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Intensity of physical exertion and triggering of myocardial infarction: a case-crossover study

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Aims Acute myocardial infarction (AMI) can be precipitated or triggered by discrete transient exposures including physical exertion. We evaluated whether the risk of having an AMI triggered by physical exertion exhibits an exposure–response relationship, and whether it varies by ambient temperature or by taking place indoors or outdoors.

Methods and results We conducted a case-crossover study within the Myocardial Infarction Registry in Augsburg, Germany in 1999–2003. One thousand three hundred and one patients reported levels of activity and time spent outdoors on the day of AMI and three preceding days in an interview. The case-crossover analyses showed an association of physical exertion with AMI symptom onset within 2 h, which was strong for strenuous exertion (METs $\geq$ 6) [relative risk (RR) 5.7, 95% confidence interval (CI) 3.6–9.0), and still significant for moderate exertion (METs = 5) (RR 1.6, 95% CI 1.2–2.1) compared to very light or no exertion. Strenuous exertion outside was associated with a four-fold larger RR of AMI symptom onset than exertion performed indoors, which was not explained by temperature.

Conclusion This study confirms previous results and shows a graded exposure–response relationship between physical exertion intensity and triggering of AMI onset. These findings may have implications for behavioural guidance of people at risk of AMI.

Keywords Myocardial infarction • Trigger • Epidemiology • Case-crossover • Strenuous exertion • Onset

Introduction

An individual's long-term risk of acute myocardial infarction (AMI) is determined, at least in part, by 'traditional' chronic risk factors, such as lipid levels, long-term average blood pressure, and smoking habits, and 'novel' risk factors associated with increased vascular inflammation, such as C-reactive protein.[1] However, conditional on an individual's baseline risk, acute events may be precipitated or triggered by discrete transient exposures including physical, psychological, or chemical stressors.[2]

It has previously been shown that AMI can be triggered by isolated bouts of vigorous physical activity.[3–7] However, whether lower levels of physical exertion can also trigger AMI, and the exposure–response relationship, have not been examined. According to the Council on Clinical Cardiology, cardiovascular disease patients should avoid exercise in extremely hot or cold environments,[8] but to our knowledge no previous case-crossover study quantified an interaction of strenuous exertion with its environmental circumstances.

We conducted a case-crossover study within the Cooperative Health Research in the Region of Augsburg (KORA) Myocardial Infarction Registry in Augsburg, southern Germany, to evaluate whether the risk of having AMI triggered by physical exertion exhibits an exposure–response relationship and whether the risk varies by temperature or by whether the physical activity takes place indoors or outdoors.

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Methods

Patient population

Cases were drawn from the complete case series of the population based KORA Myocardial Infarction Registry from February 1999 to December 2003. Details of the recruitment procedures and data collection have been described elsewhere.[9]

Briefly, hospitalized 24 h survivors of AMI who were 25–74 years of age and residents of the study area were routinely entered into this registry. The diagnosis of AMI was based on symptoms, enzyme elevation, and electrocardiogram, by applying the criteria established within the MONICA framework.[10] Interviews were conducted by trained research nurses on the general ward as soon as possible after the event (median: 9 days). All patients gave written informed consent for participation; the research protocol was approved by the KORA review board. After the patient's discharge from hospital, clinical data were abstracted from the medical record according to a standardized protocol. The time of onset of the AMI was defined as the time of symptom onset (typical symptoms) or the time when symptoms were most severe (atypical symptoms).[11]

Data on symptoms in connection with the current event, as well as socio-demographic characteristics, medical history, and smoking status of the patients were collected as part of the registry's routine interview. For this specific study, patients were additionally interviewed about their activities on the day of their AMI and the preceding 3 days using a standardized data collection form (diary interview). Patients reported time spent outdoors, means of transportation, premonitory symptoms, times spent sleeping, and levels of activity while awake. Using categories based on the standard MET levels of similar activities (*Table 1*), the maximum activity level was recorded for each hour of the day. Strenuous exertion was defined as self-reported activities representing vigorous exertion (6 MET) or higher (*Table 1*). Physical exertion was defined as moderate exertion (5 MET) or higher. Patients were not informed of the hypothesized hazard period and equal importance was given to the assessment of activity levels across the 4 days.

Table 1 Activity codes for the diary interview

Activity code	Description	Type of activity (selection)
1	Sleeping	
2	Lying, reclining	Sunbathing, lying on a couch watching television
3	Sitting, very light exertion, light exertion (METs 2–4)	Eating, reading, sitting watching television, standing in line, strolling, car driving, office work
5	Moderate exertion (deep breathing, MET 5)	Normal walking, slow biking, hunting, fishing, slow dancing, downhill skiing, riding, curling
6	Vigorous exertion (with panting, overheating, MET 6)	Slow jogging, speed-walking, tennis, swimming, cross country skiing, sportive skiing, fast biking, ice hockey, overhead work, laying brick
7	Heavy exertion (with gasping, much sweating, MET 7)	Running, fast jogging, soccer, basketball, squash shovelling heavy snow, mixing cement
8	Extreme or peak exertion (MET 8)	Sprinting, jogging uphill, pushing or pulling with all one's might, unusually hard work

Of the total of 2089 cases of confirmed non-fatal AMI who were interviewed, there were 1613 cases who were able to and consented to participate in the diary interview. Of these, 1560 reported a discrete identifiable onset of AMI symptoms. One thousand three hundred and one provided information on the timing of exposures to activity levels and time spent outdoors for the day of AMI onset and the day before.

Temperature data

Hourly means of temperature in Augsburg were obtained through the Bavarian Air Monitoring Network. Daily midnight to midnight average temperatures were calculated.

Statistical analyses

For the analysis of triggering by physical exertion, we employed the case-crossover approach.[6] This approach contrasts exposure in the hazard period to exposure in control periods within the same individual, using standard methods for matched case–control analysis. Cases serve as their own controls, which eliminates potential confounding by time invariant factors. For each case, we compared exposure during the hazard period immediately preceding onset of infarction symptoms with the same subjects' exposure during two control periods 24 and 48 h earlier. We did not make any restrictions with regard to duration of exposure. By matching on time of day, we controlled for confounding by circadian pattern. Using conditional logistic regression models (in SAS 9.1.3 for Windows), we computed odds ratios as measure of relative risk (RR) of having an AMI during periods of exposure to moderate or strenuous exertion compared to the risk during periods of lower levels of exertion within an individual. We used a two-sided alpha level (type I error rate) of 5%.

We evaluated the empirical induction period of strenuous exertion estimating the association of strenuous exertion with AMI onset in the hour of symptom onset while controlling for strenuous exertion during each of the six 1 h periods before. The risk of AMI symptom onset was elevated for a 2 h period immediately following strenuous exertion (while being unchanged between 2 and 4 h, and unstable beyond 4 h). In a model that included indicators for 5, 6, and 7 and higher METs in the hour of AMI and the 6 previous hours, a similar pattern for the induction time of moderate and strenuous exertion was apparent. Therefore, in all subsequent analyses, we considered a 2 h hazard period immediately after exposure, and defined the maximum activity level within the 2 h case and control periods as exposures.

To examine the exposure–response relationship of exertion intensity and risk of onset of AMI symptoms, we introduced indicator variables for exertion classified as 5 METs (moderate), 6 METs (vigorous), and 7+ METs (heavy and peak), using METs 4 and lower as reference group in the model. Based on the results of this model, we included moderate (METs 5) and strenuous ($\geq$METs 6) exertion simultaneously into all subsequent models.

To evaluate whether the effect of moderate and strenuous exertion varied over chronic risk factors, we estimated their effects by age, sex, comorbidities, medication, smoking status, and frequency of physical exertion during control periods (as a measure of habitual exertion). We used the likelihood ratio test to assess potential interactions between these risk factors and exercise intensity.

We assessed possible interactions between moderate and strenuous exertion and being outdoors by including the three exposures and their products in the model. The control selection strategy inherent in the study design (control periods collected in the diary directly precede the case period) prevented us from estimating a main effect for temperature, because the control periods for an environmental exposure should be chosen farther from the case periods, and bidirectionally.[12] Therefore, in order to examine the interaction of ambient temperature and exertion, we stratified matched sets into cold (below first quartile), average (between first and third quartiles), and hot (above third quartile) temperature, excluding 12% of the matched sets that were discordant with respect to temperature category.

Sensitivity analyses

We had previously[13] shown that time spent in cars, on public transport, or riding bicycles was associated with onset of non-fatal AMI. Since biking is also a form of physical exertion, we included it as potential confounder in a multivariate model. We also included an indicator for 'getting out of bed', since it has been associated with the onset of AMI symptoms.[14]

We repeated the analyses excluding all 338 subjects who reported premonitory symptoms in the diary interview, because there may be a null-bias due to both confounding and control selection bias among patients with premonitory symptoms.

We assessed the sensitivity of the results to matching frequency by varying the number of control periods to a single control 24 h before symptom onset, to three control periods 24, 48, and 72 h before symptom onset, or included all potential control periods beginning 24 up to 72 h before symptom onset and controlled for time of day using indicator variables.

Because subjects may recall and report earlier exposures with less fidelity than more proximate exposures, we compared reported physical exertion in the first control period 24 h before the AMI symptom onset to the earlier control periods 48 and 72 h before symptom onset. We found that subjects were equally likely to report exposure in each of the control periods.

Results

Patients' characteristics and activities

Most of the 1301 cases were men, and the median age was 61 years (*Table 2*). They were slightly younger, more healthy, and more likely to be German, and reported more often typical AMI onset symptoms, than the 788 interviewed cases with insufficient diary data or no discrete identifiable onset. AMI symptom onset occurred more frequently in the morning hours than during other times of the day ($P < 0.001$).

Figure 1A shows the timing of episodes of strenuous exertion and physical exertion over the 4 days prior to AMI symptoms. Men were more likely than women to report strenuous exertion during the 4 days before AMI symptom onset (20.5 vs. 8.9%, $P < 0.001$).

Daily mean temperature ranged from −13 to 28°C, with the cooler quartile below 4°C and the warmer quartile being above 17°C.

Table 2 Individual characteristics and temperature exposure of acute myocardial infarction survivors recruited from the Augsburg Coronary Event Registry between February 1999 and December 2003

	N = 1301	%
Male	999	77
Female	302	23
Age (years)		
25–54	383	29
55–64	438	34
65–74	480	37
First myocardial infarction	1106	85
Disease history before onset		
Angina pectoris	290	22
Hypertension	933	72
Diabetes	341	26
None of the above	265	20
Medication before admission to hospital		
Anti-platelet medication	343	26
Statins	180	14
ACE blockers	209	16
ß-blockers	378	29
None of the above	689	53
Missing data	30	2
Smoking status		
Smoker	467	36
Non-smoker	442	34
Ex-smoker	394	30
Physical exertion during three control days (MET >=5)		
Data not sufficient	164	13
No exercise	419	32
0 h/day < exercise < 1 h/day	196	15
Exercise 1–3 h/day	320	25
Exercise >3 h/day	223	17
Daily mean temperature on case and control day		
Below 4°C	287	22
4–17°C	586	45
Above 17°C	272	21
Case and control periods not in the same quantile	158	12

Association of exertion and onset of acute myocardial infarction

Figure 1B shows that patients reported that they engaged in moderate or strenuous exertion more frequently in the 2 h before AMI symptom onset than at other times. Relative to periods of rest or low levels of exertion, the risk of AMI onset was significantly elevated for moderate, strenuous, and higher levels of exertion

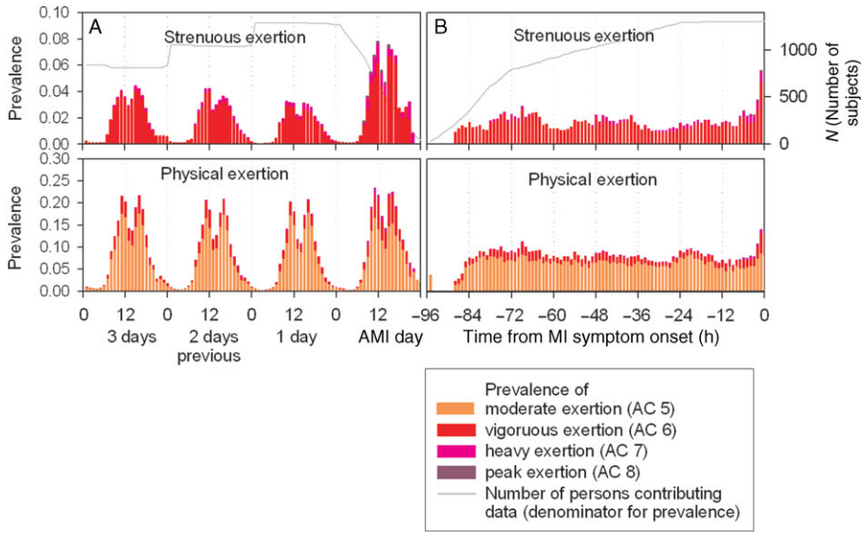

Figure 1 Distribution of exertion over time, shown as mean by hour of day (*A*) and hours before AMI symptom onset (*B*), respectively.

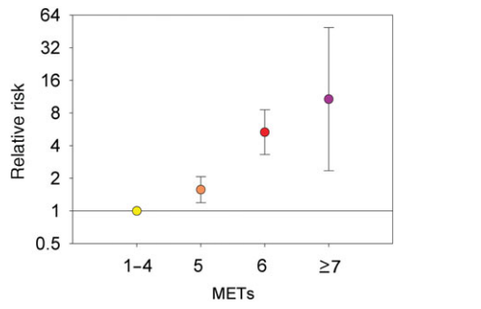

Figure 2 Exposure–response relationship of the association of exertion intensity and AMI symptom onset within 2 h afterward.

(*Figure 2*), and the RR was observed to increase progressively with increasing exertion level ($P_{trend} < 0.001$). The risk of AMI symptom onset was about five times higher either during or within 2 h of engaging in strenuous exertion than during times of very light or no exertion [RR 5.7, 95% confidence interval (CI) 3.6–9.0] (*Table 3*). Even moderate exertion was associated with a higher risk compared to very light or no exertion (RR 1.6, 95% CI 1.2–2.1).

Table 3 shows that the effect of moderate and strenuous exertion on AMI onset was relatively constant across history of smoking, disease status, and reported use of common cardiovascular medications. The effect of strenuous exertion but not of moderate exertion was stronger among older compared with younger subjects. Subjects with hypertension had a two-fold increase in AMI onset risk associated with moderate exertion, whereas in subjects without hypertension the risk was not elevated. The effect of an isolated bout of physical exertion on AMI onset varied greatly by the frequency of physical exertion in the control days. The 223 subjects who reported an average of more than 3 h recordings of physical exertion per control day did not appear to have an increased risk of AMI associated with moderate or strenuous exertion. On the other hand, the 196 subjects who reported infrequent or no exertion had 26-fold increase in AMI onset risk associated with strenuous exertion and a 3.5-fold increase associated with moderate exertion.

The RR of AMI symptom onset was significantly higher when strenuous exertion was performed outdoors compared to when the same level of exertion was performed indoors ($P = 0.008$), while there was no interaction with moderate exertion (*Figure 3*).

The effect of physical exertion was not significantly altered by outdoor temperature levels (*Table 3*). Analyses of the interaction with times spent outdoors within the temperature strata showed no alteration in estimate for the high and average temperature groups, and could not be performed for the low temperature group because of low numbers (results not shown).

Sensitivity analyses

The effect of moderate and strenuous exertion remained unchanged when we included biking, and getting out of bed, as potential confounders. The estimate for strenuous exertion was somewhat higher when we excluded patients with premonitory symptoms (RR 7.6, 95% CI 4.4–13), while the estimate remained unchanged for moderate exertion (RR 1.6, 95% CI 1.2–2.2). Our results were not materially altered in analyses in which we used one or three instead of two control periods. When using all available control periods, the estimated RR for strenuous exertion was higher (RR 8.35, 95% CI 6.02, 11.60) while it remained unchanged for moderate exertion.

Table 3 Frequency of exposure to moderate and strenuous exertion in case periods, and relative risks of acute myocardial infarction symptom onset within 2 h after moderate or strenuous exertion compared to very light or no exertion within an individual, according to patients' characteristics and temperature levels

	Physical exertion (MET 5)		Strenuous exertion (MET 6+)		*P*-value for interaction[a]
	$N_{exp.cases}$	RR (95% CI)	$N_{exp.cases}$	RR (95% CI)	
All	153	1.5 (1.2–2.1)	90	5.7 (3.6–9.0)	
Sex					
Female	34	1.3 (0.7–2.3)	7	11 (1.3–96)	
Male	119	1.7 (1.2–2.3)	83	5.6 (3.5–8.9)	0.563
Age (years)					
35–54	41	1.4 (0.8–2.4)	36	3.0 (1.7–5.4)	
55–64	55	2.2 (1.3–3.5)	32	11 (4.0–28)	
65–74	57	1.3 (0.8–2.1)	22	21 (4.9–90)	0.014
Disease before onset					
Angina pectoris	33	1.8 (1.0–3.2)	6	–	
No angina pectoris	120	1.5 (1.1–2.1)	84	5.3 (3.3–8.4)	0.168
Hypertension	119	1.9 (1.4–2.7)	47	4.6 (2.5–8.4)	
No hypertension	34	0.9 (0.5–1.6)	43	6.9 (3.4–14)	0.034
Diabetes	31	1.7 (0.9–2.9)	19	7.6 (2.5–23.1)	
No diabetes	122	1.5 (1.1–2.1)	71	5.4 (3.3–8.9)	0.849
None of the above	25	0.9 (0.5–1.7)	35	6.2 (2.9–13)	
At least one of the above	128	1.8 (1.3–2.4)	55	5.3 (3.0–9.3)	0.147
Medication					
Anti-platelet medication	33	1.6 (0.9–2.7)	10	16 (2.0–134)	
No anti-platelet medication	117	1.6 (1.2–2.3)	78	5.3 (3.3–8.4)	0.484
Statins	23	2.5 (1.1–5.3)	11	24 (3.0–196)	
No statins	127	1.5 (1.1–2.0)	77	5.0 (3.2–8.0)	0.162
ACE blockers	22	2.1 (1.0–4.2)	4	3.5 (0.6–20.8)	
No ACE blockers	128	1.5 (1.1–2.1)	84	5.8 (3.6–9.3)	0.60
ß-blockers	47	2.1 (1.2–3.5)	7	2.6 (0.7–9.6)	
No ß-blockers	103	1.5 (1.0–2.0)	81	6.1 (3.8–10)	0.228
None of the above	85	1.5 (1.1–2.3)	66	5.6 (3.3–9.4)	
At least one of the above	65	1.7 (1.1–2.6)	22	5.9 (2.3–15.1)	0.955
Smoking status					
Smoker	55	1.8 (1.1–3.0)	35	3.6 (1.9–6.8)	
Ex-smoker	42	1.8 (1.1–3.0)	34	12 (4.7–32)	
Never smoker	56	1.3 (0.8–2.0)	21	6.0 (2.3–15)	0.181
Physical exertion during three control days (MET >=5)					
Exercise <1 h/day	56	3.5 (2.0–5.9)	31	26 (6.2–113)	
Exercise 1–3 h/day	54	1.0 (0.6–1.6)	36	10 (4.3–25)	
Exercise >3 h/day	43	0.7 (0.4–1.2)	23	1.2 (0.6–2.4)	<0.001
Being concurrently outdoors					
No	41	1.4 (0.8–2.4)	27	2.3 (1.2–4.4)	
Yes	112	1.3 (0.7–2.3)	63	10 (4.3–24)	0.009
Daily mean temperature					
Case and control below 4°C	38	1.7 (0.9–3.4)	20	13 (3.1–59)	
4–17°C	68	1.4 (1.0–2.1)	36	4.4 (2.3–8.6)	
Above 17°C	21	1.5 (0.8–2.9)	23	5.5 (2.0–15.2)	0.694

*Testing for an interaction between exercise intensity and categories of patients' characteristics with the likelihood ratio test.

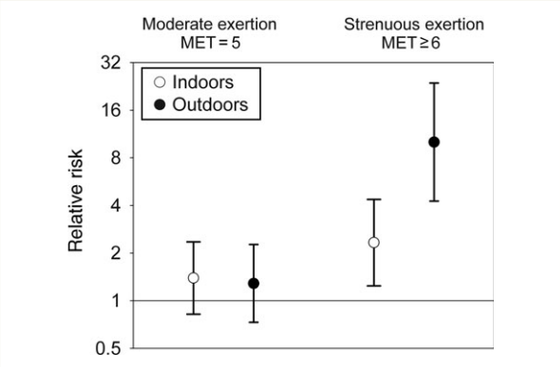

Figure 3 Influence of time spent outdoors on the association of moderate and strenuous exertion with AMI symptom onset within 2 h afterward.

Discussion

In this study, we estimated that an individual's risk of AMI onset was transiently increased 5.7-fold (95% CI 3.6–9.0) within 2 h of an episode of strenuous exertion and 1.6-fold (95% CI 1.2–2.1) within 2 h of moderate exertion compared to periods of rest or lower levels of exertion. Regular physical exertion was protective against the risk associated with moderate and strenuous exertion episodes. Interestingly, strenuous activity outside was associated with a four-fold larger RR of AMI symptom onset than exertion performed indoors. This was not observed for moderate exertion and was not explained by outdoor temperature. The association with strenuous exertion was stronger among older compared with younger subjects. We observed no clear evidence for effect modification by medication, comorbidities, or smoking habits.

Our results are consistent with prior studies that have demonstrated an increased risk of non-fatal AMI in association with isolated episodes of vigorous exertion.[3–5,7,15] Proposed mechanisms include abrupt changes in heart rate and blood pressure with subsequent haemodynamic stress and disruption of vulnerable atherosclerotic plaques and thrombotic occlusion of a coronary vessel;[16] platelet activation resulting in enhanced thrombogenicity;[17,18] and increased oxygen demand.[16]

Furthermore, our results provide further evidence that the effect of vigorous exertion on transient AMI risk is much greater among sedentary individuals than those who regularly engage in vigorous exertion.[3–5,7,15] This protective effect of regular exercise might be due to lower heart rate and peak systolic pressure at a given workload,[19] beneficial effects on lipid profile, platelet aggregability, fibrinolytic potential and arteriosclerosis,[19] and mitigated inflammation[20] and thereby decreased plaque vulnerability.

While previous studies have found that the effect of vigorous exertion as a trigger of AMI was limited to the time of exertion and extended up to 30–60 min after the activity,[3,4,21] in our data the effect persisted up to 2 h following the episode of exertion. This was not explained by differences of analytic technique, since the 2 h effect period was confirmed when using the same approach as the previous authors. The difference, however, may be either due to a real difference in the duration of the effect period in our population or due to misclassification of the timing of exposure. In contrast to the study of Mittleman *et al.*,[3] in this study, the timing of activity was ascertained based on hourly bins of 'clock-time' rather than relative to the timing of MI symptom onset. Thus, in the present study, exposures that occurred concurrently with MI onset were recorded in the same hour, whereas approximately half of the exposures that occurred earlier, but still within 1 h of MI symptoms would have been recorded for the preceding hour of 'clock time'.

The transient risk of AMI symptom onset within 2 h appeared to increase significantly with increasing intensity of physical exertion. Moderate exertion was associated with AMI symptom onset, particularly in persons with hypertension or who reported infrequent physical activity. This result emphasizes the importance of increasing endurance very carefully when introducing a cardio protective training program to sedentary or hypertensive patients.

The results of some of the subgroup analyses were not consistent and may reflect the smaller sample sizes within the subgroups. Evaluation of similar subgroup analyses in other studies will be required to clarify these results.

The RR of AMI symptom onset was significantly higher when strenuous exertion (but not moderate exertion) was performed outdoors compared to when it was performed indoors. This may be explained by outdoor environmental conditions which may play an important role as additional stressors, such as outside temperature. Although the results of prior studies are not entirely consistent, the risk of myocardial infarction and sudden cardiac death has been reported to be higher on cold days,[22–24] and extremely hot days[25] compared to average temperatures. Underlying mechanisms for the triggering of AMI by low temperature might be (i) an increase in the sympathetic tone with elevated heart rate and blood pressure,[26,27] (ii) increased blood viscosity due to an increase in serum fibrinogen levels, and (iii) increased thrombocyte aggregability.[28–30] Temperature might, therefore, release the same internal triggers of AMI as strenuous activity and act as an effect modifier.

In the present study, the temperature stratified analyses did not show any difference in the risk of AMI symptom onset after strenuous exertion, nor did it explain the interaction of strenuous exertion and time spent outdoors. However, there were not enough extremely hot or cold days to evaluate whether extremes of temperature alter the risk of AMI associated with exertion. We were not able to assess the direct effect of temperature on AMI simultaneously with the individual exposure effect, because the day to day correlation and trend in temperature would lead to biased estimates of association in our case-crossover approach.[12]

Alternatively, a higher risk of strenuous exertion outdoors may be explained by the specific characteristics of the activities that were performed outdoors and that differ from exertion occurring indoors. Descriptive statistics showed that the outdoor strenuous activities in the control periods were mostly leisure time activities, whereas in the case periods they were more likely to be work related. The data available are not sufficiently detailed to further assess this hypothesis.

Strengths and limitations

While the case-crossover method controls for fixed exposures by design, confounding by time-varying factors may still have occurred. We controlled for coinciding circadian patterns of exertion and MI onset by matching on time of day in our main analyses. Furthermore, in a sensitivity analysis, we found that the RR of MI onset was similar in the morning and at other times of the day. This implies that the absolute increment in risk associated with exertion may be somewhat higher in the morning than at other times of the day. When we additionally adjusted for measured potential confounders, attenuation of the main effects did not occur.

Information bias may have occurred with respect to diary data, since individuals' activities were assessed retrospectively in a diary interview. Strenuous exertion is a rare exposure and thus relatively easy to remember, but memory of the case day may be clearer than that of previous days. However, the observed frequency of activities during the 4 days preceding the AMI suggested consistent reporting over the time. If recall bias were strong, these daily frequencies would have decreased with increasing time to event. Earlier work addressing the role of exertion in triggering AMIs included a population-based control group matched for age, sex, and precinct.[5] The authors found comparable results for strenuous exertion when using the case-crossover approach and the case–control approach. Overall, recall bias might have lead to an overestimation of the results of this study but cannot explain the strong associations.

Conclusion

This study confirms previous results and shows a graded exposure–response relationship between physical exertion intensity and triggering of AMI onset. These findings may have implications for behavioural guidance of people at risk of AMI.

Conflict of interest: none declared.

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The above article uses a new reference style being piloted by the EHJ that shall soon be used for all articles.

CLINICAL VIGNETTE

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Aortic valvular endocarditis with mobile vegetations and intracoronary embolism: demonstration by cardiac multislice computed tomography

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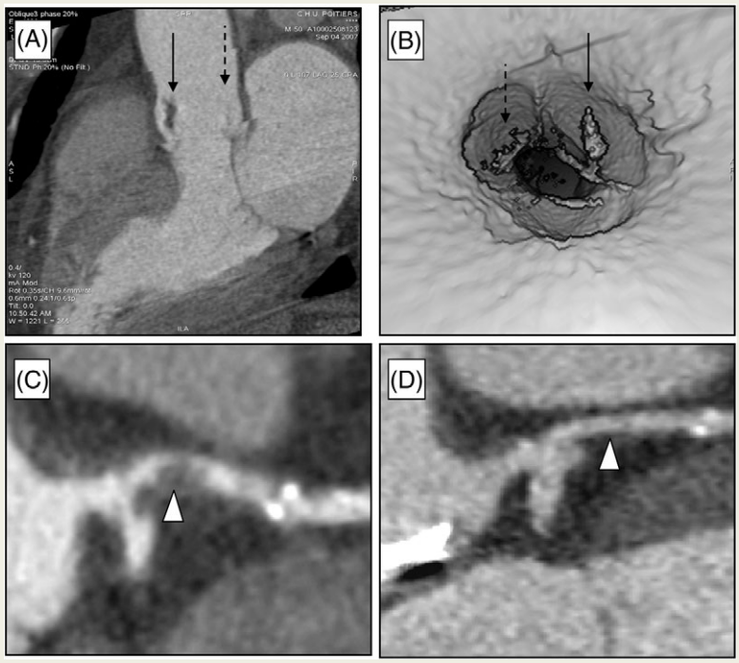
A 51-year-old patient, treated for dental abscess 3 months ago, was admitted for an acute coronary syndrome and fever. He had a fever of 38.5°C and a diastolic murmur. Blood cultures were positive for *Streptococcus anginosus*. Echocardiography confirmed an aortic valvular endocarditis with severe aortic regurgitation and highly mobile vegetations. Because of the vegetations, non-invasive coronary angiography using ECG-gated 64-multislice computed tomography (MSCT) was performed instead of conventional angiography. MSCT demonstrated two mobile vegetations with a length of 5 mm on the left aortic cusp and 12 mm on the right aortic cusp with systolic protrusion in the aortic root near the coronary artery ostia (Panels A and B). MSCT revealed also significant lesions on the three major coronary arteries and an intraluminal non-calcified soft tissue in the proximal left anterior descending artery (LAD) (Panel C). The patient was referred for surgery without complementary invasive coronary angiography. A triple coronary artery bypass grafting and an aortic replacement with a Hall Kaster prosthesis were performed. Vegetations were confirmed by the surgeon. Five days later, a second MSCT was performed because of increased troponin levels. The three patent coronary artery bypass graft were visualized. Contrary to the previous MSCT, the proximal LAD was founded nearly normal without unusual intraluminal soft tissue visualized previously (Panel D). We thought that intracoronary septic embolism was the first possible mechanism in view of the association of mobile vegetations near the coronary artery ostia, and intracoronary soft tissue with similar attenuation value of 47 ± 35 HU which disappear in a few days. Several papers have reported the usefulness of MSCT in case of aortic endocarditis, but MSCT evidence of coronary embolism was not reported before.



Panels A and B. ECG-gated MSCT after intravenous contrast injection reveal a mobile vegetation on the left coronary cusp (broken arrow) and a larger vegetation on the right coronary cusp (arrow). Coronal multiplanar reconstruction in systole (Panel A) with corresponding virtual angioscopy (Panel B).

Panels C and D. ECG-gated MSCT before surgery. Curved multiplanar reconstruction of the proximal left anterior descending (LAD) artery. Arrow head: unusual non-calcified tissue in the lumen of the proximal LAD (Panel C), but not present 6 days after surgery (Panel D), corresponding to the intracoronary embolism.