Triggering Myocardial Infarction by Sexual Activity

Low Absolute Risk and Prevention by Regular Physical Exertion

James E. Muller, MD; Murray A. Mittleman, MD, DrPH; Malcolm Maclure, ScD; Jane B. Sherwood, RN; Geoffrey H. Tofler, MB; for the Determinants of Myocardial Infarction Onset Study Investigators

Objective.—To determine the relative risks of nonfatal myocardial infarction (MI) triggered by sexual activity among the general population and in patients with prior coronary heart disease.

Design.—Relative risks and effect modification were calculated by the case-crossover method, a new epidemiologic technique designed to quantify the transient change in risk following exposure to a potential disease trigger.

Setting/Participants.—A total of 1774 patients with MI were interviewed in 45 hospitals throughout the United States. Data were gathered on potential triggers of MI occurring immediately prior to the event and during the previous year. Results are presented for the 858 patients who were sexually active in the year prior to the MI, with attention to the 273 patients who had coronary artery disease prior to their index MI, and the effect of regular exertion on risk.

Main Outcome Measure.—The relative risk of nonfatal MI following sexual activity.

Results.—Of the 858 patients, 79 (9%) reported sexual activity in the 24 hours preceding MI, and 27 (3%) reported sexual activity in the 2 hours preceding onset of symptoms of MI. The relative risk of MI occurring in the 2 hours after sexual activity was 2.5 (95% confidence interval [CI], 1.7-3.7). The relative risk of triggering onset of MI among patients with a history of prior angina (2.1 [95% CI, 0.8-5.8]) or prior MI (2.9 [95% CI, 1.3-6.5) was not greater than that observed in those without prior cardiac disease. Sexual activity was a likely contributor to the onset of MI in only 0.9% of cases and regular exertion was associated with decreasing risk.

Conclusions.—Sexual activity can trigger the onset of MI. However, the relative risk is low, and since the absolute hourly risk of MI is extremely low, the absolute risk increase caused by sexual activity also is extremely low (1 chance in a million for a healthy individual). Moreover, the relative risk is not increased in patients with a prior history of cardiac disease and regular exercise appears to prevent triggering. These findings should be useful for counseling patients and decreasing the fear of sexual activity that often prevents complete rehabilitation from cardiovascular disease.

(*JAMA*. 1996;275:1405-1409)

From the Department of Medicine, Harvard Medical School (Drs Muller, Mittleman, and Tofler); Cardiovascular Division, Department of Medicine, Deaconess Hospital (Drs Muller, Mittleman, and Tofler and Ms Sherwood); and Department of Epidemiology, Harvard School of Public Health (Drs Mittleman and Maclure), Boston, Mass.

A complete list of the Determinants of Myocardial Infarction Onset Study Investigators appears in *N Engl J Med*. 1993;329:1677-1683.

Reprints: James E. Muller, MD, Institute for Prevention of Cardiovascular Disease, Cardiovascular Division, Deaconess Hospital, 1 Autumn St, Fifth Floor, Boston, MA 02215.

ANECDOTAL case reports suggest that sexual activity can trigger myocardial infarction (MI),[1-3] but the relative risk of MI after sexual activity, the frequency of sexually triggered MIs, and possible methods of reducing risk have not been studied. The lack of data on the cardiac risk of sexual activity causes a major problem for patients who have cardiac disease, their spouses, and the health care professionals who provide counsel on this topic.[4,5]

Development of the field of study of triggering of cardiovascular disease has provided data and a new epidemiologic technique to address these issues.[6-10] Over the past 6 years, members of the National Heart, Lung, and Blood Institute–funded Myocardial Infarction Onset Study interviewed more than 1700 patients approximately 1 week following MI to identify possible triggers, including sexual activity. Prior reports from this study have quantitated triggering of MI by heavy physical exertion[10] and anger,[7] and have demonstrated that regular physical exertion protects against triggering by heavy physical exertion.[10]

For editorial comment see p 1447.

The data on sexual activity as a trigger of MI from the Onset Study provide information on risk in the subset of patients known to have coronary artery disease prior to the MI for which they were interviewed.

METHODS

Study Population

The Onset Study was conducted in 22 community hospitals and 23 tertiary care centers.[10] Between August 1989 and March 1993, a total of 1774 patients (1236 men and 538 women, aged 20-92 years) were interviewed a median of 4 days (range, 0-30 days) following their MIs.

Interviewers identified eligible patients by reviewing coronary care unit admission logs and patient charts. For inclusion, patients were required to meet all of the following criteria: an elevated creatine kinase level, with positive MB isoenzymes; identifiable onset of pain or other typical symptoms of MI onset; and ability to complete a structured inter-

view. The protocol was approved by the institutional review board at each participating center, and informed consent was obtained from each patient.

Interview

Interviewers were trained by personal instruction, a training manual, and an instructional videocassette and through ongoing feedback from the study coordinator. Approximately one third of the interviews were audiotaped for randomly selected quality control checks of the coding accuracy.

The interview identified the time, place, and quality of pain and other symptoms of MI and the timing and intensity of exposure to potential triggers of MI onset. To control for possible confounding due to the co-occurrence of outbursts of anger, we classified patients as either exposed or not exposed to outbursts of anger during the hazard and control period, using the onset of anger scale.[7] Sexual intercourse was the activity studied, and patients were asked the following sequence of questions: "When was the last time before your heart attack that you had sexual intercourse?" Subjects reporting sexual intercourse in the preceding 12 months were then asked the time of the second occurrence of sexual intercourse prior to the MI and "On average, over the past year how often did you have sexual intercourse?" In addition, patients who reported sexual intercourse within 26 hours of the onset of MI symptoms were asked whether or not they had engaged in sexual intercourse during any of the 26 hours before their symptoms began. To assess whether regular physical exertion could alter the risk that sexual activity might trigger an MI, we collected data on the usual frequency of heavy exertion as previously described.[10,11] For the present study, a cut point of 6 METs (metabolic equivalents of oxygen consumption) was selected prospectively because regular exertion at this level has been shown to reduce the risk of triggering MI by isolated bouts of heavy exertion.[10]

Study Design

A new epidemiologic technique, called the case-crossover design (Figure 1), was developed for the Onset Study.[8-10] This approach was developed to assess the change in risk of an acute event during a brief "hazard period" following exposure to a potential trigger. With this method, control information for each patient is based on his or her past exposure experience.[8-10]

A 2-hour hazard period immediately preceding MI onset was compared with 2 types of control data obtained from the patients: their usual frequency of sexual activity during the past year and their actual sexual activity in the comparable 2-hour "control period" at the same time on the day before the MI. To help maintain comparability of reporting of exposures for the hazard and control periods, the interview treated the 26-hour period before MI onset as 1 long hazard period.

Statistical Analysis

The analysis of case-crossover data is a new application of standard methods for stratified data analysis.[8,9,12,13] In this analysis, the stratifying variable is the individual patient, as in a crossover experiment.

The ratio of the observed frequency of sexual activity in the hazard period to the expected frequency (from the control information) was used to calculate estimates of the odds ratio as a measure of relative risk.[8,9] The primary method of analysis was to determine expected frequencies on the basis of usual annual frequency of sexual activity. The amount of person-time exposed to sexual activity was estimated by multiplying the reported usual annual frequency of exposure by the duration of the hypothesized hazard period. Unexposed person-time was then calculated by subtracting the exposed person-time in hours from the number of hours in a year. Hazard periods of varying durations were analyzed using methods for cohort studies with sparse data in each stratum.[8,9,14]

As a secondary method of analysis, the expected frequency in the hazard period was determined based on the frequency of sexual activity in the control period on the day prior to onset. By matching on time of day, potential confounding by clock time due to the circadian variation in MI incidence and the timing of sexual activity throughout the day was controlled for in this analysis.[9] Relative risks were computed using standard methods for matched-pair case-control studies. However, instead of concordant and discordant pairs of subjects, each patient contributed a pair of intervals, ie, a hazard and a control period, which were either concordant or discordant for sexual activity.[8-10,12] Ninety-five percent confidence intervals (CIs) and 2-sided P values were computed using exact methods based on the binomial distribution.[12] Conditional logistic regression was used to control for within-person confounding.[8,9]

Modification of the relative risk was assessed by comparing relative risks in subgroups, defined by different levels of the potential effect modifier. The patients were divided into 3 age groups chosen a priori on the basis of prior publications from the Onset Study,[7,10] and

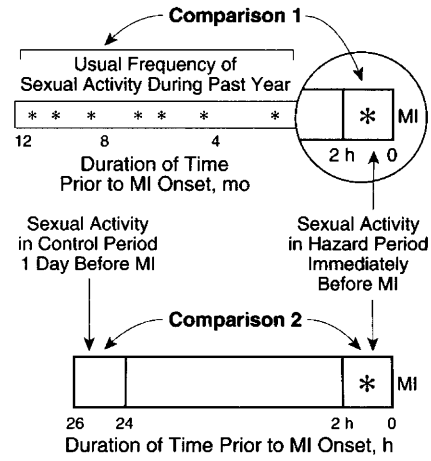

Figure 1.—The case-crossover study design. The 2-hour period prior to myocardial infarction (MI) is defined as the hazard period. Comparison 1 contrasts exposure to potential triggers in the hazard period with the frequency of exposure expected based on the reported usual frequency over the prior year. In comparison 2, exposure in the hazard period is compared with exposure in a control period at the same time on the preceding day. Reproduced from *Circulation*[7] with permission from the American Heart Association.

the relative risk within each group was estimated. Differences in the relative risks between subgroups were compared using the χ^2 test for homogeneity.[12] To estimate induction time (the length of time from sexual activity to the onset of MI), we calculated relative risks for each 1-hour period before MI onset, statistically controlling for possible exposure to subsequent episodes of sexual intercourse.[15]

RESULTS

The characteristics of the patients interviewed are presented in Table 1. Of the 1774 patients with MI who were interviewed, 141 (8%) chose not to answer the questions regarding sexual activity. Of the remaining 1633 patients, 858 (48%) reported that they were sexually active in the year preceding MI. Included in the group of 1633 patients were 643 patients who had a history of a prior MI or angina; of these patients, 273 (42%) were sexually active.

Because subjects who were not sexually active did not contribute information to the assessment of the relative risk of triggering MI onset, all analyses of risk are based on the data from the 858 sexually active patients. Table 2 shows the distribution of the usual frequency of sexual activity among these patients. Of these patients, 79 (9%) reported sexual activity in the 24 hours preceding MI, and 27 (3%) reported sexual activity in the 2-hour period preceding symptom onset.

Table 1.—Characteristics of the Study Population*

Characteristic	Sexually Active (n=858)	Not Sexually Active (n=775)
Mean (SD) age, y	54.9 (11.4)	67.8 (11.1)
Age, y		
<50	291 (34)	50 (6)
50-69	479 (56)	354 (46)
≥70	88 (10)	371 (48)
Sex		
Male	704 (82)	438 (57)
Female	154 (18)	337 (44)
Medical history		
MI initial presentation of CAD	585 (68)	405 (52)
Prior MI	192 (22)	272 (35)
Prior angina	186 (22)	235 (30)
Hypertension	337 (39)	388 (50)
Diabetes mellitus	124 (14)	171 (22)
Medication use prior to MI		
Aspirin	251 (29)	233 (30)
Calcium channel blockers	169 (20)	218 (28)
β-Blockers	151 (18)	177 (23)
ACE inhibitors	85 (10)	99 (13)
Type of hospital where interviewed		
Community hospital	381 (44)	431 (56)
Tertiary care center (admitted)	191 (22)	126 (16)
Tertiary care center (transferred)	286 (33)	218 (28)

*Values given are number (percentage) unless otherwise indicated. MI indicates myocardial infarction; CAD, coronary artery disease; and ACE, angiotensin-converting enzyme.

Table 2.—Usual Frequency of Sexual Activity Among the 858 Sexually Active Patients

Usual Frequency	No. (%)
>2/wk	188 (22)
1-2/wk	200 (23)
1-3/mo	258 (30)
1-11/y	212 (25)

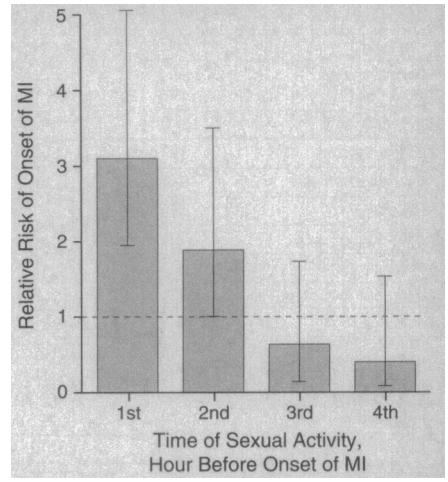

Figure 2.—Induction time of onset of myocardial infarction (MI) after sexual activity. Each of the 4 hours prior to MI onset was assessed as an independent hazard period, and sexual activity in each hour was compared with the control intervals. Only the 2 1-hour periods immediately prior to MI onset were associated with an increased risk, suggesting an induction time of less than 2 hours. Error bars indicate 95% confidence intervals. The dotted line represents the baseline risk.

Figure 2 shows the induction time, ie, the time from sexual activity to the onset of MI, with relative risks for each 1-hour period before MI onset, controlling for subsequent exposure. The relative risk was increased only during the first 2 hours after sexual activity, indicating that the induction time was less than 2 hours. The risk that an MI would begin in the 2-hour period immediately following sexual activity was 2.5 (95% CI, 1.7-3.7).

In an analysis using the frequency of sexual activity during the equivalent 2-hour period on the day prior to onset as the control information, 27 patients had engaged in sexual activity only in the 2-hour hazard period, compared with 8 in the control period (the same 2-hour period the day before). None had engaged in sexual activity at both times. This analysis yielded a relative risk for MI of 3.4 (95% CI, 1.5-8.6).

Only 2 patients reported both heavy exertion of at least 6 METs and an episode of sexual intercourse during the hazard period. No patients reported exposure to both in the control period. No patients reported exposure to both sexual activity and outbursts of anger as assessed by the onset of anger scale[7] in either the hazard or the control period. The relative risk associated with sexual activity was not materially altered in a conditional logistic regression analysis that simultaneously controlled for exposure to heavy physical exertion of at least 6 METs and outbursts of anger.

Potential modifiers of the relative risk were examined (Table 3) and a statistically significant protective effect of regular exercise was observed (Figure 3). The relative risks of MI onset in the 2 hours following sexual activity decreased from 3.0 to 1.9 to 1.2 for patients who engaged in heavy physical exertion (≥6 METs) once or not at all, twice, and 3 or more times per week, respectively (P_{trend}=.01).

Among patients with a prior MI, the relative risk was 2.9, (95% CI, 1.3-6.5), which was similar to that among subjects without such a history (2.5 [95% CI, 1.6-3.8]; P=.75). Among patients with a history of angina, the relative risk of sexual activity triggering MI was 2.1 (95% CI, 0.8-5.8), and was similar to the risk observed among those without angina (2.6 [95% CI, 1.7-3.9]). There were too few women who reported sexual activity in the 2-hour hazard period preceding MI to determine if the relative risk varied by sex.

COMMENT

These findings from the Myocardial Infarction Onset Study confirm the impression, based on anecdotal reports,[1-3] that sexual activity can trigger MI. The relative risk of an MI occurring in the 2 hours following sexual activity was 2.5 (95% CI, 1.7-3.7) in patients without prior cardiac disease and 2.9 (95% CI, 1.3-6.5) in those with a history of prior MI. The findings indicate that regular exercise can reduce, and possibly eliminate, the small and transient increased risk of MI associated with sexual activity.

Additional studies are required to more precisely quantify the low risk that appears to be present. However, the validity of the main conclusion of the study, that the absolute risk of sexual activity triggering MI is low, even in patients with prior coronary disease, is not threatened by low levels of relative risk. Only 27 (1.5%) of the 1774 patients interviewed in the Onset Study reported sexual activity in the 2 hours prior to MI. Thus, after correction for chance occurrence, sexual activity was a likely contributor to the onset of MI in only approximately 0.9% of cases in this study.

None of the 88 sexually active individuals aged 70 years or older experienced an occurrence of MI in the 2 hours after intercourse. This may be due to the relatively small numbers of individuals in this age group and their lower frequency of sexual activity. On the other hand, the risk could actually be lower among these patients because of the altered β-adrenergic sensitivity of the elderly.[16]

Since the case-crossover design uses self-matching for control information, there is no variability in traditional chronic risk factors[8-10] for MI within each stratum. Thus, the case-crossover method eliminates the possibility of confounding

Table 3.—Potential Modifiers of Relative Risk of Myocardial Infarction Onset Following Sexual Activity*

Characteristic	No.	No. With Sexual Activity During 2 h Before MI	Relative Risk (95% Confidence Interval)	P†
All cases	858	27	2.5 (1.7-3.7)	...
Age, y				
<50	291	15	3.2 (1.9-5.4)	
50-69	479	12	2.1 (1.2-3.8)	.29
≥70	88	0	...	
Sex				
Male	704	25	2.7 (1.8-4.0)	.33
Female	154	2	1.3 (0.3-5.2)	
Clinical history				
Angina	186	4	2.1 (0.8-5.8)	.71
No angina	672	23	2.6 (1.7-3.9)	
Hypertension	337	12	3.3 (1.8-5.9)	.26
No hypertension	521	15	2.1 (1.3-3.5)	
Obese	290	8	2.3 (1.1-4.6)	.73
Not obese	568	19	2.6 (1.7-4.2)	
Prior infarction	192	6	2.9 (1.3-6.5)	.75
No prior infarction	666	21	2.5 (1.6-3.8)	
Smoking				
Current smoker	357	11	2.3 (1.3-4.3)	.77
Nonsmoker	497	16	2.6 (1.6-4.4)	
Exertion ≥6 METs				
≤1/wk	679	23	3.0 (2.0-4.5)	
2/wk	33	1	1.9 (0.2-17.1)	.01
≥3/wk	146	3	1.2 (0.4-3.7)	
Medications				
β-Blocker use	151	4	2.3 (0.9-6.4)	.89
No β-blocker use	707	23	2.5 (1.7-3.8)	
Aspirin use	251	6	1.8 (0.8-4.1)	.35
No aspirin use	607	21	2.8 (1.8-4.4)	

*Obesity was defined as body mass index above 29 kg/m². METs indicates metabolic equivalents of oxygen consumption; and ellipses, not applicable.
†Test for homogeneity was used for 2 categories, test for trend when 3 or more categories were compared.

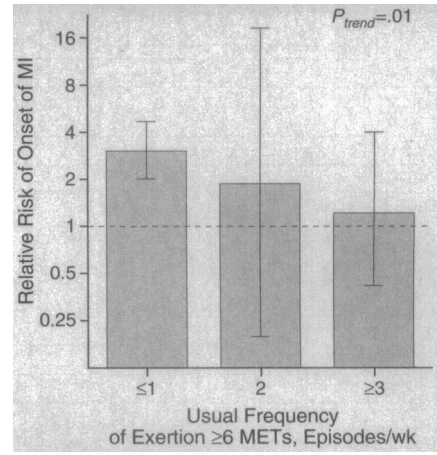

Figure 3.—Modification by regular exertion of the risk that sexual activity might trigger myocardial infarction (MI). Error bars indicate 95% confidence intervals. The dotted line represents the baseline risk. METs indicates metabolic equivalents of oxygen consumption.

by these chronic underlying cardiac risk factors. However, the method is susceptible to confounding by potential triggers that vary over time in each individual (eg, morning hours, heavy exertion, anger). An analysis that took into account these other well-documented triggers of MI onset did not materially alter the results, indicating that sexual activity can trigger the onset of MI independently of these other triggers.[7]

The present study provides information of great value for counseling the more than 500 000 patients in the United States who survive MI each year and the 11 million patients with existing cardiac disease.[17] Prior to the availability of these data, there was no published information on the risk of sexual activity in patients with cardiac disease. Counseling, which was often ineffective in decreasing fear of triggering a cardiac event, was based on the presumed "physiological equivalence" of sexual activity and climbing stairs.[18]

With these data, health care professionals counseling patients with cardiac disease can reassure them that, although their baseline risk of MI is increased, sexual activity has been documented to have a low likelihood of triggering an MI. The risk is particularly low for patients who engage in regular exercise, which has been shown to decrease the cardiac work required during sexual activity[19] and also decrease the risk of triggering onset of MI.

The most powerful information for counseling is not the relative risk of a potential trigger, but the absolute risk difference the activity produces. While the Onset Study findings can be used to calculate relative risk, they alone are not sufficient to determine absolute risk because the baseline risk of these patients, all of whom experienced an MI, is not measured. However, data on baseline risk from previously studied populations, similar to that from which the Onset Study patients originated, can be combined with relative risks from the Onset Study to estimate absolute risks.

Data from the Framingham Heart Study indicate that the baseline risk that a 50-year-old, nonsmoking, nondiabetic man will experience an MI is approximately 1% per year, or approximately 1 chance in a million per hour.[20,21] Since the relative risk of MI is approximately doubled by sexual activity, by engaging in sexual activity such an individual would only increase his hourly risk to 2 in a million, and only for a 2-hour period.

The baseline yearly risk of reinfarction or death for an individual with a prior MI is approximately 10%, and less than 3% if the individual can exercise beyond 7 METs without symptoms on an exercise test.[22] For individuals in the 10% annual risk group, sexual activity would transiently double the risk from 10 in a million per hour to only 20 in a million per hour.

Although sexual activity doubles the risk of MI, the effect of sexual activity on annual risk is negligible because the absolute risk difference is small, the risk is transient, and the activity is relatively infrequent. For example, for the individual free of cardiac disease described herein, weekly sexual activity would only increase his annual risk of MI from 1% to 1.01%. Other potential triggers of MI such as heavy exertion and episodes of anger may cause a greater increase in annual risk because of their more frequent occurrence.

The present study has several potential limitations. No data were collected on risk during sexual intercourse with the patient's usual partner vs another partner, or the risk of different sexual activities and positions. In addition, because the data are based on self-report by patients, misclassification of exposure may occur. For example, patients may be reluctant to report that sexual activity occurred prior to their MI. The effect of such a bias would be to reduce the magnitude of the estimated relative risk. In an effort to minimize such reporting bias, as well as to maintain patient confidentiality, efforts were made to ensure the patient's privacy during the interview. Furthermore, to obtain

comparable reporting of sexual activity for all of the hourly intervals during the 26 hours preceding the MI, patients were not informed of the duration of the hypothesized hazard period.

Another limitation of this study is the potential bias created by the lack of data on the possibility that sexual activity might be more likely to cause sudden death than nonfatal MI. If this were the case, the risk of sexual activity triggering nonfatal MI would be underestimated. In addition, reassurance of patients requires consideration of the possibility that sexual activity could trigger either sudden death or nonfatal MI. However, for the following reasons, we do not believe this potential bias threatens the overall conclusion of the study. First, the baseline risk of sudden death is much lower than the baseline risk of nonfatal MI. Summary data for the United States indicate that there are 1.2 million MIs per year that do not cause sudden cardiac death vs approximately 300 000 sudden cardiac deaths, indicating that MI not associated with sudden death is 4 times more likely than sudden cardiac death.[17] Second, data for the related trigger of heavy exertion do not indicate a selective triggering of sudden cardiac death over nonfatal MI. The increase in risk of nonfatal MI caused by heavy exertion is similar to the increase in risk of sudden death caused by exertion.[10] Third, there are no data suggesting that sexual activity is more likely to trigger sudden cardiac death than MI. On the contrary, the limited data available suggest that sudden cardiac death following intercourse is not frequent; in a series of more than 5000 sudden death cases, less than 0.6% occurred after intercourse.[3] Finally, the effect of such a bias, if it exists, on the overall conclusion of the study is minimal. Even if the absolute risk estimates were low by a factor of 10, the total risk difference associated with sexual activity would be quite small.

Because of the small number of exposed cases and the relatively large number of potential modifiers of interest, there were insufficient data to definitively evaluate possible effect modification by these and other potential modifiers. Additional studies are required to confirm these results. However, the apparent dose-response effect of exercise and the finding in our prior study that regular exercise markedly reduces the risk of triggering onset of MI by heavy exertion suggest that the relative protection offered by regular exercise is a real effect.

Determination of whether the increase in risk of MI onset following sexual intercourse is altered by these factors is of importance for understanding the pathophysiology of MI onset. However, it has only limited significance for individual behavior, because the absolute risk produced by the infrequent potential trigger of sexual activity is too small to be of practical significance.

Based on these data, physicians should strongly encourage patients with known coronary artery disease to participate in a cardiac rehabilitation program and perform regular physical exercise. Such exercise can decrease the cardiac work required for sexual activity and reduce the risk of triggering onset of MI. By communicating the data on absolute risk, it should be possible to improve rehabilitation of patients following MI and to decrease the sexual dysfunction currently caused by unrealistic fears of resumption of sexual activity.[23]

This work was supported by a grant (HL41016) from the National Heart, Lung, and Blood Institute, Bethesda, Md.

We are grateful for the assistance of Kathleen Carney in the preparation of the manuscript; to the Onset Study interviewers for their dedication; to Richard P. Mulry and Lucy Perriello, MA, for help in the conduct of the study and feedback on the development of the questionnaire; to Rosa Maria Hernandez de Sierra, RN, and Diane Walkoff for excellent technical assistance; and to the Onset interviewers who collected the valuable information.

References

1. Trimble GX. The coital coronary. *Med Aspects Hum Sex.* 1970;5:64.
2. Nalbangtil I, Yigitbasi O, Kiliccioglu B. Sudden death in sexual activity. *Am Heart J.* 1976;91:405-406.
3. Ueno M. The so-called coition death. *Jpn J Leg Med.* 1963;17:330-340.
4. Hellerstein HK, Friedman EH. Sexual activity and the postcoronary patient. *Arch Intern Med.* 1970;125:987-999.
5. Papadopoulos C, Shelley SI, Piccolo M, Beaumont C, Barnett L. Sexual activity after coronary bypass surgery. *Chest.* 1986;90:681-685.
6. Muller JE, Abela GS, Nesto RW, Tofler GH. Triggers, acute risk factors and vulnerable plaques: the lexicon of a new frontier. *J Am Coll Cardiol.* 1994;23:809-813.
7. Mittleman MA, Maclure M, Sherwood JB, et al, for the Determinants of Myocardial Infarction Onset Study Investigators. Triggering of acute myocardial infarction onset by episodes of anger. *Circulation.* 1995;92:1720-1725.
8. Maclure M. The case-crossover design: a method for studying transient effects on the risk of acute events. *Am J Epidemiol.* 1991;133:144-153.
9. Mittleman MA, Maclure M, Robins JM. Control sampling strategies for case-crossover studies: an assessment of relative efficiency. *Am J Epidemiol.* 1995;142:91-98.
10. Mittleman MA, Maclure M, Tofler GH, Sherwood JB, Goldberg RJ, Muller JE, for the Determinants of Myocardial Infarction Onset Study Investigators. Triggering of acute myocardial infarction by heavy physical exertion: protection against triggering by regular exertion. *N Engl J Med.* 1993;329:1677-1683.
11. Lee I, Paffenbarger RS, Hsieh C. Time trends in physical activity among college alumni, 1962-1988. *Am J Epidemiol.* 1992;135:915-925.
12. Rothman KJ. *Modern Epidemiology.* Boston, Mass: Little Brown & Co; 1986.
13. Wacholder S, Silverman DT, McLaughlin JK, Mandel JS. Selection of controls in case-control studies. *Am J Epidemiol.* 1992;135:1019-1028.
14. Greenland S, Robins JM. Estimation of a common effect parameter from sparse follow-up data. *Biometrics.* 1985;41:55-68.
15. Rothman KJ. Induction and latent periods. *Am J Epidemiol.* 1981;114:253-259.
16. Esler M, Kaye D, Thompson J, et al. Effects of aging on epinephrine secretion and regional release of epinephrine from the human heart. *J Clin Endocrinol Metabol.* 1995;80:435-442.
17. *1995 Statistical Supplement to Heart and Stroke Facts.* Dallas, Tex: American Heart Association; 1995.
18. Tardif GS. Sexual activity after a myocardial infarction. *Arch Phys Med Rehabil.* 1989;70:763-766.
19. Stein RA. The effect of exercise training on heart rate during coitus in the post myocardial infarction patient. *Circulation.* 1977;55:738-740.
20. Anderson KM, Wilson PW, Odell PM, Kannel WB. An updated coronary risk profile: a statement for health professionals. *Circulation.* 1991;83:356-362. American Heart Association Scientific Statement.
21. Anderson KM, Odell PM, Wilson PW, Kannel WB. Cardiovascular disease risk profiles. *Am Heart J.* 1993;121:293-298.
22. Moss AJ, Benhorin J. Prognosis and management after a first myocardial infarction. *N Engl J Med.* 1990;322:743-753.
23. Papadopoulos C, Larrimore P, Cardin S, Shelley SI. Sexual concerns and needs of the postcoronary patient's wife. *Arch Intern Med.* 1980;140:38-41.