED, is often at least as important a contributing factor to a patient's ED as any physiological effect, particularly with contemporary β -blockers.^{82,85,86} In one of the few studies addressing sexual function in women, antihypertensive therapies did not appear to adversely impact sexual function,⁷⁶ although thiazide diuretics and aldosterone may be associated with decreased vaginal lubrication or menstrual irregularities.^{79,87}

Cardiovascular drugs that can improve symptoms or survival should not be withheld because of concerns about their adverse impact on sexual function. If a patient being treated with a cardiovascular drug complains of sexual dysfunction, efforts should be made to assess whether the sexual dysfunction is more likely related to underlying vascular or cardiac disease, the nocebo effect, or anxiety or depression, as discussed below. There are no good data to recommend a specific class of cardiovascular drug to improve sexual function or activity in patients with CVD.

In patients who clearly develop ED as a result of thiazide diuretic therapy, it is reasonable to switch to a loop diuretic. Some male patients treated with spironolactone may experience antiandrogen side effects (eg, ED, decreased libido, and gynecomastia) that compromise their sexual function and activity, in which case eplerenone may be a reasonable alternative. In male patients with clearly established β -blocker-induced sexual dysfunction, nebivolol (which has nitric oxide-mediated vasodilating properties and a lower incidence of ED than other β -blockers) may be considered provided the β -blocker is not being administered specifically for survival improvement for the patient with systolic heart failure or after MI.^{88,89} Treatment of ED with a phosphodiesterase-5 (PDE5) inhibitor (discussed below) may be a reasonable alternate strategy.

Pharmacotherapy for Sexual Dysfunction

PDE5 Inhibitors

Recommendations

1. **PDE5 inhibitors are useful for the treatment of ED in patients with stable CVD (Class I; Level of Evidence A).**^{5,46,48,49,90–106}
2. **The safety of PDE5 inhibitors is unknown in patients with severe aortic stenosis or HCM (Class IIb; Level of Evidence C).**
3. **PDE5 inhibitors should not be used in patients receiving nitrate therapy (Class III; Level of Evidence B).**^{98,107,108}
4. **Nitrates should not be administered to patients within 24 hours of sildenafil or vardenafil administration or within 48 hours of tadalafil administration (Class III; Level of Evidence B).**^{98,107,108}

PDE5 inhibitors are effective for the treatment of ED.^{90–92} PDE5 inhibitors prevent the breakdown of cyclic GMP, thereby result-

ing in increased nitric oxide concentration and vasodilation, which enhances erectile function. Three PDE5 inhibitors are approved for the treatment of ED in the United States: sildenafil, tadalafil, and vardenafil. Sildenafil and tadalafil are also approved for the treatment of pulmonary hypertension. Sildenafil and vardenafil are relatively short-acting, with half-lives of approximately 4 hours; tadalafil is long-acting, with a half-life of 17.5 hours. These agents cause systemic vasodilation and mild reductions in systolic (≤ 10 mm Hg) and diastolic (≤ 8 mm Hg) systemic blood pressure.^{27,91,93,94,98} These reductions may be greater in patients with underlying CAD and higher baseline blood pressures.⁹¹

PDE5 inhibitors are generally safe and effective for the treatment of ED in patients with systemic arterial hypertension, stable CAD, and compensated heart failure.^{5,46,48,49,90–106} No studies have shown one agent to be more effective or safer than the others. Despite occasional anecdotal case reports linking PDE5 inhibitors to cardiac events, large trials and meta-analyses suggest that they are not associated with an increase in MI or cardiac events.^{91,95,99,103} When administered with cardiovascular drugs that reduce systemic blood pressure, PDE5 inhibitors are associated with small additive reductions in systemic blood pressure but no increase in adverse cardiac events.^{93,94,109} In some patients, the concomitant use of PDE5 inhibitors and α -blocking agents may result in symptomatic hypotension.¹¹⁰ Thus, when both are indicated, the lowest α -blocker dose should be initiated and tolerated by the patient before the patient begins the lowest dose of a PDE5 inhibitor. PDE5 inhibitors should not be administered to treat ED in patients who are already receiving PDE5 inhibitor therapy for pulmonary hypertension. Vardenafil (but not sildenafil or tadalafil) carries a precautionary statement about prolongation of the corrected QT interval and should be avoided in patients with congenital QT prolongation or a history of torsade de pointes and in those taking medications known to prolong the QT interval (eg, class IA or III antiarrhythmic agents).

Organic nitrates (both short-acting forms such as sublingual nitroglycerin and long-acting forms such as isosorbide mononitrate) are nitric oxide donors and remain an absolute contraindication to PDE5 inhibitor use, because this combination may result in unpredictable and precipitous reductions in systemic blood pressure.¹⁰⁷ Patients with chest pain or acute MI should not be administered nitrates until at least 24 hours after the last dose of sildenafil or vardenafil⁹⁸ and until 48 hours after the last dose of tadalafil.¹⁰⁸ Healthcare providers should question patients presenting with chest pain about PDE5 inhibitor use before administering nitrates. Other than nitrates, patients with chest pain or an acute coronary syndrome who have taken a PDE5 inhibitor can be treated with all other cardiovascular medications. In patients undergoing chronic nitrate therapy who desire to use PDE5 inhibitors, the need for continued nitrate therapy (or an alternate therapy) should be evaluated, particularly in patients who have undergone complete revascularization.

The writing group is unaware of any reported deaths attributable to PDE5 inhibitor use in patients with left ventricular outflow obstruction (fixed or dynamic). As with all vasodilators, caution is advised when the use of these

drugs is considered in patients with severe aortic stenosis or HCM.

PDE5 inhibitor use has been explored in females for treatment of arousal disorders and has largely been shown to be no more effective than placebo.¹¹¹ The safety of PDE5 inhibitor use in females with CVD has not been established.

Local and Topical Estrogen Therapy

Recommendation

- 1. Nonsystemic (local or topical) estrogen use for the treatment of dyspareunia in women with CVD is reasonable (Class IIa; Level of Evidence C).**

Vaginal dryness and pain with sexual intercourse are prevalent symptoms among sexually active postmenopausal women.¹ Estrogen administered via the vaginal route is an effective and Food and Drug Administration–approved treatment for relief of symptoms of vaginal atrophy typically seen in menopausal and postmenopausal women. Topical estrogen preparations can also be used on the vulva to treat insertional pain at the vaginal introitus. Concerns about estrogen therapy and increased cardiovascular risk were raised by the results of several large trials of women who received oral combination therapy with estrogen and progesterone^{112,113}; however, trials with estrogen therapy alone have not reported increased cardiac risk.^{114,115} Because systemic absorption with vaginal administration is minimal,¹¹⁶ and focal vulval application is expected to be even less, topical estrogen therapy is unlikely to pose any cardiac risk in women with CVD.

Herbal Medications

Recommendation

- 1. It may be reasonable to caution patients with CVD regarding the potential for adverse events with the use of herbal medications with unknown ingredients that are taken for treatment of sexual dysfunction (Class IIb; Level of Evidence C).**

Numerous herbal medications are advertised to patients for the treatment of sexual dysfunction. Some of these medications may contain drugs, such as PDE5 inhibitors (or chemically similar substances),^{117,118} yohimbine,¹¹⁹ or L-arginine.¹²⁰ Such drugs can interact with cardiovascular medications, have vasoactive or sympathomimetic properties, can elevate or reduce systemic blood pressure, or have been associated with adverse outcome in patients with CAD.^{98,107,119–122} Therefore, it may be reasonable to caution patients with CVD about the use of herbal medications with unknown ingredients advertised for the treatment of sexual dysfunction.

Psychological Issues of Sexual Activity and CVD

Recommendation

- 1. Anxiety and depression regarding sexual activity should be assessed in patients with CVD (Class I; Level of Evidence B).^{2,3}**

Psychological distress¹²³ and decreased sexual function or activity^{2,3} are associated¹²⁴ and are common in patients with CVD. In patients with CAD,^{2,3} heart failure,⁵¹ CHD,⁶⁷ recent MI,¹²⁵ CABG,¹²⁶ ICD implantation,⁶⁴ or cardiac transplantation,¹²⁷ sexual activity frequency and satisfaction often decline because of anxiety on the part of the patient or partner that sexual activity will worsen the underlying cardiac condition or cause death.^{3,128} Changes in sexual activity after a cardiac event may impair the patient's quality of life, negatively affect psychological health, and strain marital or other important intimate relationships,^{67,125,127} which in turn may lead to depression and anxiety. The resultant depression may be an important contributing cause of ED in men and of female sexual problems, including decreased libido, difficulty with arousal and orgasm, and dyspareunia.^{2,67,124,129}

Patient and Partner Counseling

Recommendation

- 1. Patient and spouse/partner counseling by healthcare providers is useful to assist in resumption of sexual activity after an acute cardiac event, new CVD diagnosis, or ICD implantation (Class I; Level of Evidence B).**^{130–134}

Although sexual counseling of patients and partners with CVD is an important component of recovery, it is rarely provided.^{135–138} Potential reasons for this include the provider's lack of experience or comfort discussing sexual issues, inadequate knowledge on issues regarding sexual activity and CVD, and limited time.^{135–138} Studies show that most CVD patients (and their partners) believe they have been inadequately educated on this topic by healthcare providers^{139,140} and desire more information on how to resume their normal sexual activity.^{140–142} Partners of patients with CVD often have considerable anxiety about sexual activity, which may adversely impact the sexual activity of the couple.^{62,143,144} When information on sexual activity is provided to the patient, it is more likely to be provided in written form than verbally, more likely to be provided to men than to women, and rarely provided to the partner.¹⁴⁵

A discussion about sexual activity is appropriate for men and women of all ages who have CVD. Initiation of a discussion of sexual issues by the healthcare provider can facilitate an open discussion of the patient's and partner's sexual concerns.¹⁴⁶ Questionnaires can be used to facilitate a discussion of sexual issues.¹⁴⁷ General suggestions to the

patient may include being well rested at the time of sexual activity, avoiding unfamiliar surroundings and partners to minimize stress during sexual activity, avoiding heavy meals or alcohol before sexual activity, and using a position that does not restrict respiration.^{79,130,142,148} The achievement of orgasm may require a greater degree of exertion and may not be a realistic initial goal in some patients. Randomized trials have demonstrated that sexual counseling in patients with CVD results in increased knowledge, a higher likelihood of return to sexual activity, improved sexual desire and satisfaction, and increased confidence and reduced fear in resuming sexual activity.^{130–134}

Summary

Sexual activity is an important component of patient and partner quality of life, and it is reasonable for most patients with CVD to engage in sexual activity. It is reasonable that patients with CVD who wish to engage in sexual activity undergo a comprehensive history and physical examination beforehand. Those with stable symptoms and good functional capacity generally have a low risk of adverse cardiovascular events with sexual activity. Patients with unstable or severe symptoms should first be treated and stabilized before engaging in sexual activity. Exercise testing can provide additional information as to the safety of sexual activity in patients with indeterminate or unclear risk.

Cardiovascular medications are uncommonly the true cause of ED, and those that can improve symptoms and survival should not be withheld because of concerns about the potential impact on sexual function. PDE5 inhibitors have proved safe and effective in many patients with stable CVD; however, nitrate use is an absolute contraindication for PDE5 inhibitor administration.

Anxiety and depression are important considerations in patients with CVD and can contribute to reduced or impaired sexual activity. Sexual counseling of CVD patients and their partners is an important component of recovery; unfortunately, it is rarely provided.

Further research is needed on sexual activity in specific cardiovascular conditions, particularly with regard to the effects of sexual activity in females and in older adults. When possible, pharmacotherapy, device and surgical intervention, registries, and longitudinal studies of patients with CVD should specifically include data on sexual activity and function. Future studies of interventions to improve sexual activity in the context of CVD, including sexual counseling, should address sexual concerns and activity of both men and women, young and old, and both patients and partners.

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References

- Lindau ST, Schumm LP, Laumann EO, Levinson W, O'Muircheartaigh CA, Waite LJ. A study of sexuality and health among older adults in the United States. *N Engl J Med*. 2007;357:762–774.
- Kriston L, Gunzler C, Agyemang A, Bengel J, Berner MM; SPARK Study Group. Effect of sexual function on health-related quality of life mediated by depressive symptoms in cardiac rehabilitation: findings of the SPARK project in 493 patients. *J Sex Med*. 2010;7:2044–2055.
- Friedman S. Cardiac disease, anxiety, and sexual functioning. *Am J Cardiol*. 2000;86:46F–50F.
- Debusk R, Drory Y, Goldstein I, Jackson G, Kaul S, Kimmel SE, Kostis JB, Kloner RA, Lakin M, Meston CM, Mittleman M, Muller JE, Padma-Nathan H, Rosen RC, Stein RA, Zusman R. Management of sexual dysfunction in patients with cardiovascular disease: recommendations of The Princeton Consensus Panel. *Am J Cardiol*. 2000;86:175–181.
- Kostis JB, Jackson G, Rosen R, Barrett-Connor E, Billups K, Burnett AL, Carson C 3rd, Cheitlin M, Debusk R, Fonseca V, Ganz P, Goldstein I, Guay A, Hatzichristou D, Hollander JE, Hutter A, Katz S, Kloner RA, Mittleman M, Montorsi F, Montorsi P, Nehra A, Sadowsky R, Shabsigh R. Sexual dysfunction and cardiac risk (the Second Princeton Consensus Conference). *Am J Cardiol*. 2005;96:313–321.
- Bonow RO, Cheitlin MD, Crawford MH, Douglas PS. Task Force 3: valvular heart disease. *J Am Coll Cardiol*. 2005;45:1334–1340.
- Graham TP Jr, Driscoll DJ, Gersony WM, Newburger JW, Rocchini A, Towbin JA. Task Force 2: congenital heart disease. *J Am Coll Cardiol*. 2005;45:1326–1333.
- Zipes DP, Ackerman MJ, Estes NA 3rd, Grant AO, Myerburg RJ, Van Hare G. Task Force 7: arrhythmias. *J Am Coll Cardiol*. 2005;45:1354–1363.
- Thompson PD, Balady GJ, Chaitman BR, Clark LT, Levine BD, Myerburg RJ. Task Force 6: coronary artery disease. *J Am Coll Cardiol*. 2005;45:1348–1353.
- Maron BJ, Ackerman MJ, Nishimura RA, Pyeritz RE, Towbin JA, Udelson JE. Task Force 4: HCM and other cardiomyopathies, mitral valve prolapse, myocarditis, and Marfan syndrome. *J Am Coll Cardiol*. 2005;45:1340–1345.
- Pelliccia A, Fagard R, Bjørnstad HH, Anastassakis A, Arbustini E, Assanelli D, Biffi A, Borjesson M, Carrè F, Corrado D, Delise P, Dorwarth U, Hirth A, Heidbuchel H, Hoffmann E, Mellwig KP, Panhuyzen-Goedkoop N, Pisani A, Solberg EE, van-Buuren F, Vanhees L, Blomstrom-Lundqvist C, Deligiannis A, Dugmore D, Glikson M, Hoff PI, Hoffmann A, Hoffmann E, Horstkotte D, Nordrehaug JE, Oudhof J, McKenna WJ, Penco M, Priori S, Reybrouck T, Senden J, Spataro A, Thiene G; Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology; Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. Recommendations for competitive sports participation in athletes with cardiovascular disease: a consensus document from the Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J*. 2005;26:1422–1445.
- Heidbuchel H, Corrado D, Biffi A, Hoffmann E, Panhuyzen-Goedkoop N, Hoogsteen J, Delise P, Hoff PI, Pelliccia A; Study Group on Sports Cardiology of the European Association for Cardiovascular Prevention and Rehabilitation. Recommendations for participation in leisure-time physical activity and competitive sports of patients with arrhythmias and potentially arrhythmogenic conditions, part II: ventricular arrhythmias, channelopathies and implantable defibrillators. *Eur J Cardiovasc Prev Rehabil*. 2006;13:676–686.
- Heidbuchel H, Panhuyzen-Goedkoop N, Corrado D, Hoffmann E, Biffi A, Delise P, Blomstrom-Lundqvist C, Vanhees L, Ivarhoff P, Dorwarth U, Pelliccia A; Study Group on Sports Cardiology of the European Association for Cardiovascular Prevention and Rehabilitation. Recommendations for participation in leisure-time physical activity and competitive sports in patients with arrhythmias and potentially arrhythmogenic conditions, part I: supraventricular arrhythmias and pacemakers. *Eur J Cardiovasc Prev Rehabil*. 2006;13:475–484.
- Bonow RO, Carabello BA, Chatterjee K, de Leon AC Jr, Faxon DP, Freed MD, Gaasch WH, Lytle BW, Nishimura RA, O'Gara PT, O'Rourke RA, Otto CM, Shah PM, Shanewise JS. 2008 Focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1998 guidelines for the management of patients with valvular heart disease). *Circulation*. 2008;118:e523–e661.
- Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, Hochman JS, Krumholz HM, Kushner FG, Lamas GA, Mullany CJ, Ornato JP, Pearle DL, Sloan MA, Smith SC Jr, Alpert JS, Anderson JL, Faxon DP, Fuster V, Gibbons RJ, Gregoratos G, Halperin JL, Hiratzka LF, Hunt SA, Jacobs AK. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction) [published corrections appear in *Circulation*. 2005;111:2013–2014; *Circulation*. 2007;115:e411; and *Circulation*. 2010;121:e441]. *Circulation*. 2004;110:e82–e292.
- Anderson JL, Adams CD, Antman EM, Bridges CR, Califf RM, Casey DE Jr, Chavey WE 2nd, Fesmire FM, Hochman JS, Levin TN, Lincoff AM, Peterson ED, Theroux P, Wenger NK, Wright RS. ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non-ST-Elevation Myocardial Infarction). *Circulation*. 2007;116:803–877.
- Heart Failure Society of America; Lindenfeld J, Albert NM, Boehmer JP, Collins SP, Ezekowitz JA, Givertz MM, Katz SD, Klapholz M, Moser DK, Rogers JG, Starling RC, Stevenson WG, Tang WH, Teerlink JR, Walsh MN. HFSA 2010 comprehensive heart failure practice guideline. *J Card Fail*. 2010;16:e1–e194.
- Bohlen JG, Held JP, Sanderson MO, Patterson RP. Heart rate, rate-pressure product, and oxygen uptake during four sexual activities. *Arch Intern Med*. 1984;144:1745–1748.
- Chen X, Zhang Q, Tan X. Cardiovascular effects of sexual activity. *Indian J Med Res*. 2009;130:681–688.
- Krüger T, Exton MS, Pawlak C, von zur Mühlen A, Hartmann U, Schedlowski M. Neuroendocrine and cardiovascular response to sexual arousal and orgasm in men. *Psychoneuroendocrinology*. 1998;23:401–411.
- Exton MS, Bindert A, Kruger T, Scheller F, Hartmann U, Schedlowski M. Cardiovascular and endocrine alterations after masturbation-induced orgasm in women. *Psychosom Med*. 1999;61:280–289.
- Stein RA. Cardiovascular response to sexual activity. *Am J Cardiol*. 2000;86:27F–29F.
- Little WA, Honour AJ, Sleight P. Direct arterial pressure, heart rate and electrocardiogram during human coitus. *J Reprod Fertil*. 1974;40:321–331.
- Carmichael MS, Warburton VL, Dixon J, Davidson JM. Relationships among cardiovascular, muscular, and oxytocin responses during human sexual activity. *Arch Sex Behav*. 1994;23:59–79.
- Exton NG, Truong TC, Exton MS, Wingenfeld SA, Leygraf N, Saller B, Hartmann U, Schedlowski M. Neuroendocrine response to film-induced sexual arousal in men and women. *Psychoneuroendocrinology*. 2000;25:187–199.
- Hellerstein HK, Friedman EH. Sexual activity and the postcoronary patient. *Arch Intern Med*. 1970;125:987–999.
- Cheitlin MD. Sexual activity and cardiac risk. *Am J Cardiol*. 2005;96:24M–28M.
- DeBusk RF. Sexual activity in patients with angina. *JAMA*. 2003;290:3129–3132.
- Drory Y. Sexual activity and cardiovascular risk. *Eur Heart J Suppl*. 2002;4(suppl H):H13–H18.
- Dahabreh IJ, Paulus JK. Association of episodic physical and sexual activity with triggering of acute cardiac events: systematic review and meta-analysis. *JAMA*. 2011;305:1225–1233.
- Muller JE, Mittleman MA, Maclure M, Sherwood JB, Tofler GH; Determinants of Myocardial Infarction Onset Study Investigators. Triggering myocardial infarction by sexual activity: low absolute risk and prevention by regular physical exertion. *JAMA*. 1996;275:1405–1409.
- Möller J, Ahlbom A, Hulting J, Diderichsen F, de Faire U, Reuterwall C, Hallqvist J. Sexual activity as a trigger of myocardial infarction: a case-crossover analysis in the Stockholm Heart Epidemiology Programme (SHEEP). *Heart*. 2001;86:387–390.
- Moss AJ, Benhorin J. Prognosis and management after a first myocardial infarction. *N Engl J Med*. 1990;322:743–753.

34. Ueno M. The so-called coition death [in Japanese]. *Nihon Hoigaku Zasshi*. 1963;17:330–340.
35. Krauland W, Underwarter T. Herzinfarkt und Sexualität aus der Sicht des Rechtsmediziners [in German]. *Sexualmedizin*. 1976;10:xx–xxiii.
36. Parzeller M, Raschka C, Bratzke H. Sudden cardiovascular death during sexual intercourse: results of a legal medicine autopsy study [in German]. *Z Kardiol*. 1999;88:44–48.
37. Paolillo V, Marra S, Chiappa E, Boncompagni F, Oddenino G, Spadaccini F, Angelino PF. Influence of sleep, wakefulness and some daily activities on ventricular ectopic activity in recent myocardial infarction (author's translation) [in Italian]. *G Ital Cardiol*. 1981;11:12–22.
38. Kavanagh T, Shephard RJ. Sexual activity after myocardial infarction. *Can Med Assoc J*. 1977;116:1250–1253.
39. Fries R, König J, Schäfers HJ, Böhm M. Triggering effect of physical and mental stress on spontaneous ventricular tachyarrhythmias in patients with implantable cardioverter-defibrillators. *Clin Cardiol*. 2002;25:474–478.
40. DeBusk RF, Blomqvist CG, Kouchoukos NT, Luepker RV, Miller HS, Moss AJ, Pollock ML, Reeves TJ, Selvester RH, Stason WB, Wagner GS, Willman VL. Identification and treatment of low-risk patients after acute myocardial infarction and coronary-artery bypass graft surgery. *N Engl J Med*. 1986;314:161–166.
41. Stein RA. The effect of exercise training on heart rate during coitus in the post myocardial infarction patient. *Circulation*. 1977;55:738–740.
42. Sable C, Foster E, Uzark K, Bjornsen K, Canobbio MM, Connolly HM, Graham TP, Gurvitz MZ, Kovacs A, Meadows AK, Reid GJ, Reiss JG, Rosenbaum KN, Sagerman PJ, Saidi A, Schonberg R, Shah S, Tong E, Williams RG. Best practices in managing transition to adulthood for adolescents with congenital heart disease: the transition process and medical and psychosocial issues: a scientific statement from the American Heart Association. *Circulation*. 2011;123:1454–1485.
43. Papadopoulos C, Shelley SI, Piccolo M, Beaumont C, Barnett L. Sexual activity after coronary bypass surgery. *Chest*. 1986;90:681–685.
44. Lukkariinen H, Lukkariinen O. Sexual satisfaction among patients after coronary bypass surgery or percutaneous transluminal angioplasty: eight-year follow-up. *Heart Lung*. 2007;36:262–269.
45. Clark AM, Scott J, Schopflocher D, Myers J, Paterson I, Warburton D, Jones L, Haykowsky M. A meta-analysis of the effects of exercise training on left ventricular remodeling following myocardial infarction: start early and go longer for greatest exercise benefits on mortality. *Eur J Cardiovasc Prev Rehabil*. 2011;18(suppl 1):S1. Abstract P111.
46. Bocchi EA, Guimarães G, Mocelin A, Bacal F, Bellotti G, Ramires JF. Sildenafil effects on exercise, neurohormonal activation, and erectile dysfunction in congestive heart failure: a double-blind, placebo-controlled, randomized study followed by a prospective treatment for erectile dysfunction. *Circulation*. 2002;106:1097–1103.
47. Freitas D, Athanazio R, Almeida D, Dantas N, Reis F. Sildenafil improves quality of life in men with heart failure and erectile dysfunction. *Int J Impot Res*. 2006;18:210–212.
48. Katz SD, Parker JD, Glasser DB, Bank AJ, Sherman N, Wang H, Sweeney M. Efficacy and safety of sildenafil citrate in men with erectile dysfunction and chronic heart failure. *Am J Cardiol*. 2005;95:36–42.
49. Webster LJ, Michelakis ED, Davis T, Archer SL. Use of sildenafil for safe improvement of erectile function and quality of life in men with New York Heart Association classes II and III congestive heart failure: a prospective, placebo-controlled, double-blind crossover trial. *Arch Intern Med*. 2004;164:514–520.
50. Rastogi S, Rodriguez JJ, Kapur V, Schwarz ER. Why do patients with heart failure suffer from erectile dysfunction? A critical review and suggestions on how to approach this problem. *Int J Impot Res*. 2005;17(suppl 1):S25–S36.
51. Jaarsma T. Sexual problems in heart failure patients. *Eur J Cardiovasc Nurs*. 2002;1:61–67.
52. Jaarsma T, Dracup K, Walden J, Stevenson LW. Sexual function in patients with advanced heart failure. *Heart Lung*. 1996;25:262–270.
53. Schwarz ER, Kapur V, Bionat S, Rastogi S, Gupta R, Rosanio S. The prevalence and clinical relevance of sexual dysfunction in women and men with chronic heart failure. *Int J Impot Res*. 2008;20:85–91.
54. Stanek EJ, Oates MB, McGhan WF, Denofrio D, Loh E. Preferences for treatment outcomes in patients with heart failure: symptoms versus survival. *J Card Fail*. 2000;6:225–232.
55. Steinke EE, Wright DW, Chung ML, Moser DK. Sexual self-concept, anxiety, and self-efficacy predict sexual activity in heart failure and healthy elders. *Heart Lung*. 2008;37:323–333.
56. Flynn KE, Piña IL, Whellan DJ, Lin L, Blumenthal JA, Ellis SJ, Fine LJ, Howlett JG, Keteyian SJ, Kitzman DW, Kraus WE, Miller NH, Schulman KA, Spertus JA, O'Connor CM, Weinfurt KP; HF-ACTION Investigators. Effects of exercise training on health status in patients with chronic heart failure: HF-ACTION randomized controlled trial [published correction appears in *JAMA*. 2009;302:2322]. *JAMA*. 2009;301:1451–1459.
57. Belardinelli R, Lacalaprice F, Faccenda E, Purcaro A, Perna G. Effects of short-term moderate exercise training on sexual function in male patients with chronic stable heart failure. *Int J Cardiol*. 2005;101:83–90.
58. Steinke EE. Intimacy needs and chronic illness: strategies for sexual counseling and self-management. *J Gerontol Nurs*. 2005;31:40–50.
59. Bowater SE, Thorne SA. Management of pregnancy in women with acquired and congenital heart disease. *Postgrad Med J*. 2010;86:100–105.
60. Pelliccia A, Zipes DP, Maron BJ. Bethesda Conference #36 and the European Society of Cardiology Consensus Recommendations revisited: a comparison of U.S. and European criteria for eligibility and disqualification of competitive athletes with cardiovascular abnormalities. *J Am Coll Cardiol*. 2008;52:1990–1996.
61. Vazquez LD, Sears SF, Shea JB, Vazquez PM. Sexual health for patients with an implantable cardioverter defibrillator. *Circulation*. 2010;122:e465–e467.
62. Steinke EE. Sexual concerns of patients and partners after an implantable cardioverter defibrillator. *Dimens Crit Care Nurs*. 2003;22:89–96.
63. Mickley H, Petersen J, Nielsen BL. Subjective consequences of permanent pacemaker therapy in patients under the age of retirement. *Pacing Clin Electrophysiol*. 1989;12:401–405.
64. Steinke EE, Gill-Hopple K, Valdez D, Wooster M. Sexual concerns and educational needs after an implantable cardioverter defibrillator. *Heart Lung*. 2005;34:299–308.
65. Sears SF, Kovacs AH, Conti JB, Handberg E. Expanding the scope of practice for cardiac rehabilitation: managing patients with implantable cardioverter defibrillators. *J Cardiopulm Rehabil*. 2004;24:209–215.
66. Vigil M, Kaemmerer M, Seifert-Klauss V, Niggemeyer E, Nagdyman N, Trigas V, Bauer U, Schneider KT, Berger F, Hess J, Kaemmerer H. Contraception in women with congenital heart disease. *Am J Cardiol*. 2010;106:1317–1321.
67. Vigil M, Hager A, Bauer U, Niggemeyer E, Wittstock B, Köhn FM, Hess J, Kaemmerer H. Sexuality and subjective wellbeing in male patients with congenital heart disease. *Heart*. 2009;95:1179–1183.
68. Maron BJ. Hypertrophic cardiomyopathy: a systematic review. *JAMA*. 2002;287:1308–1320.
69. Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980–2006. *Circulation*. 2009;119:1085–1092.
70. Maron MS, Olivetto I, Betocchi S, Casey SA, Lesser JR, Losi MA, Cecchi F, Maron BJ. Effect of left ventricular outflow tract obstruction on clinical outcome in hypertrophic cardiomyopathy. *N Engl J Med*. 2003;348:295–303.
71. Maron MS, Olivetto I, Zenovich AG, Link MS, Pandian NG, Kuvlin JT, Nistri S, Cecchi F, Udelson JE, Maron BJ. Hypertrophic cardiomyopathy is predominantly a disease of left ventricular outflow tract obstruction. *Circulation*. 2006;114:2232–2239.
72. Maron BJ. Risk stratification and role of implantable defibrillators for prevention of sudden death in patients with hypertrophic cardiomyopathy. *Circ J*. 2010;74:2271–2282.
73. Maron BJ. Contemporary insights and strategies for risk stratification and prevention of sudden death in hypertrophic cardiomyopathy [published correction appears in *Circulation*. 2010;122:e7]. *Circulation*. 2010;121:445–456.
74. Lue TF. Erectile dysfunction. *N Engl J Med*. 2000;342:1802–1813.
75. Medical Research Council Working Party on Mild to Moderate Hypertension. Adverse reactions to bendrofluzide and propranolol for the treatment of mild hypertension: report of Medical Research Council Working Party on Mild to Moderate Hypertension. *Lancet*. 1981;2:539–543.
76. Grimm RH Jr, Grandits GA, Prineas RJ, McDonald RH, Lewis CE, Flack JM, Yunis C, Svendsen K, Liebson PR, Elmer PJ. Long-term effects on sexual function of five antihypertensive drugs and nutritional hygienic treatment in hypertensive men and women: Treatment of Mild Hypertension Study (TOMHS). *Hypertension*. 1997;29(part 1):8–14.
77. Bansal S. Sexual dysfunction in hypertensive men: a critical review of the literature. *Hypertension*. 1988;12:1–10.

78. Düsing R. Sexual dysfunction in male patients with hypertension: influence of antihypertensive drugs. *Drugs*. 2005;65:773–786.
79. Steinke EE, Jaarsma T. Impact of cardiovascular disease on sexuality. In: Moser DK, Riegel B, eds. *Cardiac Nursing*. St. Louis, MO: Saunders; 2008.
80. Franzen D, Metha A, Seifert N, Braun M, Höpp HW. Effects of beta-blockers on sexual performance in men with coronary heart disease: a prospective, randomized and double blinded study. *Int J Impot Res*. 2001;13:348–351.
81. Baumhäkel M, Schlimmer N, Kratz M, Hacket G, Jackson G, Böhm M. Cardiovascular risk, drugs and erectile function: a systematic analysis. *Int J Clin Pract*. 2011;65:289–298.
82. Erdmann E. Safety and tolerability of beta-blockers: prejudices & reality. *Indian Heart J*. 2010;62:132–135.
83. Jackson G, Betteridge J, Dean J, Eardley I, Hall R, Holdright D, Holmes S, Kirby M, Riley A, Sever P. A systematic approach to erectile dysfunction in the cardiovascular patient: a Consensus Statement: update 2002. *Int J Clin Pract*. 2002;56:663–671.
84. Ko DT, Hebert PR, Coffey CS, Sedrakyan A, Curtis JP, Krumholz HM. Beta-blocker therapy and symptoms of depression, fatigue, and sexual dysfunction. *JAMA*. 2002;288:351–357.
85. Silvestri A, Galetta P, Cerquetani E, Marazzi G, Patrizi R, Fini M, Rosano GM. Report of erectile dysfunction after therapy with beta-blockers is related to patient knowledge of side effects and is reversed by placebo. *Eur Heart J*. 2003;24:1928–1932.
86. Cocco G. Erectile dysfunction after therapy with metoprolol: the Hawthorne effect. *Cardiology*. 2009;112:174–177.
87. Duncan L, Bateman DN. Sexual function in women: do antihypertensive drugs have an impact? *Drug Saf*. 1993;8:225–234.
88. Boydak B, Nalbantgil S, Fici F, Nalbantgil I, Zoghi M, Ozerkan F, Tengiz I, Ercan E, Yilmaz H, Yoket U, Onder R. A randomised comparison of the effects of nebivolol and atenolol with and without chlorthalidone on the sexual function of hypertensive men [published correction appears in *Clin Drug Investig*. 2007;27:864]. *Clin Drug Investig*. 2005;25:409–416.
89. Brixius K, Middeke M, Lichtenthal A, Jahn E, Schwinger RH. Nitric oxide, erectile dysfunction and beta-blocker treatment (MR NOED study): benefit of nebivolol versus metoprolol in hypertensive men. *Clin Exp Pharmacol Physiol*. 2007;34:327–331.
90. Olsson AM, Persson CA; Swedish Sildenafil Investigators Group. Efficacy and safety of sildenafil citrate for the treatment of erectile dysfunction in men with cardiovascular disease. *Int J Clin Pract*. 2001;55:171–176.
91. Kloner RA. Cardiovascular effects of the 3 phosphodiesterase-5 inhibitors approved for the treatment of erectile dysfunction. *Circulation*. 2004;110:3149–3155.
92. Morales AM, Mirone V, Dean J, Costa P. Vardenafil for the treatment of erectile dysfunction: an overview of the clinical evidence. *Clin Interv Aging*. 2009;4:463–472.
93. Kloner RA, Brown M, Prisant LM, Collins M; Sildenafil Study Group. Effect of sildenafil in patients with erectile dysfunction taking antihypertensive therapy. *Am J Hypertens*. 2001;14:70–73.
94. Kloner RA, Mitchell M, Emmick JT. Cardiovascular effects of tadalafil in patients on common antihypertensive therapies. *Am J Cardiol*. 2003;92:47M–57M.
95. Padma-Nathan H, Eardley I, Kloner RA, Laties AM, Montorsi F. A 4-year update on the safety of sildenafil citrate (Viagra). *Urology*. 2002;60(suppl 2):67–90.
96. Thadani U, Smith W, Nash S, Bittar N, Glasser S, Narayan P, Stein RA, Larkin S, Mazzu A, Tota R, Pomerantz K, Sundaresan P. The effect of vardenafil, a potent and highly selective phosphodiesterase-5 inhibitor for the treatment of erectile dysfunction, on the cardiovascular response to exercise in patients with coronary artery disease. *J Am Coll Cardiol*. 2002;40:2006–2012.
97. Arruda-Olson AM, Mahoney DW, Nehra A, Leckel M, Pellikka PA. Cardiovascular effects of sildenafil during exercise in men with known or probable coronary artery disease: a randomized crossover trial. *JAMA*. 2002;287:719–725.
98. Cheitlin MD, Hutter AM Jr, Brindis RG, Ganz P, Kaul S, Russell RO Jr, Zusman RM; Technology and Practice Executive Committee. Use of sildenafil (Viagra) in patients with cardiovascular disease [published correction appears in *Circulation*. 1999;100:2389]. *Circulation*. 1999;99:168–177.
99. Mittleman MA, Maclure M, Glasser DB. Evaluation of acute risk for myocardial infarction in men treated with sildenafil citrate. *Am J Cardiol*. 2005;96:443–446.
100. Pickering TG, Shepherd AM, Puddey I, Glasser DB, Orazem J, Sherman N, Mancia G. Sildenafil citrate for erectile dysfunction in men receiving multiple antihypertensive agents: a randomized controlled trial. *Am J Hypertens*. 2004;17(part 1):1135–1142.
101. DeBusk RF, Pepine CJ, Glasser DB, Shpilsky A, DeRiesthal H, Sweeney M. Efficacy and safety of sildenafil citrate in men with erectile dysfunction and stable coronary artery disease [published correction appears in *Am J Cardiol*. 2004;94:543–544]. *Am J Cardiol*. 2004;93:147–153.
102. Fox KM, Thadani U, Ma PT, Nash SD, Keating Z, Czorniak MA, Gillies H, Keltai M; CAESAR I (Clinical American and European Studies of Angina and Revascularization) Investigators. Sildenafil citrate does not reduce exercise tolerance in men with erectile dysfunction and chronic stable angina. *Eur Heart J*. 2003;24:2206–2212.
103. Kloner RA, Jackson G, Hutter AM, Mittleman MA, Chan M, Warner MR, Costigan TM, Vail GM. Cardiovascular safety update of tadalafil: retrospective analysis of data from placebo-controlled and open-label clinical trials of tadalafil with as needed, three times-per-week or once-a-day dosing. *Am J Cardiol*. 2006;97:1778–1784.
104. Van AH, Zumbé J, Stauch K, Hanisch JU. The Real-Life Safety and Efficacy of vardenafil (REALISE) study: results in men from Europe and overseas with erectile dysfunction and cardiovascular or metabolic conditions. *J Sex Med*. 2010;7:3161–3169.
105. Shabsigh R, Duval S, Shah M, Regan TS, Juhász M, Veltry LG. Efficacy of vardenafil for the treatment of erectile dysfunction in men with hypertension: a meta-analysis of clinical trial data. *Curr Med Res Opin*. 2007;23:2453–2460.
106. Goldstein I, Kim E, Steers WD, Pryor JL, Wilde DW, Natanegara F, Wong DG, Ahuja S. Efficacy and safety of tadalafil in men with erectile dysfunction with a high prevalence of comorbid conditions: results from MOMENTUS: multiple observations in men with erectile dysfunction in National Tadalafil Study in the US [published correction appears in *J Sex Med*. 2007;4:522]. *J Sex Med*. 2007;4:166–175.
107. Webb DJ, Freestone S, Allen MJ, Muirhead GJ. Sildenafil citrate and blood-pressure-lowering drugs: results of drug interaction studies with an organic nitrate and a calcium antagonist. *Am J Cardiol*. 1999;83:21C–28C.
108. Kloner RA, Hutter AM, Emmick JT, Mitchell MI, Denne J, Jackson G. Time course of the interaction between tadalafil and nitrates. *J Am Coll Cardiol*. 2003;42:1855–1860.
109. Kloner R. Erectile dysfunction and hypertension. *Int J Impot Res*. 2007;19:296–302.
110. Kloner RA, Jackson G, Emmick JT, Mitchell MI, Bedding A, Warner MR, Pereira A. Interaction between the phosphodiesterase 5 inhibitor, tadalafil and 2 alpha-blockers, doxazosin and tamsulosin in healthy normotensive men. *J Urol*. 2004;172(part 1):1935–1940.
111. Chivers ML, Rosen RC. Phosphodiesterase type 5 inhibitors and female sexual response: faulty protocols or paradigms? *J Sex Med*. 2010;7(part 2):858–872.
112. Hulley S, Grady D, Bush T, Furberg C, Herrington D, Riggs B, Vittinghoff E; Heart and Estrogen/progestin Replacement Study (HERS) Research Group. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. *JAMA*. 1998;280:605–613.
113. Rossouw JE, Anderson GL, Prentice RL, LaCroix AZ, Kooperberg C, Stefanick ML, Jackson RD, Beresford SA, Howard BV, Johnson KC, Kotchen JM, Ockene J; Writing Group for the Women's Health Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *JAMA*. 2002;288:321–333.
114. Anderson GL, Limacher M, Assaf AR, Bassford T, Beresford SA, Black H, Bonds D, Brunner R, Brzyski R, Caan B, Chlebowski R, Curb D, Gass M, Hays J, Heiss G, Hendrix S, Howard BV, Hsia J, Hubbell A, Jackson R, Johnson KC, Judd H, Kotchen JM, Kuller L, LaCroix AZ, Lane D, Langer RD, Lasser N, Lewis CE, Manson J, Margolis K, Ockene J, O'Sullivan MJ, Phillips L, Prentice RL, Ritenbaugh C, Robbins J, Rossouw JE, Sarto G, Stefanick ML, Van Horn L, Wactawski-Wende J, Wallace R, Wassertheil-Smolter S; Women's Health Initiative Steering Committee. Effects of conjugated equine estrogen in postmenopausal women with hysterectomy: the Women's Health Initiative randomized controlled trial. *JAMA*. 2004;291:1701–1712.

115. Cherry N, Gilmour K, Hannaford P, Heagerty A, Khan MA, Kitchener H, McNamee R, Elstein M, Kay C, Seif M, Buckley H; ESPRIT team. Oestrogen therapy for prevention of reinfarction in postmenopausal women: a randomised placebo controlled trial. *Lancet*. 2002;360:2001–2008.
116. Ponzzone R, Biglia N, Jacomuzzi ME, Maggiorotto F, Mariani L, Sismondi P. Vaginal oestrogen therapy after breast cancer: is it safe? *Eur J Cancer*. 2005;41:2673–2681.
117. Savaliya AA, Shah RP, Prasad B, Singh S. Screening of Indian aphrodisiac ayurvedic/herbal healthcare products for adulteration with sildenafil, tadalafil and/or vardenafil using LC/PDA and extracted ion LC-MS/TOF. *J Pharm Biomed Anal*. 2010;52:406–409.
118. Oh SS, Zou P, Low MY, Koh HL. Detection of sildenafil analogues in herbal products for erectile dysfunction. *J Toxicol Environ Health A*. 2006;69:1951–1958.
119. Kearney T, Tu N, Haller C. Adverse drug events associated with yohimbine-containing products: a retrospective review of the California Poison Control System reported cases. *Ann Pharmacother*. 2010;44:1022–1029.
120. Aoki H, Nagao J, Ueda T, Strong JM, Schonlau F, Yu-Jing S, Lu Y, Horie S. Clinical assessment of a supplement of Pycnogenol and L-arginine in Japanese patients with mild to moderate erectile dysfunction. *Phytother Res*. May 27, 2011. doi:10.1002/ptr.3462. <http://onlinelibrary.wiley.com/doi/10.1002/ptr.3462/abstract>. Accessed May 27, 2011.
121. Musso NR, Vergassola C, Pende A, Lotti G. Yohimbine effects on blood pressure and plasma catecholamines in human hypertension. *Am J Hypertens*. 1995;8:565–571.
122. Schulman SP, Becker LC, Kass DA, Champion HC, Terrin ML, Forman S, Ernst KV, Kelemen MD, Townsend SN, Capriotti A, Hare JM, Gerstenblith G. L-arginine therapy in acute myocardial infarction: the Vascular Interaction With Age in Myocardial Infarction (VINTAGE MI) randomized clinical trial. *JAMA*. 2006;295:58–64.
123. Moser DK, Dracup K, Evangelista LS, Zambroski CH, Lennie TA, Chung ML, Doering LV, Westlake C, Heo S. Comparison of prevalence of symptoms of depression, anxiety, and hostility in elderly patients with heart failure, myocardial infarction, and a coronary artery bypass graft. *Heart Lung*. 2010;39:378–385.
124. Roose SP, Seidman SN. Sexual activity and cardiac risk: is depression a contributing factor? *Am J Cardiol*. 2000;86:38F–40F.
125. Mosack V, Steinke EE. Trends in sexual concerns after myocardial infarction. *J Cardiovasc Nurs*. 2009;24:162–170.
126. Lai YH, Hsieh SR, Ho WC, Chiou AF. Factors associated with sexual quality of life in patients before and after coronary artery bypass grafting surgery. *J Cardiovasc Nurs*. 2011;26:487–496.
127. Phan A, Ishak WW, Shen BJ, Fuess J, Philip K, Bresee C, Czer L, Schwarz ER. Persistent sexual dysfunction impairs quality of life after cardiac transplantation. *J Sex Med*. 2010;7:2765–2773.
128. Kazemi-Saleh D, Pishgou B, Assari S, Tavallaii SA. Fear of sexual intercourse in patients with coronary artery disease: a pilot study of associated morbidity. *J Sex Med*. 2007;4:1619–1625.
129. Nicolosi A, Moreira ED Jr, Villa M, Glasser DB. A population study of the association between sexual function, sexual satisfaction and depressive symptoms in men. *J Affect Disord*. 2004;82:235–243.
130. Steinke EE, Swan JH. Effectiveness of a videotape for sexual counseling after myocardial infarction. *Res Nurs Health*. 2004;27:269–280.
131. Klein R, Bar-on E, Klein J, Benbenishty R. The impact of sexual therapy on patients after cardiac events participating in a cardiac rehabilitation program. *Eur J Cardiovasc Prev Rehabil*. 2007;14:672–678.
132. Steinke EE, Wright DW. The role of sexual satisfaction, age, and cardiac risk factors in the reduction of post-MI anxiety. *Eur J Cardiovasc Nurs*. 2006;5:190–196.
133. Froelicher ES, Kee LL, Newton KM, Lindskog B, Livingston M. Return to work, sexual activity, and other activities after acute myocardial infarction. *Heart Lung*. 1994;23:423–435.
134. Dhabuwala CB, Kumar A, Pierce JM. Myocardial infarction and its influence on male sexual function. *Arch Sex Behav*. 1986;15:499–504.
135. Jaarsma T, Strömberg A, Fridlund B, De Geest S, Mårtensson J, Moons P, Norekval TM, Smith K, Steinke E, Thompson DR; UNITE Research Group. Sexual counselling of cardiac patients: nurses' perception of practice, responsibility and confidence. *Eur J Cardiovasc Nurs*. 2010;9:24–29.
136. Steinke EE, Patterson-Midgley P. Sexual counseling of MI patients: nurses' comfort, responsibility, and practice. *Dimens Crit Care Nurs*. 1996;15:216–223.
137. Vassiliadou A, Stamatopoulou E, Triantafyllou G, Gerodimou E, Toulia G, Pistolas D. The role of nurses in the sexual counseling of patients after myocardial infarction. *Health Sci J*. 2008;2:111–118.
138. Steinke EE, Mosack V, Barnason S, Wright DW. Progress in sexual counseling by cardiac nurses, 1994 to 2009. *Heart Lung*. 2011;40:e15–e24.
139. Bedell SE, Duperval M, Goldberg R. Cardiologists' discussions about sexuality with patients with chronic coronary artery disease. *Am Heart J*. 2002;144:239–242.
140. Steinke E, Patterson-Midgley P. Sexual counseling following acute myocardial infarction. *Clin Nurs Res*. 1996;5:462–472.
141. Steinke EE, Patterson-Midgley P. Importance and timing of sexual counseling after myocardial infarction. *J Cardiopulm Rehabil*. 1998;18:401–407.
142. Akdolun N, Terakye G. Sexual problems before and after myocardial infarction: patients' needs for information. *Rehabil Nurs*. 2001;26:152–158.
143. Arenhall E, Kristofferzon ML, Fridlund B, Nilsson U. The female partners' experiences of intimate relationship after a first myocardial infarction. *J Clin Nurs*. 2011;20:1677–1684.
144. Arenhall E, Kristofferzon ML, Fridlund B, Malm D, Nilsson U. The male partners' experiences of the intimate relationships after a first myocardial infarction. *Eur J Cardiovasc Nurs*. 2011;10:108–114.
145. Ivarsson B, Fridlund B, Sjöberg T. Information from health care professionals about sexual function and coexistence after myocardial infarction: a Swedish national study. *Heart Lung*. 2009;38:330–335.
146. Hardin SR. Cardiac disease and sexuality: implications for research and practice. *Nurs Clin North Am*. 2007;42:593–603.
147. Jaarsma T, Steinke EE, Gianotten WL. Sexual problems in cardiac patients: how to assess, when to refer. *J Cardiovasc Nurs*. 2010;25:159–164.
148. Steinke EE, Mosack V, Wright DW, Chung ML, Moser DK. Risk factors as predictors of sexual activity in heart failure. *Dimens Crit Care Nurs*. 2009;28:123–129.

KEY WORDS: AHA Scientific Statements ■ cardiovascular disease