Reviews

The Cardiovascular Response to Sexual Activity: Do We Know Enough?

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Summary: Interest in comprehensive cardiac rehabilitation over the past 25 years spawned a series of small investigations concerning the heart rate, blood pressure, and ischemic response to sexual intercourse. This information was adequate for advising patients about return to sexual activity after a myocardial infarction or cardiac surgery. However, the introduction of medications for erectile dysfunction enabled impotent cardiac patients to engage in sexual activity and has highlighted the need for more detailed information concerning cardiovascular physiology during coitus. Review of the medical literature indicates a remarkable paucity of such data despite dramatic advances in most other aspects of cardiovascular physiology and pathophysiology. This brief paper gives an overview of the current knowledge of the cardiovascular response to sexual activity and, within the framework of advances in cardiology, highlights areas where it appears important to fill in the knowledge gap.

Key words: sildenafil, hemodynamics, sexual activity

Introduction

The last quarter of the century has seen remarkable changes in all fields of medicine as far apart as cardiology, endocrinology, and urology. Among the many new aspects in the field of cardiology is an appreciation of the interaction of the autonomic nervous system with the heart, and the realization that blunted cardiac autonomic function may, in diseased hearts, be a marker of electrical instability. Advances in reproductive

physiology have vastly improved the treatment of infertility, and concomitant with this has come a greater understanding of male and female human sexuality. The most recent addition to the sexual revolution has been the introduction of oral medication that is highly effective for the treatment of male erectile dysfunction.¹

One area of investigation which appears to have lagged behind almost every other aspect of urology and cardiology, yet which bridges the two disciplines, is an understanding of the cardiovascular physiologic response to sexual activity. Because of the personal and sensitive nature of human sexual activity, the small studies of cardiovascular function that have been performed have been limited to such measurements as heart rate, blood pressure, and, occasionally, to electrocardiographic abnormalities documented by Holter monitoring.^{2–19} Even those studies published in the 1990s limit themselves to measurements and techniques available a quarter of a century ago.^{4, 7, 8}

Until recently, there was little reason, other than an academic one, to delve into more detail. However, with the introduction of sildenafil as the first oral medication for the treatment of erectile dysfunction, the importance of knowledge in this area has become apparent. Although generally safe and well tolerated, sildenafil may cause profound and dangerous hypotension when used in conjunction with nitrates—agents that are commonly prescribed for patients with coronary artery disease. As a result of concerns about sildenafil use in patients with cardiac disease, the American College of Cardiology and the American Heart Association recently produced an expert consensus document that reviews the use of sildenafil in light of current knowledge of sexual activity.²⁰ A striking aspect of this carefully produced and thoughtful document is the paucity of detailed studies that have investigated the cardiovascular response to sexual activity. The following overview briefly reviews the current knowledge about this response and highlights gaps in knowledge that would benefit from intensive investigation.

Sexual Activity and Cardiovascular Response—What Do We Know?

Table I summarizes the main published studies of cardiovascular parameters recorded during sexual activity. These studies can be divided into two groups; those performed in normal sub-

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TABLE I Summary of main studies of the cardiovascular response to sexual activity

First author (Ref.)	Subjects (age)	HR at orgasm (range)	Systolic BP at orgasm (range)	ECG/Holter	Comments
Normal subjects					
Nemec 1976 (5)	10 male (40–61)	114 ± 14	163 ± 11	Occasional ectopy	No difference between man or woman on bottom
Larson 1980 (21)	9 male (40–61)	123 (88–155)	146 (130–168)	No arrhythmia	Mean HR and BP identical at orgasm as at top of 22 stairs
Bohlen 1984 (3)	10 male (25–43) 2 intercourse positions 2 non-intercourse stimulation	127 ± 23 (intercourse) 102 ± 14 (self/wife stimulation)	N/A	Not done	MVO ₂ measured. Double product maximum with man on top
Subjects with cardiac disease					
Johnston 1979 (13)	Men post-MI (n = 9) or post-CABG (n = 15) (37–66)	110 (100–150)	N/A	1 subject had increased ventricular ectopy	1 patient had peak heart rate of 92 with wife and 150 with girlfriend
Larson 1980 (21)	8 men "CAD" (39–66)	115	144 (122–170)		Stair climbing (22 steps) associated with higher BP (mean 164; range 136–180) but similar HR to peak sexual values
Jackson 1980 (11)	35 with CAD (5 women) (36–70)	122 ± 7	N/A	SVT in one patient	Angina with intercourse in 65% patients Abolished by beta blockade which was associated with reduction in peak HR to 82 ± 6 bpm
Stein 1977 (18)	16 men post-MI (40–54)	127 (120–130) before exercise training 120 (115–122) after training	N/A	No	
Hellerstein 1970 (10)	14 men mean age 48 post MI	118 (90–140)	N/A	4/14 with ST depression 2 with PVCs	ETT performed to same HR as peak sexual rate. Concluded that intercourse requires only "modest oxygen costs"
Drory 1995 (7) 1996 (8)	88 men (36–66) Most post MI No cardiac medications	118 (80–185)	N/A	Complex PVCs in 13%. 31% with ischemia (symptomatic in 7%)	Arrhythmia not predicted by ETT but ischemia during intercourse only occurred when also present on ETT
Mann 1980 (26)	3 women (mean age 35) 8 men Untreated hypertension	124 (men) 139 (women)	144/76 (women) 155/87 (men) precoital to 216/ 127 (women) and 237/138 (men)		BP recorded during a study of intra-arterial ambulatory blood pressure measurement

Abbreviations: BP = blood pressure, CABG = coronary artery bypass grafting, ETT = exercise tolerance testing, HR = heart rate, MI = myocardial infarction, N/A = not available, PVC = premature ventricular contraction, SVT = supraventricular tachycardia.

jects and those in patients with coronary artery disease. It can be seen that the peak heart rate achieved during sexual intercourse is approximately 125 beats/min with a peak systolic blood pressure of 150–160 mmHg. Based on these data it has generally been estimated that the energy expended during sexual activity is equivalent to walking up one or two flights of stairs.^{10, 21}

It is generally recommended that a patient may return to normal sexual activity almost immediately following hospital discharge for myocardial infarction provided that he has no inducible ischemia on predischARGE exercise treadmill testing. Unfortunately, the simplicity of such advice is not matched by the potential complexity surrounding sexual activity in the cardiac patient. Thirty years ago, physicians rarely counseled their patients after a myocardial infarction on resumption of sexual activity, and studies demonstrated that many patients never resumed such activity at its prior frequency, if at all.²² Although today's cardiologists have the data listed above on which to rely in order to recommend the early resumption of sexual relations, there are still reasons why patients may not do so. The multiplicity of drugs prescribed to modern-day patients often includes medications that may impair sexual function. Postinfarction depression and associated impotence are common,²³ and routine counseling on return to sexual activity is almost certainly lacking in many cases. Even when sexual activity is resumed, it often continues at a lesser frequency than before the myocardial infarction.²⁴

Although return to sexual activity after a myocardial infarction is generally safe, it is important to recognize that all the studies of the cardiovascular response to sexual activity (including assessment of the risk of triggering a myocardial infarction²⁵) were performed in subjects able to perform sexually, unaided by pharmacologic therapy. We cannot accurately extrapolate from these small studies the risks of drug-facilitated intercourse in previously impotent patients. Since a major cause of erectile dysfunction is vascular disease, not only the patient post infarction but also many other previously impotent patients may have coronary artery disease, either symptomatic or asymptomatic. Caution is also warranted in the patient with poorly controlled hypertension. Indeed, the very sparse data on sexual activity in patients with hypertension suggest that peak systolic blood pressure may reach more than 225 mmHg.²⁶

With the use of sildenafil there is a risk that some patients who previously would have been physically unable to have sexual intercourse may now be exposed to sexually induced myocardial ischemia. In the expert consensus document, several recommendations were given for the cardiac patient who wishes to take sildenafil.²⁰ Apart from the obvious contraindications in patients requiring nitrate therapy, those patients who are taking a combination of antihypertensive medications were considered to be at high risk of sildenafil-induced hypotension.^{27, 28} Exercise treadmill testing, in order to uncover stress-induced ischemia, was suggested in those patients with suspected or known coronary artery disease. The authors pointed out that the ability to achieve more than 5 METS without myocardial ischemia during an exercise tolerance test made the risk of coitus-induced ischemia unlikely. However,

they also stressed that firm data are lacking and that the physical and emotional stress of sexual intercourse can be excessive in some people, particularly in those who have not performed this activity in some time or are not in good condition.

Why Should Sexual Activity Provoke Myocardial Ischemia—Is It Simply the Workload?

Sexual activity clearly has both a physical and emotional component, and the various components of the sexual response are characterized by a complex interaction of the sympathetic and parasympathetic arms of the autonomic nervous system. The parasympathetic nervous system is responsible for the production and maintenance of erection, whereas the sympathetic nervous system, by virtue of its vasoconstrictor effect on the smooth muscle in the erectile tissue arteries, is responsible for maintaining flaccidity and producing detumescence after orgasm. It is intriguing that penile detumescence after orgasm is mediated by noradrenergically induced tonic vasoconstriction, yet preorgasmic tachycardia is also sympathetically mediated and occurs at a time when erection is maintained. This demonstrates the fine balance between the neural and biochemical components of normal sexual physiology.

Among the advances in cardiovascular pathophysiology in the last decade is a recognition that endothelial dysfunction in patients with coronary artery disease is associated with paradoxical coronary artery vasoconstriction during mental and physical exertion.^{29–33} This phenomenon is also sympathetically mediated and can be entirely blocked by the intracoronary administration of phentolamine.³⁴ One might therefore postulate that during sexual intercourse, when sympathetic tone is high, patients with coronary artery disease (and hence endothelial dysfunction) may be prone to coronary artery vasoconstriction. In one study, 31% of patients with documented coronary artery disease had electrocardiographic evidence of myocardial ischemia (painless or painful) during sexual intercourse.⁷ Although this was a highly selected population, it demonstrates that, despite relatively modest metabolic demands, significant ischemia may occur.

Since psychological factors are clearly very potent during sexual activity, it is easy to understand why tachycardia or ischemia in some patients may seem excessive for the amount of physical work. While it is likely that myocardial ischemia during sexual intercourse is provoked by a combination of neurally mediated and physical factors, at present this remains unproven. In the era of oral medications for erectile dysfunction, this is a direction of research that is clearly important.

What More Do We Need to Know about the Cardiovascular Response to Sexual Activity?

Several aspects of cardiovascular function are fertile for investigation. Among them are the effects of sexual intercourse on ventricular electrical stability, on cardiac hemodynamics,

and on coronary artery tone. In patients with left ventricular dysfunction, it is conceivable that relatively unopposed sympathetic tone may lead not only to myocardial ischemia but also to an increased propensity to ventricular arrhythmias. Studies utilizing Holter monitoring have demonstrated that some patients do develop ventricular arrhythmias during sexual intercourse,⁸ but their clinical significance is unknown.

Noninvasive tests of ventricular stability more sensitive than Holter monitoring have recently been developed. Among the newer cardiac tests is the measurement of T-wave alternans.^{35–38} T-wave alternans, a microvolt phenomenon, is heart rate related and, when present, is a marker of an increased propensity to sudden cardiac death. T-wave alternans during exercise testing occurs in a significant proportion of patients with coronary artery disease and left ventricular dysfunction.³⁷ Concomitant with the development of newer cardiac studies has been the introduction of laboratories dedicated to the study of sexual function and dysfunction. In these laboratories, sexual arousal can be induced by the combined use of erotic visual stimuli (delivered individually to the patient through special spectacles) and genital stimulation. At the same time a variety of physiologic events can be monitored. The occurrence of T-wave alternans could easily be recorded in the laboratory during sexual activity and could be correlated with similar electrical abnormalities detected during standard exercise testing.

The portability of the equipment used to provoke sexual arousal would also permit more invasive cardiac studies. It should be possible to determine cardiac output utilizing Swan-Ganz catheterization and even to perform left-sided cardiac catheterization and coronary angiography. Physical and mental stress testing has been performed successfully during cardiac catheterization, and quantitative coronary angiography has demonstrated that diseased coronary arteries show a paradoxical vasoconstrictor response to mental stress and physical exertion.^{29–33} It is likely that the same finding may occur with sexual activity, but it is also possible that the autonomic influences on the heart may be different and that patients with coronary disease may have more (or less) paradoxical vasoconstriction during sex than during mental or physical exertion. Clearly, it is important to know the likely effect that sexual intercourse has on coronary artery tone when advising a patient with severe coronary artery disease about the use of pharmacologic therapy for erectile dysfunction.

A particularly intriguing question is the role that drugs prescribed for erectile dysfunction have on coronary tone. Sildenafil was originally developed as an antianginal agent and has potent vasodilator properties. Oral phentolamine is now being investigated as a therapy for impotence. In preliminary clinical trials, this drug has been shown to be effective for erectile dysfunction³⁹ and, as noted, it is effective in preventing stress-induced coronary vasoconstriction when administered into the coronary arteries. Whether or not the blood level achieved after oral phentolamine would be sufficient to prevent paradoxical vasoconstriction is unknown but, if sufficient, such an agent might reduce the likelihood of ischemia during sexual activity.

The twenty-first century will continue to see advances in cardiology, urology, and every other discipline in medicine. With the aging of America and the desire to continue a healthy lifestyle into an increasingly older age, an increasing number of older patients will be requesting help with erectile dysfunction—a condition formerly believed to be an inevitable result of aging. As physicians we have a duty to respond to these requests, but we are also duty bound to respond in an educated manner. In order to do so, we must acknowledge that most of the framework of knowledge in this field was gathered about a quarter of a century ago. As we enter the new millennium, we must insist that our understanding of cardiovascular sexual physiology and pathophysiology catches up with the remarkable advances in other aspects of cardiovascular function that have been made in the last 25 years.

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