Sexual Activity and Chronic Heart Failure

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Little has been published about sexual function in chronic heart failure (CHF) and knowledge among clinicians in this regard is sparse. To review data regarding sexual function and dysfunction in patients with CHF, 2 of the authors (S.A.M. and P.A.U.) independently conducted a literature search using the MEDLINE database. English-language articles and cited bibliographies published between January 1996 and November 2006 were reviewed. Search terms included *heart failure* or *CHF* or *ventricular dysfunction* or *heart disease* in conjunction with *sexual activity*, *erectile dysfunction*, *impotence*, or *sex*. Articles were selected for inclusion if they had a primary focus on CHF and sexual function or dysfunction. Critical reviews of the literature, observational studies using self-reported patient surveys, and prospective, blinded, randomized, placebo-controlled trials were included. Articles were not excluded on the basis of patient sample size but were excluded if the article concerned a broad aspect of cardiovascular disease rather than CHF. When properly screened and treated, most patients with CHF can safely engage in sexual activity and be treated for erectile dysfunction with sildenafil, provided that they do not have active ischemia and do not require treatment with nitrates. Clinicians should know the physiological requirements of sexual activity and the impact CHF has on sexual performance. Fear of a cardiac event during intercourse can interfere with patients' ability to perform and enjoy sex, and so it is important that the physician be able to counsel patients with CHF about sexual activity.

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BP = blood pressure; CHF = chronic heart failure; ED = erectile dysfunction; EF = ejection fraction; HR = heart rate; MET = metabolic equivalent task; NYHA = New York Heart Association; VAD = ventricular assist device

The physiological effects and clinical aspects of sexual function have been extensively studied and reported in patients who have angina or who have experienced a myocardial infarction[1-5] and in those who have undergone coronary artery bypass graft surgery[6] or heart transplant.[7] However, less has been published about sexual function in patients with chronic heart failure (CHF), and clinicians are therefore less well informed on this topic.

The purpose of this review is to provide clinicians with a succinct knowledge base regarding sexual activity in CHF. We first review the hemodynamics of sexual activity in normal physiological states and in CHF. Second, we examine the relationship between erectile dysfunction (ED) and CHF and explore the use of phosphodiesterase inhibitors and alternative therapies to treat ED in these patients. Third, we develop an approach that the clinician can use in counseling patients with CHF regarding sexual activity. Finally, the article touches briefly on gaps in the literature and areas for future study.

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METHODS

Two authors (S.A.M. and P.A.U.) independently conducted a literature search by using the MEDLINE database. English-language articles and cited bibliographies published between January 1996 and November 2006 were reviewed. Search terms included *heart failure* or *CHF* or *ventricular dysfunction* or *heart disease* in conjunction with *sexual activity*, *erectile dysfunction*, *impotence*, or *sex*. Articles were selected for inclusion if they had a primary focus on CHF and sexual function or dysfunction. Critical reviews of the literature were included, as were observational studies using self-reported patient surveys, and prospective, blinded, randomized, placebo-controlled trials. Articles were not excluded on the basis of patient sample size but were excluded if the article focused on a broad aspect of cardiovascular disease rather than on CHF.

METABOLIC AND HEMODYNAMIC DEMANDS OF SEXUAL ACTIVITY

Sexual activity consists of 4 stages: baseline resting, foreplay, stimulation, and orgasm. In young healthy couples, heart rate (HR) and blood pressure (BP) increase modestly during each stage; peak coital HRs occur during orgasm and range between 140 and 180 beats/min, with a mean increase in systolic BP of 80 mm Hg and in diastolic BP of 50 mm Hg.[8-10] Respiratory rates and tidal volumes also markedly increase.

In a study that investigated the hemodynamic parameters during sexual activity of middle-aged men with or without coronary artery disease at home using 24-hour ambulatory electrocardiographic monitors, the mean HR at the time of orgasm was 117.4 beats/min, with a mean BP of 162/89 mm Hg.[2]

Interestingly, the peak coital HRs were submaximal, lower than those found during the patients' normal daily

activities (mean, 120 beats/min). Minute oxygen consumption has also been measured during sexual activity and is expressed as metabolic equivalent tasks (METs) (1 MET is defined as the energy expended at rest, which is equivalent to body oxygen consumption of 3.5 mL/kg/min).[8] In a study by Bohlen et al,[8] self- and partner stimulation required 1.7 and 1.8 METs, respectively, whereas coitus required 2.5 METs with the "woman-on-top" and 3.3 METs with the "man-on-top." During coitus, peak HRs and maximal oxygen consumption were maintained for approximately 15 seconds.

To put these measurements into perspective, 2.0 to 3.0 METs represent the energy expended walking on level ground at 2 to 2.5 mph or doing light housework (dusting) and 3.0 to 4.0 METs that expended while walking on level ground at 3 to 4 mph, climbing stairs slowly, or doing general housework (vacuuming).[7] By comparison, sexual activity requires 2.0 to 3.0 METs in the preorgasmic stage and 3.0 to 4.0 METs during orgasm.[8]

However, contrasting hemodynamic results were reported in a middle-aged man with severe CHF due to ischemic cardiomyopathy and pulmonary hypertension.[11] As measured by an implanted ambulatory hemodynamic monitor, his HR increased during physical exercise (brisk walking) to parallel his level of activity (HR increased from 70 to 105 beats/min; right ventricular pressures, from 50 to 74 mm Hg systolic and from 10 to 13 mm Hg diastolic; and pulmonary artery diastolic pressure, from 28 to 35 mm Hg). During sexual activity, the patient's HR increased only minimally during the preorgasmic phase but increased rapidly at orgasm, peaking at 131 beats/min (vs 53 beats/min before foreplay). A marked increase in right ventricular pressure, from 38/8 to 101/21 mmHg, and a corresponding doubling in diastolic pulmonary pressures were also observed. This difference can likely be explained by activation of the sympathetic nervous system and the influence of psychological or emotional factors (arousal, fear, anxiety) in the patient with CHF during sexual activity.

SEXUAL FUNCTION AND CHF

The American Heart Association has estimated that more than 4.9 million people in the United States have CHF.[12] As CHF progresses, patients experience an increase in fatigue, shortness of breath, palpitations, or angina, decreasing their quality of life and potentially interfering with their ability to perform sexually.

Jaarsma et al[13] studied 62 patients (men, 82%; women, 18%) with New York Heart Association (NYHA) functional class III and IV CHF to determine the effect of low ejection fraction (EF) on sexual interest, sexual function, and marital relationships. A 6-minute walk test was used to determine the exercise tolerance of the patients and echocardiography to measure their EF. Sexual function was assessed using the self-reported sexual adjustment scale. Most patients (73%) reported a marked or complete loss in sexual interest. Sexual activity markedly decreased or ceased in 76%. More than half of the patients reported a reduction in the level of satisfaction they experienced during sex, and 58% reported frequent or complete inability to perform sexually because of CHF.

Interestingly, whereas a meaningful association was found between patients' scores on the sexual adjustment scale and their symptom severity as ascertained from their history and their exercise tolerance on the 6-minute walk test, no such association between EF and sexual function was observed. This finding is consistent with prior studies, which showed that exercise capacity in CHF is related not to resting EF but rather to increased HR and stroke volume in the face of abnormal preload response, autonomic dysregulation, and increased vascular resistance.[14,15]

Subsequently, Westlake et al[16] reported similar results in a study of 63 patients with CHF (men, 69.4%) and their spouses. In this study, most patients (62%) reported that their frequency of sex had slightly or markedly decreased, 30% that they had stopped having sex, 22% that they had no interest in sex, and 59% that they had had slight or constant problems performing.

Little has been published to guide clinicians in counseling their patients with CHF about sexual function. In a review article about intimacy needs and chronic illness, Steinke[17] suggests advising patients with CHF to use a "semi-reclining or on-bottom" position during coitus, which decreases the level of physical exertion, and to stop and rest if dyspnea occurs. She recommends encouraging foreplay because it allows the patient and the partner to determine the patient's exercise tolerance and to express affection if exercise capacity is so diminished that more strenuous activities are not physically possible.

ERECTILE DYSFUNCTION AND CHF

Erectile dysfunction affects 60% to 70% of CHF outpatients.[18] Patients with CHF often use tobacco, are obese, and have hypertension, diabetes mellitus, coronary artery disease, or hyperlipidemia; other predisposing factors for ED include polypharmacy and depression.[19] Erectile dysfunction, cardiovascular disease, and depression seem to form a mutually reinforcing triad[20]; ED is caused by and can itself cause depression, as does CHF when patients become symptomatic and have reduced quality of life.

Rastogi et al[21] explored why patients with CHF develop ED, concluding that multiple factors may be involved. In

addition to the decreased exercise capacity mentioned previously, patients with CHF have arterial compliance abnormalities and often atherosclerosis, which reduces blood flow into the penile corpora cavernosa. Furthermore, endothelial dysfunction either decreases the production or increases the breakdown of nitric oxide, a powerful vasodilator that promotes penile engorgement by relaxing the penile chambers.[22] Elevated levels of circulating endothelin and other potent vasoconstrictors are also observed in patients with CHF and interfere with their ability to achieve and maintain an erection. Erectile dysfunction can be caused or worsened by many of the medications that are commonly prescribed for the treatment of CHF, including digoxin, β-blockers, diuretics, and spironolactone.[21] Interestingly, angiotensin-converting enzyme inhibitors and angiotensin receptor blockers appear to favorably affect sexual function. Currently, insufficient data exist to evaluate the effect of biventricular pacemakers and cardiac resynchronization therapy on sexual function.

The treatment of ED in patients with CHF begins with optimization of CHF management.[21] Sexual function improves as CHF symptoms are reduced and exercise capacity increases. Rastogi et al[21] recommend avoiding when possible drugs such as digoxin and thiazide diuretics which can contribute to ED. They also recommend replacing the peripherally acting propranolol with metoprolol or carvedilol (which has the added benefit of α-blockade) and replacing spironolactone with the more selective mineralocorticoid receptor antagonist eplerenone. These recommendations have not been studied in randomized trials, and few patients with CHF are currently treated with propranolol. The mainstay of pharmacological therapy for ED is a class of drugs known as phosphodiesterase-5 inhibitors, which includes sildenafil, vardenafil, and tadalafil. The drugs work by inhibiting the breakdown of cyclic guanosine monophosphate, a second messenger of prostacyclin and nitric oxide, resulting in relaxation of the smooth muscle of the corpora cavernosa, increased blood flow to the penis (vasodilation), and better erection. In a recent review on ED in CHF, Schwarz et al[23] comprehensively discussed the underlying mechanisms of these drugs. All 3 drugs appear to be equally effective in the treatment of ED; however, the newer drugs, vardanafil and tadalafil, have not been as well studied and therefore fewer data are available regarding their safety in patients with CHF.[20]

However, Ghofrani et al[24] compared the hemodynamic profiles of the 3 drugs in patients with pulmonary arterial hypertension, showing that the drugs differ in time to peak hemodynamic effect, pulmonary selectivity, and effect on arterial oxygenation. In the most recent consensus statement of the American College of Cardiology and American Heart Association, CHF is a relative contraindication for the use of sildenafil.[25] Concern has been raised that patients with CHF who take both vasodilators and sildenafil are at risk for profound hypotension.

Webster et al[19] studied sildenafil use in 35 patients with NYHA class II or III CHF in a placebo-controlled randomized and blinded crossover trial. Patients were included if they did not use nitrates, had a history of chronic ED, and had negative stress test results. They were randomized to either 6 weeks of placebo with a switch to 6 weeks of 50 mg of sildenafil, or the reverse. The patients, who were asked to keep a diary on the use of the drug, were followed up at 2-week intervals to assess for adverse effects of the drug and symptoms of CHF exacerbation. The patients also completed questionnaires that were designed to elicit information regarding erectile function, symptoms of depression, or perceptions of CHF symptoms. No significant change in HR or BP was observed with sildenafil use, and erectile function significantly improved when patients were taking sildenafil vs placebo. When taking sildenafil, patients also experienced a decrease in depressive symptoms and an increase in quality of life. No major hemodynamic events occurred during the study. All patients were successfully treated with diuretics as outpatients, and none reported any of the adverse effects that are common with sildenafil use.

In a fixed-dose, double-blind, randomized, placebo-controlled, 2-way crossover study, Bocchi et al[18] evaluated the efficacy and safety of sildenafil in 23 male patients with CHF, as well as its effects on exercise and neurohormonal activation. To assess safety, patients were randomized to receive either 50 mg of sildenafil or placebo before undergoing a 6-minute walk test and then a maximal exercise test. The next day, the patients crossed over to the second treatment and repeated the protocol. Those who tolerated the drug were advanced to phase 2, an open-label, home-based, prospective study in which patients took a flexible dose of 25 mg to 150 mg of sildenafil (dose was determined on the basis of efficacy and adverse effects) 1 to 2 hours before sexual activity and then recorded the drug's efficacy using the 15-question International Index of Erectile Function. In the safety phase of the study, sildenafil reduced HR at rest, during the 6-minute walk test, and until the 8th minute of the maximal exercise test. In addition, mean ± SD resting systolic BP decreased from 116±18 to 108±18 (P=.004); diastolic BP, from 69±9 to 63±11 (P<.05). During the exercise phase, sildenafil reduced the mismatch between pulmonary ventilation and perfusion while also increasing peak oxygen consumption and exercise time. No difference was found in plasma norepinephrine levels between the sildenafil and placebo groups. This study showed that sildenafil was well tolerated and that it led to an improvement in most of the patient's scores on the Interna-

tional Index of Erectile Function. The effective mean ± SD dose for erection was 58±30 mg, with efficacy rates similar to those in prior studies (between 62% and 83%).[26]

More recently, Katz et al[27] performed a multicenter, prospective, randomized, double-blind, placebo-controlled, flexible-dose study to investigate the efficacy and tolerability of sildenafil in patients with stable CHF. Patients were included if they had a known EF of 40% or less, stable symptoms for 4 weeks or more, no signs of congestion on examination, stable oral drug regimen without nitrate use for 4 weeks or more, and no CHF-related emergency department or inpatient hospital visits for 3 months or more. Most patients in the study had mild CHF, although approximately 10% in both groups had class III CHF. The patients, who ranged in age from 37 years to 83 years, included those with comorbid conditions (diabetes mellitus, hyperlipidemia, hypertension, ischemic heart disease, previous coronary bypass surgery). Patients were excluded if they used nitrates, were at high cardiovascular risk, or had hypotension, uncontrolled hypertension, hypertrophic or restrictive cardiomyopathy, primary uncorrected severe valvular heart disease, a history of myocarditis, or recent implantable defibrillator firing. Significant improvements in erectile function, sexual desire, and both intercourse and overall satisfaction were seen in the sildenafil vs placebo group. More than 90% of the adverse effects of the drug were transient and of mild to moderate severity; they included headache, facial flushing, and chromatopsia. Few cardiovascular adverse events occurred in either group.

Some cases of sudden cardiac death have been reported in men with coronary artery disease who used sildenafil.[28] In vitro, sildenafil has been noted to block the rapid component of the delayed rectifier potassium current in a dose-dependent fashion. It therefore has the potential to act as a class III antiarrhythmic drug at high plasma concentrations. Piccirillo et al[29] investigated the effects of sildenafil on cardiac repolarization in men with NYHA class II CHF, with the hypothesis that patients with an altered phase would be at higher risk for drug-induced QT prolongation, which leads to malignant arrhythmia and sudden cardiac death.

In this study, 10 men with ischemic dilated cardiomyopathy and 10 healthy controls received a single dose of 50 mg of sildenafil at rest. Measurements were then taken of the QT intervals and dispersion as well as of dynamic variables such as the slope of the line drawn when QT is plotted against HR and the index of QT variability. Spectral analysis of HR and BP variability was also performed to measure changes in autonomic cardiovascular control after sildenafil administration. With respect to the measurements of the QT intervals, the baseline mean QT was significantly longer in the patients with CHF than in controls, reflecting CHF-related structural changes in the heart such as hypertrophy and dilatation; however, overlap was observed for variance in QT in patients with CHF and controls. In patients with CHF who took sildenafil, the QT interval and QT dispersion remained unchanged, but both the QT variance and the QT-RR slope increased.

Sildenafil significantly lowered mean ± SD systolic BP in both groups (107±3 to 100±2 mm Hg, with sildenafil; 112±2 to 103±2 mm Hg, for controls; $P<.05$) and slightly reduced diastolic BP in the controls but not in the patients with CHF. Mean ± SD HR also increased in both groups with sildenafil (by 13%±3% in the CHF group and 8%±3% in the control group [$P<.05$]). The mild systemic vasodilatory effects were well tolerated by both groups; none of the men had adverse reactions to sildenafil. The increase in HR may be attributable to a reflex decrease in sinus vagal activity and/or an increase in sympathetic modulation. These autonomic changes, not blockage of the potassium channel, were also thought to be responsible for the changes in QT variance and QT-RR slope. The changes in the dynamic QT variables correlate with CHF severity and could favor the induction of malignant arrhythmias; however, at the 50-mg dose taken for ED, no direct effect on cardiac repolarization (QT interval) was observed.

The studies that evaluated sexual dysfunction and treatment of ED in patients with CHF are listed in Table 1. Taken together, these studies show that ED in patients with mild to moderate CHF can be safely and effectively treated with sildenafil, provided that patients are appropriately screened before initiation of therapy. In these patients, sildenafil treatment seems to improve quality of life, decrease symptoms of depression, and improve compliance with CHF regimens.

This review would not be complete without a word on second-line therapies for ED. Although they have not been well studied, androgen replacement therapy, intraurethral suppositories, penile injection therapy, penile prostheses, and vacuum-assisted erection devices do not appear to have adverse effects in patients with CHF.[21] For those who are unable or unwilling to try these treatment options, penile angioplasty or surgical revascularization is an option, provided that arterial lesions are localized.

Exercise training may be an appropriate adjunctive or substitute therapy for patients with CHF and ED who cannot take phosphodiesterase-5 inhibitors. In a prospective study of 59 patients with stable CHF (NYHA class II or III), Belardinelli et al[30] randomized 30 patients to 1 hour of moderate cycle ergometer exercise training 3 times a week for 8 weeks and 29 patients to no exercise. All patients underwent cardiopulmonary exercise testing and brachial

TABLE 1. **Studies Evaluating Sexual Dysfunction and Treatment of ED in Patients With CHF***

Reference	Study population	Design	Results
Jaarsma et al[13]	50 men and 12 women, class III and IV CHF	Self-reported SAS	73% reported loss of interest in sexual activity; 76%, decreased frequency or cessation of sexual activity; >50%, decreased satisfaction; 58%, inability to perform. No relation found between EF and sexual function
Westlake et al[16]	43 men and 20 women with EF <40% and CHF for ≥6 mo, and their spouses	Self-reported PAIS†	62% reported decreased frequency; 30%, complete cessation; 22%, lack of interest; 59%, problems performing
Webster et al[19]	35 men with class II or III CHF	Prospective, placebo-controlled, crossover trial to assess safety and efficacy of 50 mg of sildenafil	Sildenafil caused a mean ± SEM asymptomatic decrease in blood pressure of 6±3 mm Hg; erectile function and depression scores improved with sildenafil
Bocchi et al[18]	22 men with class II or III CHF and 1 man with class IV CHF	Prospective, double-blind, placebo-controlled, randomized study to evaluate safety and efficacy of sildenafil	Sildenafil reduced resting heart rate and blood pressure. During exercise, sildenafil reduced pulmonary ventilation/perfusion mismatch, increased oxygen consumption, and exercise time. Sildenafil improved most scores on the IIEF
Katz et al[27]	137 men with class I, II, or III CHF	Multicenter, prospective, randomized, double-blind, placebo-controlled, flexible-dose study	Sildenafil significantly improved scores on the IIEF; >90% of adverse events were transient and of mild/moderate severity (headache, facial flushing, chromotopsia)
Piccirillo et al[29]	10 men with class II CHF and 10 controls	Prospective study of effects of 50 mg of sildenafil on QT interval in CHF patients and healthy controls	With sildenafil, QT interval and QT dispersion remained unchanged but both QT variance and QT-RR slope increased in patients with CHF. In both groups, sildenafil caused mild reduction in blood pressure and increase in heart rate that were well tolerated
Belardinelli el al[30]	59 men with class II and III CHF (39 who exercised and 20 controls)	Prospective randomized study of effects of moderate exercise training on sexual function, quality of life, functional capacity, and brachial artery vasomotor response	Exercise training improved sexual function, quality of life, functional capacity, and endothelium-dependent vasomotor relaxation independently of medications and coronary risk profile improvement

*CHF= chronic heart failure; ED = erectile dysfunction; EF = ejection fraction; IIEF = International Index of Erectile Function; PAIS = Psychosocial Adjustment to Illness Scale; SAS = sexual adjustment scale; SEM = standard error of the mean.
†The PAIS includes a sexual relations scale.

artery vasomotor testing and completed quality-of-life and sexual activity questionnaires at study entry and again at 8 weeks. In the group that exercised, sexual activity as well as functional capacity and quality of life improved independently of medications. Improvement in brachial artery endothelium-dependent vasorelaxation was the strongest predictor of improvement in sexual function. Although coronary risk factors such as hyperlipidemia and hypertension did improve with exercise training, no collinearity between the change in coronary risk profile and improvement in sexual function was noted. The incidence of hypotension or adverse events and improvement in sexual function after exercise training did not differ between exercising patients who took nitrates vs those who did not. Although the sample size in this study was small and may have overestimated the improvement in sexual function, that improvement was confirmed by the responses of the patients' partners, who also completed the questionnaire and were blinded to their partners' responses. A larger trial is needed to confirm the results of this study.

AN APPROACH TO TREATING THE SEXUALLY ACTIVE PATIENT WITH CHF

In counseling patients with CHF about sexual activity, clinicians must be able to identify those who are at risk of having a cardiac event. In June 2004, the Second Princeton Consensus Conference convened to review current evidence and develop recommendations for the management of sexual dysfunction.[28] A classification system was developed that stratifies patients into low, high, and intermediate or indeterminate cardiac risk; recommendations were made for each category.

The low-risk patient (with NYHA class I CHF) is not at increased risk for sexually induced symptoms or cardiac events, especially when on an appropriate CHF regimen. These patients are usually good candidates for treatment of ED. For high-risk patients (with NYHA class III or IV CHF), who have severe or unstable cardiac disease, sexual activity may be associated with substantial risk. These patients should abstain from sexual activity until their car-

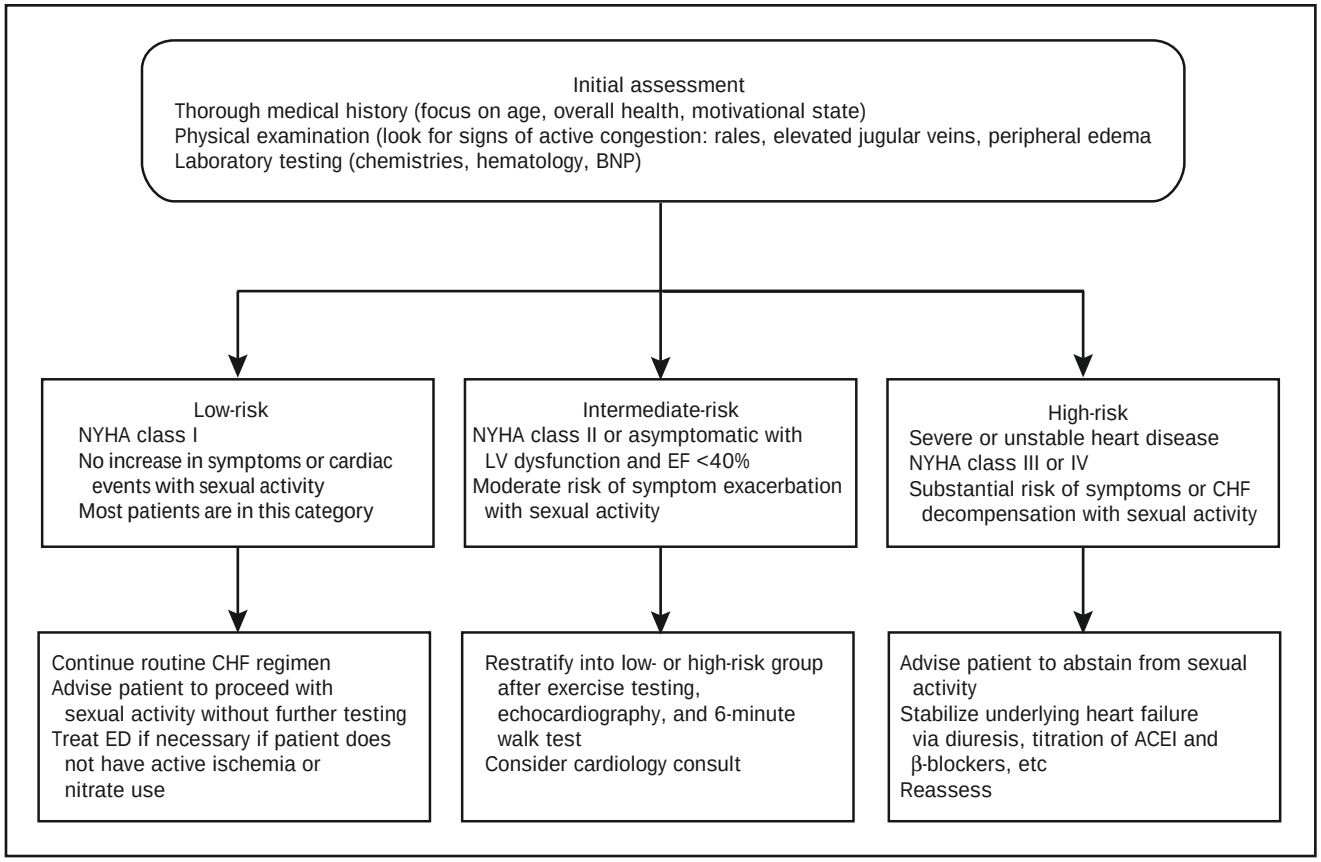

FIGURE 1. Approach to the treatment of the patient with CHF who wishes to engage in sexual activity. ACEI = angiotensin-converting enzyme inhibitor; BNP = brain natriuretic peptide; CHF = chronic heart failure; ED = erectile dysfunction; EF = ejection fraction; LV = left ventricular; NYHA = New York Heart Association.

diac condition has been assessed and treated because sexual activity could trigger decompensation. Intermediate- or indeterminate-risk patients (with NYHA class II CHF or asymptomatic left ventricular dysfunction with EF <40%), who may be at moderate risk for symptom exacerbation by sexual activity, require further cardiac testing with exercise stress testing, echocardiography, or the 6-minute walk test. Such testing allows the practitioner to reassign these patients to the low- or high-risk group and to determine their relative safety during sexual activity. A cardiology consultation may be helpful.

Initially, a thorough medical history should be elicited and a careful examination performed to assess the patient's sexual function and symptoms as well as to look for signs of active congestion. Relevant laboratory testing (hematology, chemistries) may also be helpful in stratifying patients into the low-, high-, and intermediate-risk groups. Most patients will be in the low-risk category and can continue with sexual activity and be treated for sexual dysfunction without further testing. Those in the high-risk category should be treated to stabilize their cardiac disease and then reassessed. The intermediate group requires further testing and restratification. This algorithm is applicable to male and female patients of all ages (Figure 1). Patients' overall health, age, and level of motivation should be considered when assessing whether they can safely engage in sexual activity. Table 2 provides a series of questions to assist the clinician in counseling patients with CHF who are interested in engaging in sexual activity or in being treated for ED.

TABLE 2. **Questions to Guide Physicians in Counseling Patients With CHF Who Are Interested in Sex or Treatment of ED***

1. Does the patient or the patient's partner have sexual concerns?
2. Does the patient have medical comorbidities (diabetes mellitus, hypertension, hyperlipidemia, atherosclerotic disease) that may be contributing to sexual dysfunction?
3. Do complicating factors such as ongoing ischemia or active nitrate use exist?
4. Is this patient a candidate for treatment of ED?
5. Should this patient be referred to a specialist?

* CHF = chronic heart failure; ED = erectile dysfunction.

AREAS FOR FUTURE STUDY

Because of advances in technology, patients who require mechanical support with a left ventricular assist device (VAD) are able to leave the hospital and be treated as outpatients. Activity levels, endurance, and overall energy levels have been shown to be increased in patients with CHF who have been implanted with a VAD vs those who have not. Samuels et al[31] performed a pilot study to assess the psychosocial and sexual concerns of patients discharged to home with VADs. They found that 7 of the 8 patients surveyed were interested in sexual activity (n=2) or already sexually active (n=5). None of the patients experienced any problems during sexual activity; however, they did express concerns about the device, especially the driveline. Overall, the patients seemed to have a mixed experience with sexual activity, with half reporting a change for the better. Subsequently, 3 patients were interested in sexual stimulants, including sildenafil and alprostadil, and 3 used them without adverse event. The number of patients with VADs is small but growing, with patients having VADs implanted both as bridge-to-transplant and as destination therapy. The Samuels et al study[31] suggested that sexual activity and treatment of sexual dysfunction are safe in these patients; however, the study was small, and further investigation, including research into the use of medical therapy for ED, is warranted.

Another underrepresented population regarding sexual function and dysfunction is women. Most existing studies focus entirely or primarily on male patients; the percentage of female patients with CHF who experience sexual difficulties remains unknown. However, on the basis of postmyocardial infarction data,[32] women with CHF may be equally interested in receiving counseling about sexual activity and treatment for sexual dysfunction.

More data are needed to determine the safety of the newer phosphodiesterase-5 inhibitors and the long-term safety of this class of agents in patients with CHF.[33] The cardiovascular effects of vardenafil and tadalafil, which are longer acting than sildenafil, and of other pharmacological agents used to treat ED, such as yohimbine, apomorphine, alprostadil, and testosterone, have not been well studied in patients with CHF.

Is it safe to temporarily discontinue nitrate therapy in patients who require long-acting nitrates if they wish to be treated for ED? In what levels of sexual activity can patients with moderate to severe CHF safely engage? What are the incidences of implantable cardioverter-defibrillator firing and sudden cardiac death during intercourse in patients with CHF? How safe are the therapeutic alternatives to phosphodiesterase inhibitors in patients who have taken nitrates long-term? What are the effects of long-term sildenafil use on sexual function in patients with pulmonary hypertension, and how does right-sided CHF affect sexual function? Randomized controlled trials are needed to answer these questions.

CONCLUSION

When properly screened and treated, most patients with CHF can safely engage in sexual activity. These patients can and should be treated for ED with sildenafil, but care must be taken to ensure that the patients do not have active ischemia and do not require treatment with nitrates. Patients want to receive information about the physiological requirements of sexual activity and the impact CHF has on their ability to perform sexually, but they may not know how to ask. Fear of a cardiac event during intercourse can interfere with patients' ability to perform and enjoy sex, and so it is important that physicians be able to counsel patients with CHF about sexual activity.

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