Cardiovascular effects of strenuous exercise in adult recreational hockey: the Hockey Heart Study

Research

Recherche

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Abstract

Background: More than 500 000 men play "gentlemen's" recreational hockey in Canada, but the safety of this exercise has not been studied. Exercising at extremes of intensity has been associated with an increased risk of cardiac events. Our objective was therefore to determine baseline cardiac risk factors among adult recreational hockey players and to measure any cardiac abnormalities they experienced while playing hockey.

Methods: We assessed baseline cardiac risk factors in 113 male volunteers recruited from a recreational hockey league. Each subject underwent holter electrocardiographic monitoring before, during and after at least one hockey game (maximum of 115 holter data sets). We used the data to assess exercise heart rate, arrhythmias and ST-segment changes and for correlation with symptoms and other predictors of fitness.

Results: For all participants, maximum heart rate (HR_{max}) (mean 184 [standard deviation 11] beats/min) was greater than target exercise heart rate (calculated as 55% to 85% of age-predicted HR_{max}), and in 87 (75.6%) of the 115 holter data sets, the heart rate exceeded the age-predicted HR_{max}. The mean period for which heart rate exceeded 85% of the age-predicted HR_{max} was 30 (SD 13) min. For 80 (70.1%) of 114 data sets, heart rate recovery was poor. Nonsustained ventricular tachycardia was seen in data from 2 holter monitoring sessions and ST-segment depression in data from 15 sessions.

Interpretation: The physical activity pattern that occurred during recreational hockey caused cardiac responses that might be dangerous to players' health. More specifically, the players exceeded target and maximum heart rates, had poor heart rate recovery after exercise, and had episodes of nonsustained ventricular tachycardia and ST-segment depression of uncertain clinical significance.

It is estimated that more than 500 000 men and over 70 000 women regularly play recreational hockey in Canada.[1,2] Our experience in treating men with few cardiac risk factors who experienced myocardial infarction during or shortly after participating in recreational hockey raised concerns about the activity. To our knowledge, risk factors among recreational hockey players have not been studied, and cardiac problems associated with this activity have not been investigated. Myocardial infarction and sudden death during intense physical exercise have been reported,[3–6] whereas moderate exercise is recognized as beneficial in reducing the risk of heart disease and is therefore recommended.[7–10] We questioned whether participants in recreational hockey are at higher risk or whether there is a risk associated with the activity itself.

The benefits of exercise have been well studied. Mild to moderate exercise at an intensity level of as low as 40% to 50% of maximum oxygen uptake reserve or 55% to 65% of age-predicted maximum heart rate (HR_{max}) is associated with better fitness and fewer cardiac events.[7,8,11–13] Maximum oxygen uptake can be measured in specialized cardiac rehabilitation clinics but is either not available or not widely accessible to the general public. Predicted HR_{max} can be easily calculated from age (HR_{max} = 220 – age in years).[14] Concerns have been raised about risk for cardiac events, as well as musculoskeletal injuries, with higher-intensity exercise.[7]

We hypothesized that some participants in adult recreational hockey may not have adequate conditioning for intense competitive exertion, and we therefore sought to analyze their cardiac risk factors as well as measuring heart rates, arrhythmias and unrecognized ischemia.

Methods

We conducted a descriptive, cross-sectional study of male participants playing recreational hockey in Sydney, NS. Ethical approval was obtained from Dalhousie University and the Cape Breton Regional Hospital. Volunteers were recruited by word of mouth and flyers and through the assistance of rink managers. Inclusion criteria were age greater than 35 years and participation in an adult recreational hockey league. Exclusion criteria were known heart disease such as angina, congestive heart failure, prior myocardial infarction and known arrhythmia. All participants in the study signed a consent form and answered a questionnaire about general health and cardiac risk factors. A postgame questionnaire was used to assess any cardiac symptoms occurring during the game or in the cool-down period, such as chest pain, shortness of breath, syncope and presyncope, or palpitations.

Two participants per hockey game were outfitted with a Marquette holter electrocardiographic monitor (General Electric, Milwaukee) placed on the hip under the person's hockey pads. Leads were attached with Medi-Trace (Kendall-LTP, Chicopee, Mass.) or BioTac (Kendall-LTP) connectors in a standard 7-lead

Table 1: Baseline characteristics, cardiac risk factors and results of holter monitoring for 113 male recreational hockey players

Characteristic	No. of subjects or data sets*	Mean (and SD)	No. (and %) of subjects
Baseline characteristics			
Age, yr	113	42.7 (6.9)	
Blood pressure, mm Hg			
Resting systolic	106	116 (13)	
Resting diastolic	106	72 (9)	
Pregame systolic	111	137 (18)	
Pregame diastolic	111	83 (9)	
Heart rate, beats/min			
Resting	105	59 (9)	
Pregame	108	73 (9)	
Body characteristics			
Waist to hip ratio	113	0.98 (0.06)	
BMI (kg/m^2)	106	28.4 (4.4)	
Cardiac risk factors			
Smoking > 1 cigarette/day	113		15 (13.3)
Family history of ischemic heart disease	113		41 (36.3)
Diabetes (known)	113		3 (2.6)
Hypertension (known or receiving medication)	113		6 (5.3)
Elevated total cholesterol (> 5.2 mmol/L)	106		56 (52.8)
Alcohol intake > 14 standard drinks/week†	108		17 (15.7)
Exercise ≥ 3 times per week‡	113		68 (60.2)
Heart rate data from holter monitoring§			
Observed HR_{max}, beats/min	115	184 (11)	
Observed HR_{max} as % of age-predicted maximum heart rate	115	104 (6)	
No. of beats > THR¶	115	457 (329)	
Time spent > THR, min	115	30 (13)	
% of time spent > THR	115	34 (15)	
Reduction in heart rate at 1 min after cessation of exercise, beats/min**	114	4.5 (18)	

Note: SD = standard deviation, BMI = body mass index, HR_{max} = maximum heart rate, THR = target heart rate (85% of age-predicted HR_{max}).
*There were 113 participants in the study. Not all volunteers completed the general questionnaire or underwent baseline laboratory testing. For 4 of the 113 participants, there was concern about tape malfunction during the holter monitoring session, so monitoring was repeated during a second game, for a total of 117 data sets. Of these, 2 were spoiled, so the maximum number of holter data sets was 115.
†A standard drink is 1 oz. (30 mL) spirits, 1 glass wine or 12 oz. (360 mL) beer (according to currently accepted recommendations.
‡Excluding hockey.
§Mean time of holter monitoring was 90 (SD 14) minutes.
¶Number of beats by which HR_{max} exceeded THR.
**One player removed his monitor during the game, so post-exercise heart rate was available for only 114 data sets.

fashion.[15] Monitoring was conducted throughout the hockey game (60 to 90 minutes) and for a 20-minute cool-down period after the game. Body mass index (BMI) and waist to hip ratio,[16] a 12-lead electrocardiographic tracing, and fasting sugar and cholesterol levels were also obtained.

Data from leads V1 and V5 of the 7-lead holter monitors were analyzed by Schiller Scanner software (model NP147, version 3.1, Century Advanced, Ottawa, Ont.) and reviewed by a cardiologist (P.M.) to determine observed HR_{max}, number of beats by which observed HR_{max} exceeded the target heart rate (THR), the period for which heart rate exceeded THR, heart rate 1 minute after exercise, and presence of ectopic beats, sustained arrhythmias and ST-segment depression. Age-predicted HR_{max} was calculated as 220 – age,[14] and THR was defined as 55% to 85% HR_{max}.[17,18]

ST segment changes were classified as either suspicious (flat ST segment or 1- to 2-mm ST-segment depression in one lead) or highly suspicious (ST-segment depression of more than 2 mm in one lead). ST-segment depression of less than 1 mm or upsloping ST segment was considered normal. Participants with suspicious ST-segment changes were investigated with standard exercise stress testing according to the Bruce protocol.[12] If the findings were suspicious, the participant underwent further evaluation with a 2-day nuclear sestamibi stress test.

Means and standard deviations were calculated for baseline parameters and cardiac risk factors.

Results

Approximately 2500 men participated in recreational hockey at the 5 rinks where the study was conducted. We approached a total of 161 players. Four teams of 10 players each, as well as 8 individual players, refused to participate; none of the men were excluded because of known heart disease. A total of 113 players (70.2%) were enrolled between January and March 2000. Mean age was 42.7 (range 24–62) years. Baseline characteristics (Table 1) were within normal ranges, and of the 106 participants whose BMI was determined, 58 (54.7%) had a BMI above 27. Cardiac risk factors (Table 1) and cholesterol characteristics (Table 2) were below general population levels.[19]

Symptoms reported while playing hockey included one report each of shortness of breath, palpitations, and chest pain or heaviness.

A total of 115 sets of monitoring data were available for analysis (see Table 1). Observed HR_{max} exceeded THR in all participants (Fig. 1) for a mean of 30 minutes. Observed HR_{max} exceeded age-predicted HR_{max} in 87 (75.6%) of the 115 data sets.

In 80 (70.1%) of 114 data sets, heart rate had slowed by less than 12 beats/min at 1 minute after cessation of exercise.

Holter monitoring revealed isolated supraventricular and ventricular ectopic beats, as well as short runs of supraventricular tachycardia. Atrial fibrillation occurred in one subject and nonsustained ventricular tachycardia (runs

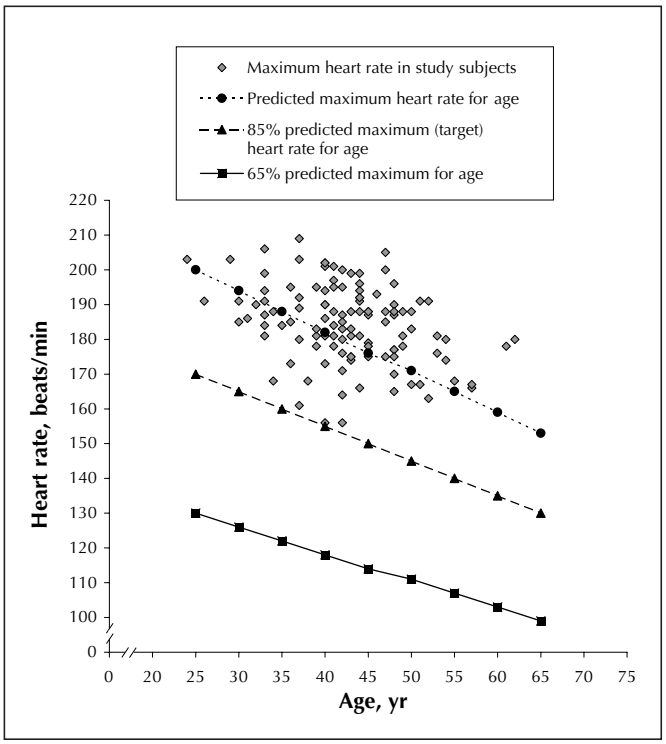

Fig. 1: Maximum heart rates compared with predicted maximum and target heart rates for age. Age-predicted maximum heart rates (as well as the 85% and 65% variations of these values) represent a replot of data in Lester and colleagues;[17] 85% of the predicted maximum represents the target heart rate.

Table 2: Cholesterol characteristics

Type of cholesterol	No. of subjects	Mean level (and SD)	Degree of dyslipidemia; no. (and %) of subjects	
			Moderate*	Severe†
Total	106	5.24 (0.97)	40 (37.7)	16 (15.1)
Triglycerides	106	1.53 (1.03)	12 (11.3)	7 (6.6)
LDL	103	3.13 (0.73)	11 (10.7)	2 (1.9)
HDL	106	1.38 (0.35)	13 (12.3)	13 (12.3)

Note: LDL = low-density lipoprotein, HDL = high-density lipoprotein.
*Moderate dyslipidemia was classified as follows: total cholesterol 5.2–6.1 mmol/L, triglycerides 2.2–3.1 mmol/L, LDL cholesterol 4.0–5.0 mmol/L and HDL cholesterol < 1.0 mmol/L.
†Severe dyslipidemia was classified as follows: total cholesterol ≥ 6.2 mmol/L, triglycerides ≥ 3.2 mmol/L, LDL cholesterol ≥ 5.1 mmol/L and HDL cholesterol < 1.0 mmol/L.

of more than 3 beats lasting less than 30 seconds) in 2 patients. Of the 2 patients with nonsustained ventricular tachycardia, one had a significant amount of arrhythmia, especially in the cool-down period. The results of subsequent sestamibi stress testing (during which he attained a heart rate of 170 beats/min with no ventricular arrhythmia) and cardiac ultrasonography were normal. The second patient had many fewer episodes of ventricular tachycardia (short runs). Exercise stress testing yielded negative results (13 metabolic equivalents [METs; 1 MET = 3.5 mL O_2/kg/min]) with no ectopy, and sestamibi study yielded positive results; however, the latter was considered a false positive because of normal wall motion.

Flat or downsloping ST-segment depression was identified. Exercise stress tests were performed for 17 other subjects, of whom 15 exhibited ST-segment depression on holter monitoring; in addition, 1 had atypical chest pain, and 1 had supraventricular arrhythmia. For 10 of the subjects with ST-segment depression, the results of exercise stress testing were normal. For the other 5, the results of exercise stress testing were abnormal, but the results of subsequent sestamibi stress testing were normal. These subjects had no significant cardiac disease.

There was a trend toward higher resting heart rates for older participants compared with younger participants (95% confidence interval [CI] –6.6 to 0.2, $p = 0.06$). Older participants had significantly lower maximum heart rates (95% CI 0.1 to 8.3, $p < 0.05$) but a greater number of beats (95% CI –249 to –8, $p < 0.05$) and they exercised a greater percentage of time above 85% of their maximum heart rate (95% CI –11 to –0.3, $p < 0.05$). Heavier hockey players tended to have fewer heart beats above the target (95% CI –9 to –233, $p = 0.07$), higher cholesterol levels (95% CI –0.8 to –0.4, $p = 0.03$) and higher triglyceride levels (95% CI –1.1 to –0.3, $p < 0.001$). Players who exercised 3 or more times per week had a slower heart rate recovery 1 minute after exercise (95% CI –13.8 to –0.2, $p < 0.05$).

Interpretation

These recreational hockey players had few risk factors associated with ischemic heart disease and sudden death. Observed HR_{max} was consistently above 85% to 100% of age-predicted HR_{max}; this finding suggests that the intensity of the exercise would increase participants' risk.

The American College of Sports Medicine[7] recommends an intensity of training from 55% or 65% to 90% of age-predicted HR_{max}. The college further cautions that "Higher-intensity exercise is associated with greater cardiovascular risk and orthopaedic injury."[7] Although the benefits of exercise are widely recognized,[8–10] there have also been reports of an increase in frequency of cardiac events and sudden death triggered by vigorous exercise.[3,4,6] Regular exercise seems to diminish this risk.[6,20]

It is important to emphasize that no adverse events occurred during our study. Franklin and colleagues[21] reported that moderate to vigorous exercise was associated with a mortality rate of 1 per 50 000 among people who exercised infrequently, a rate above that in the general population.

Willich and collaborators,[4] in a retrospective analysis, found that 7.1% of patients who presented with myocardial infarction but only 3.9% of the control group were engaged in physical exertion at the time of the myocardial infarction (relative risk 2.1). Those who exercised fewer than 4 times per week had a relative risk of 6.9, whereas the relative risk was just 1.3 for those who exercised 4 times or more per week ($p < 0.01$). Mittleman and associates[3] found that 4.4% of patients who had experienced myocardial infarction had been involved in heavy exertion. The relative risk (compared with little or no exertion) was 5.9. Again, regular exercise was shown to greatly attenuate the risk.

The subset of 19 participants (16.8%) who underwent exercise stress testing in this study all reached or exceeded 85% of age-predicted HR_{max} at a workload of 7 to 17.2 METs (mean > 10 METs). Although the sample was small, this finding indicates the relatively high intensity of recreational hockey. In addition, there is generally little or no warm-up before recreational games, nor are substitutes, interruptions or rest periods available to allow players to cool down.

The limitations of this study include the small sample size and a lack of a control group. Some subjective features were observed that were not prespecified or objectively measured, such as limited warm-up and rest periods. Finally, sestamibi stress testing is not the gold standard for coronary artery disease, and subclinical obstructions may have been missed. Cardiac catheterization was not available in our community, and in any case the potential risks associated with this procedure would have made it ethically impractical to use for asymptomatic volunteers.

It is important to maintain a balanced perspective on risks. Although the relative risk may be significantly increased during strenuous exercise, the absolute risk of sudden death is just 1 per 1.5 million episodes of vigorous exercise.[6] If 500 000 Canadian men each play hockey 30 times a year, this would account for 10 deaths.

In conclusion, the participants in our study, men playing recreational hockey, were generally healthy. No adverse events were observed, but all participants experienced high heart rates for prolonged periods of time. Arrhythmias and ST-segment changes of uncertain clinical significance were also noted.

Competing interests: None declared.

Contributors: Dr. Atwal was involved in study design and background research, recruitment of subjects, attachment of holter monitors and collection of data, as well as data analysis and writing of the manuscript. Dr. Porter was involved in study design, recruitment of subjects and manuscript preparation. Dr. MacDonald was involved in study design and background research, data analysis (including data from holter monitors, exercise stress testing, sestamibi stress testing and electrocardiography) and manuscript preparation.

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References

1. The Canadian ice skating and hockey equipment sector. Ottawa: Industry Canada; 2000. Available: strategis.ic.gc.ca/SSG/sg01066e.html (accessed 2002 Jan 4).
2. Canadian Adult Recreational Hockey Association. Corporate profile. Ottawa: The Association; [no date]. Available: www.carha.ca/sponsorship/profile.htm (accessed 2001 Dec 21).
3. Mittleman MA, Maclure M, Tofler GH, Sherwood JB, Goldberg RJ, Muller JE. Triggering of acute myocardial infarction by heavy physical exertion — protection against triggering by regular exertion. *N Engl J Med* 1993;329:1677-83.
4. Willich SN, Lewis M, Lowel H, Arntz HR, Schubert F, Schroder R. Physical exertion as a trigger of acute myocardial infarction. *N Engl J Med* 1993;329: 1684-90.
5. Jette M, Blumchen G, Treichel P, Landry F. Electrocardiographic responses to jogging in middle aged and older men and women. *Clin Cardiol* 1993;16:231-4.
6. Albert CM, Mittleman MA, Chae CU, Lee MI, Hennekens CH, Manson JE. Triggering of sudden death from cardiac causes by vigorous exertion. *N Engl J Med* 2000;343:1355-61.
7. Pollock ML, Gaesser GA, Butcher JD, Després JP, Dishman RK, Franklin BA, et al. The recommended quantity and quality of exercise for cardiorespiratory and muscular fitness, and flexibility in healthy adults. *Med Sci Sports Exerc* 1998;30(6):975-91.
8. Fletcher GF, Balady G, Blair SN, Blumenthal J, Caspersen C, Chaitman B, et al. Statement on exercise: benefits and recommendations for physical activity programs for all Americans. A statement for health professionals by the Committee on Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology, American Heart Association. *Circulation* 1996;94:857-62.
9. Pate RR, Pratt M, Blair SN. Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA* 1995;273:402-7.
10. NIH Consensus Development Panel on Physical Activity and Cardiovascular Health. Physical activity and cardiovascular health. *JAMA* 1996;276:241-6.
11. Fletcher GF, Schlant RC. The exercise test. In: Schlant RC, Alexander RW, editors. *Hurst's the heart*. New York: McGraw-Hill Inc.; 1994. p. 423-40.
12. Gibbons RJ, Balady GJ, Beasley JW, Bricker JT, Duvernoy WF, Froelicher VF, et al. ACC/AHA guidelines for exercise testing. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). *J Am Coll Cardiol* 1997;30:260-315.
13. Fletcher GF. How to implement physical activity in primary and secondary prevention. A statement for healthcare professionals from the task force on risk reduction, American Heart Association. *Circulation* 1997;96:355-7.
14. Tanaka H, Monahan KD, Seals DR. Age-predicted maximal heart rate revisited. *J Am Coll Cardiol* 2001;37:153-6.
15. Crawford MH, Bernstein SJ, Deedwania PC, DiMarco JP, Ferrick KJ, Garson A Jr, et al. ACC/AHA guidelines for ambulatory electrocardiography. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the Guidelines for Ambulatory Electrocardiography). *J Am Coll Cardiol* 1999;34:912-48.
16. Molarius A, Sans S, Kuulasmaa K, for the WHO MONICA Project. Quality assessment of data on waist and hip circumferences in the WHO MONICA Project. In: *WHO MONICA Project. MONICA Manual*. Quality assessment of survey procedures 1999. Geneva: World Health Organization; 1999. Available: www.ktl.fi/publications/monica/waisthip/waisthipqa.htm (accessed 2001 Dec 17).
17. Lester FM, Sheffield LT, Reeves TJ. Electrocardiographic changes in clinically normal older men following near maximal and maximal exercise. *Circulation* 1967;36:5-14.
18. Lester M, Sheffield LT, Trammell P, Reeves TJ. The effect of age and athletic training on the maximal heart rate during muscular exercise. *Am Heart J* 1968;76(3):370-6.
19. The changing face of heart disease and stroke in Canada 2000. Ottawa: Statistics Canada; 1999.
20. Schnohr P, Parner J, Lange P. Mortality in joggers: population based study of 4658 men. *BMJ* 2000;321:602-3.
21. Franklin BA, Conviser JM, Stewart B, Lasch J, Timmis GC. Sporadic exercise: a trigger for acute cardiovascular events? [abstract]. *Circulation* 2000; 102(Suppl II):612.

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