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Sexual Response in Cardiovascular Disease

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The cardiovascular response to sexual activity in men and women is similar to other daily activities and not excessive between couples in a long-standing relationship. The risk of an ischemic event during sexual activity is low and death very uncommon. Sexual dysfunction is common in cardiac patients and, in men, may occur before cardiac symptoms, with a time interval of 2 to 5 years. Exercise testing fails to identify nonobstructive but vulnerable-to-rupture lipid-rich plaques. Cardiac computed tomography identifies these silent plaques and triggers an aggressive risk-reducing management plan. As erectile dysfunction in men over 40 years of age is usually of vascular (endothelial dysfunction) origin, they should be considered "cardiovascular equivalents," and secondary prevention guidelines should be followed.

Cardiovascular disease (CVD) is the greatest cause of morbidity and mortality in the developed and developing world. Although women perceive breast cancer as their most common danger, they are seven times more likely to die from CVD (Jackson, 2008). Although nearly all of the literature focuses on men with erectile dysfunction (ED) and their risk of a cardiac event, it follows that men and women with or without sexual dysfunction (or disorder) will be at risk because of the CVD prevalence. Therefore, we need to consider the cardiovascular response to sexual activity and the risks, whether real or imagined.

Cardiovascular Response to Sex

The cardiovascular response to sexual activity, including intercourse, is similar to mild to moderate daily nonsexual effort. Several investigators, using ambulatory electrocardiography (ECG) and blood pressure monitoring, have compared heart rate, electrocardiographic, and blood pressure responses during sexual activity and other normal daily activities. Nemec, Mansfield, and Kennedy (1964) evaluated 10 healthy men, comparing heart rate and blood pressure responses during sexual intercourse with their wives at home. They recorded only modest changes, whether the man was on top or underneath. When the man was on top, the peak heart rate was 114 ± 14 beats per minute returning to 69 ± 12 beats per minute by 120 sec post orgasm; and when the man was underneath, a similar peak heart rate of 117 ± 4 beats per minute was recorded. The peak blood pressure responses were similar for both positions, with a systolic reading at orgasm of 160 mg Hg. Bohlen, Held, Sanderson, and Patterson (1984), again using 10 healthy men, looked at man-on-top, woman-on-top, self-stimulation, and partner stimulation and found no significant differences in heart rates or blood pressure responses. Although less information is available on women, in a postmyocardial infarction study, cardiovascular responses were similar, with a peak heart rate in men of 111 beats per minute and in women of 104 beats per minute and similar recovery times of 3.1 min and 2.6 min, respectively (Garcia-Barreto, Sin-Chesa, Rivas-Estany, Nieto, & Hemondez-Catiero, 1986). In my study of stable angina patients using 24-hr ECG monitoring, the heart rate response averaged 122 beats per minute with a range of 102 to 137 (30 men and 5 women) during intercourse compared to a maximum of 124 beats per minute during the rest of the day (Jackson, 1981). Many years on, my conclusion remains valid: "There is no reason why most patients with angina cannot have a normal sex life, and physicians should ask and advise routinely" (p. 37).

Expressed as a multiple of the metabolic equivalent of the task (MET; 1 MET is the energy expenditure at rest, which is approximately 3.5 ml of oxygen per kg body weight per minute), sexual activity between couples in a long-standing relationship is associated with a peak workload of 3 to 4 METs at orgasm, although younger couples, who may be more vigorous in their activity, may expend 5 to 6 METs (Jackson & Hutter, 2006). The average duration of intercourse is 5 to 15 min, so sexual intercourse is not an extreme or sustained

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Table 1. *Metabolic Equivalent of Task Units (METs) as a Guide to Relating Daily Activity to Sexual Activity*

Daily Activity	METs
Sexual intercourse with established partner	
Lower range (normal)	2–3
Lower range orgasm	3–4
Upper range (vigorous activity)	5–6
Lifting and carrying objects (9–20 kg)	4–5
Walking 1.6 km (1 mile) on the level in 20 min	3–4
Golf	4–5
Gardening (digging)	3–5
Do-it-yourself, wallpapering, and so forth	4–5
Light housework (e.g., ironing, polishing)	2–4
Heavy housework (e.g., making beds, scrubbing floors, cleaning windows)	3–6

cardiovascular stress. Casual sex may involve a greater cardiac workload due to lack of familiarity and age mismatch, most often an older man with a younger woman (Drory, 2002).

With our knowledge of METs, we can advise patients on sexual activity using a simple comparison with other activities, such as walking 1 mile (1.6 km) on the level in 20 min (Table 1).

Cardiac Risk

Only a small risk of myocardial infarction is associated with sexual activity. The relative risk of a myocardial infarct during the 2 hr after sexual intercourse is shown in Table 2 (Müller, Mittleman, MacLure, Sherwood, & Tofler, 1996).

The baseline absolute risk of a myocardial infarction during normal daily life is low—1 chance in a million per hour for a healthy adult and 10 chances in a million per hour for a patient with documented cardiac disease. During the 2 hr after sexual intercourse, the risk increases to 2.5 in a million for a healthy adult and 25 in a million for a patient with documented cardiac disease; more importantly, there is no increased risk in those who are physically active.

Moller et al. (2001), in a similar study in Sweden, reported identical findings. Taking a baseline annual rate of 1% for a 50-year-old man, as a result of weekly sexual activity, the risk of a myocardial infarction increases to 1.01% in those without a history of a previous myocardial infarction and to 1.1% in those with a previous history.

Coital sudden death is very rare. In three large studies, death related to sexual activity was 0.6% in Japan, 0.18% in Frankfurt, and 1.7% in Berlin. Extramarital sex was responsible for 75%, 75%, and 77%, respectively; and the victims were men in 82%, 94%, and 93% of cases, respectively (Drory, 2002). The partnership of an older man with a younger woman was the most common setting. Excessive drinking and sex following too closely to a large meal were most frequently associated with coital sudden death.

Table 2. *Relative Risk of Myocardial Infarction During the 2 Hr After Sexual Activity: Physically Fit Equals Sexually Fit*

Patient Type	Relative Risk (95% Confidence Interval)
All patients	2.5 (1.7–3.7)
Men	2.7 (1.8–4.0)
Women	1.3 (0.3–5.2)
Previous myocardial infarction	2.9 (1.3–6.5)
Sedentary life	3.0 (2.0–4.5)
Physically active	1.2 (0.4–3.7)

ED and Vascular Disease

ED is common, affecting 40% of men over 40 years of age and increasing in frequency with age (Porst & Sharlip, 2006). Over 50% of men with coronary artery disease (CAD) have ED. The common denominator is endothelial dysfunction (yet another "ED"), and the equation ED (erectile dysfunction) = ED (endothelial dysfunction) is frequently emphasized (Solomon, Man, & Jackson, 2003).

For normal vasodilatation to occur in the blood vessels that supply the penis, the endothelium must be intact and functioning normally. Once believed to be an inert monolayer of cells simply lining blood vessels, the endothelium is now recognized as having a primary role in the local regulation of vessel function and vascular homeostasis. Endothelial dysfunction is defined as an abnormal endothelial response that leads to a reduction in the bioavailability of nitric oxide (NO) and subsequently impaired vasodilatation. It has been found to be associated with several disorders of the cardiovascular system including diabetes, hypertension, hyperlipidemia, heart failure, and cigarette smoking. The vascular endothelium acts as a "plasma-tissue barrier" and has a crucial role in controlling vascular function, with the balance between endothelium-derived vasodilators and vasoconstrictors determining vascular tone and the pathophysiologic consequences (Hurrairah & Ferro, 2004). In addition, the reduction in NO bioavailability can adversely affect platelet aggregation and promote vascular wall inflammation and smooth muscle cell proliferation.

The clinical consequences of endothelial dysfunction include the development of atherosclerosis, acute coronary syndromes, cardiac failure, and ED. It is now recognized that a defect in the NO-cyclic guanosine 3'5'-monophosphate system in smooth muscle cells before the development of overt CVD in men with ED is an early marker of systemic vascular abnormalities (Solomon et al., 2003).

Table 3. *Risk Factors*

Coronary Artery Disease	Erectile Dysfunction
Age	Age
Dyslipidemia	Dyslipidemia
Diabetes	Diabetes
Smoking	Smoking
Sedentary lifestyle	Sedentary lifestyle
Obesity	Obesity
Depression	Depression
Male gender	Coronary artery disease
	Peripheral vascular disease

Table 4. *Calcium Scores*

Range	Classification	Coronary Events RR[a]
0	None	0
1–100	Mild	1.9
100–400	Moderate	4.3
400–999	Severe	7.2
>1000	Extensive	10.8

[a]RR = relative risk using zero as a reference over 3 to 5 years.

A body of literature has now identified ED as a marker for silent CAD, with an average time interval between ED and a coronary presentation of 2 to 5 years (Hodges, Kirby, Solanki, O'Donnell, & Brodie, 2007). ED and CAD share the same risk factors (Table 3), and the penile arteries and coronary arteries share the same endothelium (Jackson, 2007).

As the penile arteries are smaller than the coronary arteries (1–2 mm vs. 3–4 mm), a similar degree of endothelial dysfunction may negatively influence blood flow leading to ED while being clinically silent in the larger arteries. Sudden cardiac death from CAD or an acute coronary syndrome is frequently due to rupture of a previously silent lipid-rich plaque of <50% stenosis (narrowing of the artery). In a study of 300 men with acute chest pain and angiographically proven CAD, the prevalence of ED was 49% ($n = 147$). ED was experienced before the acute event in 99 of the 147 (67%), with a mean time interval of 38.8 months (range = 1–168 months; Montorsi, Montorsi, & Shulman, 2003). ED was, therefore, predicting plaque rupture as well as being associated with obstructive CAD. Screening for vascular risk is widely advocated in men with ED but having no cardiac symptoms, especially if over 40 years of age or diabetic. The Princeton Consensus Guidelines state that a man with ED and no cardiac symptoms is a cardiac or vascular patient until shown otherwise (Jackson, Rosen, Kloner, & Kostic, 2006).

Current advice includes exercise testing in high cardiovascular risk men in case a more detailed cardiological assessment is indicated. However, exercise ECG is of greatest value primarily in the presence of flow-limiting lesions, and thus this procedure may fail to identify the vulnerable-to-rupture subclinical plaque and <50% stenosis. As ED is a marker of both acute coronary syndromes and chronic CAD, the need for a means of assessing these subclinical lesions is required.

One such assessment procedure is multi-detector computed tomography (MDCT). This relatively non-invasive, outpatient procedure has approximately the same x-ray exposure as an invasive coronary angiogram, and recent studies have confirmed its accuracy compared with invasive angiography (Nicol & Padley, 2007). More importantly, MDCT can identify the presence of early (<50%) coronary disease, with the presence of coronary calcium as detected through MDCT almost always representing atherosclerosis (Erbel, Möhlenkamp, Kerkohoff, Budde, & Schmermund, 2007). Follow up of patients for 1 to 2 years with a negative MDCT score (i.e., absence of coronary calcium) identifies a good prognosis, and a healthy lifestyle is recommended (Table 4). In contrast, the risk associated with any degree of calcium compared with no calcium increases the risk of a cardiovascular event fourfold, and the rate of myocardial infarction and cardiovascular death in the next 3 to 5 years reaches 4.6% and 7.1% in the two high-risk groups (score of 400–999 and >1,000, respectively). More importantly, the risk is greater in the asymptomatic man (e.g., the man with ED with still increasing levels), probably because risk reduction therapy is not in place. It is of considerable interest to note the time window similarities between ED, MDCT, and CAD. Specifically, ED to CAD event is 3 to 5 years, and MDCT to CAD event is also 3 to 5 years.

Although at the present time no therapy has convincingly been able to stop the progression of coronary calcification, statins are known to substantially reduce cardiovascular risk, probably as a consequence of their anti-inflammatory actions and plaque stabilization. Currently, those at increased risk based on calcium scoring are advised on lifestyle changes and drug treatment according to standard guidelines for CVD prevention (Graham et al., 2007).

Jackson and Padley (2008) recently identified significant CAD in men presenting with ED and no cardiac symptoms, and in nondiabetic men we have shown the presence of CAD in the absence of an abnormal maximal exercise ECG. Although an exercise ECG may identify flow limiting and potentially prognostically important CAD, in this high-risk population a normal exercise ECG can be falsely reassuring, as it may not detect subclinical lesions of <50% stenosis. These findings are, therefore, in keeping with the documented link between ED and an acute coronary event. We believe that a patient with organic ED and increased cardiovascular risk factors should be treated as a "cardiovascular equivalent" with aggressive risk factor reduction. For the present, ED and CAD are established as comorbid conditions, and the occurrence of ED before CAD

presents a window of opportunity to reduce or prevent a subsequent coronary event.

Conclusion

ED and CAD share the same risk factors, and ED may well be an independent risk factor, as well as a marker for silent CAD. A full cardiac evaluation and vascular risk reduction strategy is advocated. Female sexual dysfunction has not been linked to CAD, but women are as much at risk as men of developing CAD whether they have FSD or not and should be managed accordingly. Sexual activity is not a severe form of cardiac stress, and the risk of an acute event is rare. Sexual advice should be routinely incorporated into a cardiac rehabilitation program.

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