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# Cardiorespiratory Screening in Elite Endurance Sports Athletes: The Quebec Study

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## Abstract

**Background:** Cardiorespiratory disorders are common in athletes. However, these conditions are often underdiagnosed, which potentially results in impaired performance and increased health risks. The aim of this study was to evaluate, in a research setting, the prevalence of cardiorespiratory disorders in athletes in order to determine the potential value of a screening program. **Methods:** One hundred thirty-three athletes were studied. Each subject underwent a physical examination. A eucapnic voluntary hyperventilation (EVH) test and a methacholine inhalation test were performed to confirm the diagnosis of asthma. A cardiovascular evaluation was also performed, including maximal exercise test with electrocardiogram, 24-hour ambulatory blood pressure monitoring, 24-hour Holter monitoring, and blood sampling. **Results:** Seventy-four (56%) athletes had airway hyperresponsiveness to EVH or the methacholine inhalation test. Among those with airway hyperresponsiveness, 45 (61%) athletes were only hyperresponsive to EVH, and 10 (14%) were only hyperresponsive to the methacholine inhalation test (using the criteria of a $PC_{20} \leq 4$ mg/mL). Thirty-two (24%) athletes had a known diagnosis of asthma, while 34 (26%) athletes received a new asthma diagnosis. Ninety-seven (73%) athletes were sensitized to common airborne allergens. Forty-seven (35%) athletes completed the cardiovascular evaluation. Three (6%) and 7 (15%) athletes had a previous or new diagnosis of cardiovascular disease, respectively. Resting systemic hypertension was documented in 2 (4%) athletes and exaggerated blood pressure response to exercise was found in 12 (26%) athletes. **Conclusion:** This cardiorespiratory screening data set in athletes showed a high prevalence of exercise-induced asthma and exercise hypertension, which in many cases were not previously diagnosed.

**Keywords:** athletes; cardiorespiratory diseases; sport preparticipation evaluation

## Introduction

Exercise has many beneficial effects on health.[1–3] Physical exercise is thus increasingly prescribed for cardiac, respiratory, and metabolic diseases to counteract the effects of sedentary lifestyle.[3–5] However, when practiced at a very high frequency and intensity level, physical activity may also have deleterious effects and reveal or be responsible for the development of various health problems.[6] The high levels of ventilation and increased cardiac output sustained during training induce significant physiological stress in the cardiorespiratory system. In addition, increased inhalation of irritants or pollutants may also affect the cardiorespiratory system.[6]

In the athlete population, particular attention has been paid to sudden cardiac death (SCD) detection. Sports can act as a trigger for cardiac arrest in the presence of underlying cardiovascular diseases, predisposing athletes to life-threatening ventricular arrhythmias during physical exercise.[6] However, attention should also be

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paid to other cardiovascular diseases or risk factors, such as systemic hypertension, which is less frequently assessed in this population.

Cardiorespiratory symptoms are a poor predictor of exercise-induced bronchospasm (EIB) in athletes,[7] and the majority of cardiac conditions responsible for SCD are clinically silent and rarely diagnosed on the basis of symptoms.[8] Thus, reported past or current symptoms are not prerequisites for cardiorespiratory abnormality detection. Therefore, screening may be useful to detect or prevent the development of potentially disabling diseases or their adverse effects, which may impact athletic performance and future health. The majority of preparticipation programs emphasize the need to screen for potentially fatal cardiovascular diseases, but very little is done to detect other cardiorespiratory illnesses.[9] However, in Canada, in contrast to many European countries,[10] no standardized screening program for assessing the cardiorespiratory health of athletes is currently available.

Our aim of this study was to investigate, in a research setting, the prevalence of cardiorespiratory problems among elite endurance sports athletes from Québec, and to assess the usefulness of implementing a cardiorespiratory screening program adapted to the most common conditions discovered.

# Methods

## Subjects

Data were collected at the Centre de Recherche de l'Institut Universitaire de Cardiologie et de Pneumologie de Québec in Québec City, Québec, Canada, between August 2007 and December 2009. The Québec City area had 375 identified athletes who were members of Canadian national and provincial teams from different sports. Of these, 133 willingly participated in this research study, which consisted of a systematic screening program. Exclusion criteria were pregnancy, recent upper or lower respiratory tract infection (within 3 weeks of study enrollment), and tobacco use. Athletes had to be aged 14 to 30 years and train for ≥ 10 hours per week in a competitive endurance sport. Written informed consent was obtained from each subject and/or his or her parents or guardians before inclusion in the study. The protocol was approved by our institutional ethics committee.

## Study Design

Subjects were evaluated on 2 visits. Each evaluation was performed when the athletes had not trained for ≥ 12 hours. At the first visit, each subject completed a questionnaire about medical history, exercise-induced symptoms, and sport history, and underwent a physical examination. A blood sample was taken before the pulmonary evaluation to assess glycemic and lipid profile, as well as iron status was performed. During the pulmonary evaluation, spirometry, eucapnic voluntary hyperventilation (EVH), and methacholine inhalation tests were completed to assess airway function and responsiveness. An allergy skin-prick test for common airborne allergens was also performed. The cardiovascular evaluation included a physical examination, maximal exercise test with electrocardiogram (ECG), 24-hour cardiac monitoring (Holter), and 24-hour ambulatory blood pressure monitoring (ABPM). At the end of the pulmonary evaluation, ABPM and Holter were installed for a 24-hour period, during which athletes performed their normal daily activities, including training (light-to-moderate intensity).

## Questionnaires

Current cardiorespiratory symptoms and sport history and training were reported according to The European Community Respiratory Health Survey (ECRHS).[11] The Lausanne questionnaire 2004 was completed by the athletes for screening cardiorespiratory disorders, including personal and family medical history.[12]

## Blood Sample

A blood sample was obtained for metabolic parameters measurement. Blood glucose, plasma cholesterol, and triglyceride levels were determined using an enzymatic method (Hitachi, Modular P; Roche Diagnostics). Blood samples were collected in lithium heparin collection tubes. Serum apolipoproteinB (apoB), apolipoproteinA1 (apoA1) (Cobas Integra 800; Roche Diagnostics), and ferritin (Hitachi, Modular E-170; Roche Diagnostics) were also measured. Diabetes was defined by ≥ 2 fasting blood glucose level measurements of ≥ 7.0 mmol/L (fasting for 8 hours) and an oral glucose tolerance test result of ≥ 11.1 mmol/L 2 hours after a 75-g glucose ingestion.[13] Low ferritin level was defined as ≤ 35 μg/L.[14]

## Spirometry

Pulmonary function was assessed from flow-volume curves performed according to the American Thoracic Society specifications using an American Thoracic Society–approved spirometer.[15] Predicted values were derived from Knudson et al.[16]

## EVH

An EVH test was used to assess EIB.[17] The EVH test was performed according to the method described by Anderson

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and Brannan.[18] For 6 minutes, subjects inhaled a dry-air mixture containing 5% $CO_2$ and 95% air at room temperature. The target ventilation was 30 x forced expiratory volume in 1 second ($FEV_1$). $FEV_1$ was measured before the EVH test and at 3, 5, 10, 15, 20, 25, and 30 minutes afterward. A maximum post-EVH decrease in $FEV_1 \geq 10\%$ from baseline value was considered positive.

## Methacholine Challenge

Airway hyperresponsiveness (AHR) to methacholine was measured using the tidal breathing method described by Juniper et al.[19] After measurements of $FEV_1$ and forced vital capacity (FVC), subjects inhaled 0.9% saline followed by doubling concentrations of methacholine, ranging from 0.03 to 16 mg/mL, to obtain a 20% decrease in $FEV_1$. Methacholine aerosols were generated from a Wright nebulizer with an output of 0.13 mL/min and were inhaled for 2 minutes at 5-minute intervals. $FEV_1$ was measured at 30 and 90 seconds after each inhalation and every 2 minutes until it started to improve. An acceptable-quality $FEV_1$ was obtained at each time point; otherwise, the $FEV_1$ maneuver was repeated.

## AHR

Airway hyperresponsiveness was defined as: 1) a post-EVH decrease in $FEV_1$ of $\geq$ 10% from baseline sustained for $\geq$ 5 minutes or observable at 2 consecutive time points, or 2) a concentration of inhaled methacholine causing a 20% decrease in $FEV_1$ ($PC_{20}$) $\leq$ 4 mg/mL and $PC_{20} \leq$ 16 mg/mL for athletes using inhaled corticosteroids, according to the World Anti-Doping Agency (WADA) and the International Olympic Committee Medical Commission (IOC-MC) guidelines.[20]

## Asthma Diagnosis

Criteria for asthma diagnosis were based on the Canadian Thoracic Society Asthma Management Continuum 2010[21] and the WADA/IOC-MC criteria.[20] These criteria included: 1) the presence of recurrent cardiorespiratory symptoms of bronchial obstruction, such as chest tightness, wheeze, and cough provoked by exercise or other stimuli, and 2) evidence of airflow obstruction reversibility (increased $\geq$ 12% of $FEV_1$ after inhalation of $\beta_2$-agonists), and/or the presence of AHR to bronchial-provocation tests.

## Skin-Prick Tests

Skin-prick tests were performed with a battery of 25 common airborne allergens: standard cat hair, standard cat pelt, dog hair dander, horse hair dander, cattle hair dander, ash white, common cottonwood, birch mix, American elm, box elder maple, oak mix RVW, standard grass mix #5, ragweed mix, sagebrush, English plantain, common cocklebur, *Alternaria tenuis*, *Hormodendrum*, *Aspergillus fumigatus*, house dust mix, standard *Dermatophagoides pteronyssinus*, feather mix 3 (Omega Laboratories), standard grass pollen timothy, standard grass pollen perennial ryegrass, and standard *Dermatophagoides farinae* (Hollister-Stier Laboratories LLC). Saline control (Omega Laboratories) and histamine (ALK-Abello Pharmaceuticals, Inc.) were used as negative and positive controls, respectively. A positive response was defined as a skin wheal diameter $\geq$ 3 mm.

## Maximal Aerobic Capacity Test

A progressive maximal aerobic capacity test (RAMP) was performed.[22] This test was performed on an ergocycle (Corival; Lode B.V.) or treadmill (Trackmaster TMX425; Full Vision Inc.), depending on which exercise test modality was closer to the athlete's sport. During this test, each subject breathed through a mouthpiece connected to a gas analyzer (CardiO2 CPX Ultima; Medical Graphics Co.) to obtain breath-by-breath measurements of oxygen comsumption ($VO_2$), carbon dioxide production ($VCO_{20}$), and ventilation (VE). Heart rate (HR) was continuously monitored (CardioPerfect software; Welch-Allyn Inc.) and BP was measured (STBP-780; Colin Medical Instruments Corp.) every 2 minutes. Exaggerated BP response to exercise was defined as systolic BP (SBP) $\geq$ 220 mm Hg and/or diastolic BP (DBP) $\geq$ 100 mm Hg.[23]

## BP Monitoring

Twenty-four–hour ambulatory blood pressure monitoring was performed. Blood pressure was measured every 20 minutes during the day and every 60 minutes during the night. Resting systemic hypertension was defined as SBP $>$ 135 mm Hg and/or DBP $>$ 85 mm Hg averaged during the 24-hour ABPM.[24]

## Cardiac Monitoring

Holter monitoring was used for the diagnosis of cardiac conduction disorders and disturbances in the heart's rhythm, especially if they are paroxysmal (intermittent) and asymptomatic. Twenty-four–hour Holter monitoring was used to determine minimal and maximal HR, the existence of bradycardia or tachycardia, and the number and type of extrasystoles. Analysis of repolarization disorders (ST-segment) evaluated the presence of cardiac ischemia, which may be indicative of coronary artery disease. During the 24-hour recording, the subjects continued their regular daily activities.

## Statistical Analyses

Statistical analyses were performed to assess the differences between sport categories on a set of dependent variables. For continuous variables, 1-way analyses of variances were first performed. Residual normality was verified using Shapiro-Wilk test and homogeneity of variances was assessed with Levene's test. Because both tests did not provide significant results at $P = 0.01$, an analysis of variance was used to interpret the following variables: height, daytime DBP, maximal exercise SBP, maximal exercise DBP, resting HR, predicted $FEV_1$, predicted FVC, apoA1, apoB, total (24-hour) DBP, glucose, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, nighttime DBP, total (24-hour) SBP, total cholesterol, total cholesterol/high-density lipoprotein cholesterol, triglycerides, and maximal oxygen consumption ($VO_2$max). For variables with non-normal distribution for residuals, a nonparametric Kruskal-Wallis one-way analysis of variance was performed for the following variables: daytime SPB, nighttime SBP, apoA1/apoB, EVH, ferritin level, number of trainings per week, and number of years in sport. Because homogeneity of variance provided significant results, a linear model was performed considering heterogeneous variances among sport categories for weight and maximal HR. Some variables (eg, natural logarithm of $PC_{20}$ and inverse of age) were transformed to fulfill the normality and variance assumptions. Categorical variables (ie, AHR, atopy, $PC_{20} \leq 4$ mg/mL, $PC_{20} \leq 16$ mg/mL, exercise-induced symptoms, exercise-induced cardiorespiratory symptoms, family history of asthma, family history of cardiovascular disease, positive EVH [decrease in $FEV_1 \geq 10\%$], no confirmed diagnosis of asthma, new diagnosis of asthma, new diagnosis of cardiovascular disease, previous diagnosis of asthma, previous diagnosis of cardiovascular disease, sex, ferritin level $\leq 35$ µg/L, and other diagnosis) were analyzed using a chi-square test of association. All analyses were performed using SAS software, version 9.2.

## Results

### Subjects' Characteristics

One hundred thirty-three elite athletes participating in 5 endurance sports (ie, cross-country skiing, biathlon, triathlon and cycling, long track speed skating, and swimming) on the Canadian national and provincial teams were studied, and a t test was used to compare the subjects. Athletes' characteristics according to their different sport are summarized in Table 1. There were 71 men and 62 women, aged 20 ± 4 years (mean ± SD). Differences in age ($P < 0.0001$) and sex ($P = 0.008$) were observed between sports. More men were triathlon participants and cross-country skiers, while more women were swimmers. Athletes were older in the triathlon group compared with the cross-country skiing ($P < 0.0001$), biathlon ($P = 0.002$), and swimming groups ($P = 0.0001$). Cross-country skiers were younger compared with swimmers ($P = 0.02$). Athletes had been competing in their sport for an average of 8 ± 3 years. Biathletes had been practicing their sport for a shorter period of time compared with swimmers ($P = 0.0005$) and long track speed skaters ($P = 0.04$), while swimmers had more experience than cross-country skiers ($P = 0.008$). Athletes trained for a mean of 18 ± 6 hours per week. The number of training hours was comparable between each sport except for swimmers, who trained for a greater number of hours per week ($P = 0.0003$).

### Cardiorespiratory Disorders

Athletes' cardiorespiratory function characteristics according to their sport are summarized in Table 2. Athletes had baseline $FEV_1$ of 115 ± 15% and FVC of 124 ± 15% of the predicted values (which are based on sex, age, and height). Sixty-four athletes (48%) had a percent decrease in $FEV_1$ after EVH ≥ 10%. Thirty-nine athletes (29%) had a $PC_{20}$ methacholine ≤ 16 mg/mL and 29 (22%) had a $PC_{20} \leq$ 4 mg/mL. Thus, 74 (56%) athletes had AHR to EVH or the methacholine inhalation test. Among those with AHR, 45 (61%) athletes were hyperresponsive to EVH and 10 (14%) were hyperresponsive to the methacholine inhalation test ($PC_{20} \leq 4$ mg/mL). Among the 32 (24%) athletes who reported a diagnosis of asthma, 9 (7%) had no objective evidence of asthma after testing. Among the athletes without evidence of asthma, 1 female biathlete had a diagnosis of vocal cord dysfunction confirmed by an otorhinolaryngologist. A new diagnosis of asthma was documented in 34 (26%) athletes. Eighty-two (62%) athletes reported exercise-induced cardiorespiratory symptoms and 31 (23%) had family history of asthma. Ninety-seven (73%) athletes were sensitized to common airborne allergens.

Baseline $FEV_1$ was similar in the 5 athlete groups, while FVC was higher in swimmers compared with the other athletes ($P < 0.05$). A higher percentage of decrease in $FEV_1$ after EVH was observed in swimmers compared with cross-country skiers, triathletes, and long track speed skaters ($P < 0.05$). $PC_{20}$ was lower in swimmers compared with triathletes, long track speed skaters, and biathletes ($P < 0.0001$). Therefore, AHR was more frequently observed in swimmers ($P = 0.0003$) than in other athletes. However, no difference in previous diagnosis of asthma ($P = 0.56$), family history of asthma ($P = 0.75$), exercise-induced cardiorespira-

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**Table 1.** Athletes' Characteristics

| Characteristic | All | Cross-Country Skiers | Biathletes | Triathletes/ Cyclists | Long Track Speed Skaters | Swimmers | *P* Value |
|---|---|---|---|---|---|---|---|
| **Subjects, n** | **133** | **34** | **10** | **19** | **20** | **50** | |
| Male:female | 71:62 | 24:10 | 5:5 | 13:6 | 12:8 | 17:33 | 0.008 |
| Age, mean ± SD | 20 ± 4 | 18 ± 2 | 19 ± 2 | 24 ± 5 | 19 ± 3 | 20 ± 4 | < 0.0001 |
| Weight, kg, mean ± SD | 66 ± 8 | 65 ± 7 | 63 ± 5 | 68 ± 8 | 69 ± 13 | 66 ± 9 | 0.25 |
| Height, cm, mean ± SD | 173 ± 8 | 173 ± 7 | 170 ± 6 | 176 ± 9 | 174 ± 10 | 174 ± 9 | 0.39 |
| Duration of training, mean ± SD | 8 ± 3 | 7 ± 3 | 7 ± 4 | 8 ± 4 | 10 ± 4 | 10 ± 3 | 0.005 |
| Training, h/wk, mean ± SD | 18 ± 6 | 12 ± 2 | 15 ± 5 | 17 ± 7 | 16 ± 5 | 21 ± 6 | 0.0003 |

tory symptoms ($P = 0.80$), and atopy ($P = 0.78$) was found between sports, except for a new diagnosis of asthma, which tended to be more frequent in swimmers ($P = 0.06$).

## Cardiovascular Disorders

Athletes' cardiovascular characteristics according to their sport are summarized in Table 3. Forty-seven (35%) of the 133 athletes underwent the cardiovascular evaluation. All athletes had a blood sample taken. The cardiovascular evaluation was added later in the research protocol due to procedural and methodological reasons. Swimmers did not undergo the cardiovascular evaluation for technical reasons. Mean resting HR (measured by 24-hour Holter monitoring) was 39 ± 5 bpm, and total (24-hour) mean BP at ABPM was 109/58 ± 12/7 mm Hg. During the RAMP test, maximal HR was 190 ± 8 bpm and maximal BP was 202/70 ± 29/10 mm Hg (measured at 100% of the $VO_2$max). No difference between sports was found regarding resting ($P = 0.53$) and maximal HR ($P = 0.56$). However, cross-country skiers and biathletes showed lower total DBP, daytime DBP, and nighttime DBP than triathletes and speed skaters ($P < 0.05$), while SBP was not different between sports ($P = 0.48$). Exercise SBP and DBP was similar between sports ($P = 0.52$). Mean $VO_2$max was 61 ± 9 mL/kg/min. Cross-country skiers, biathletes, and triathletes had higher $VO_2$max than long track speed skaters ($P < 0.05$).

Three (6%) of the tested athletes had a previous diagnosis of cardiovascular disease. Seven (15%) new diag-

**Table 2.** Pulmonary Characteristics

| Characteristic | All | Cross-Country Skiers | Biathletes | Triathletes/ Cyclists | Long Track Speed Skaters | Swimmers | *P* Value |
|---|---|---|---|---|---|---|---|
| **Subjects, n** | **133** | **34** | **10** | **19** | **20** | **50** | |
| $FEV_1$, % of predicted, mean ± SD | 115 ± 15 | 111 ± 12 | 113 ± 8 | 114 ± 17 | 111 ± 12 | 120 ± 17 | 0.10 |
| FVC, % of predicted, mean ± SD | 124 ± 15 | 118 ± 12 | 117 ± 11 | 120 ± 16 | 118 ± 12 | 131 ± 15 | 0.001 |
| $PC_{20}$, mg/mL, geometric mean (range) | 18.8 (0.3–128) | 53.04 (0.5–128) | 78 (4.7–128) | 33.9 (1.4–128) | 53.04 (0.5–128) | 4.8 (0.26–128) | < 0.0001 |
| EVH, % decrease in $FEV_1$, geometric mean (range) | 10 (0–62) | 8.2 (0–43.5) | 10.5 (4.1–30) | 9.1 (0–62) | 8.2 (0–43.5) | 12.1 (0–38.5) | 0.004 |
| EVH positive, n (%) | 64 (48) | 7 (35) | 6 (60) | 7 (37) | 7 (35) | 33 (66) | 0.001 |
| $PC_{20} \leq 16$ mg/mL, n (%) | 39 (29) | 5 (25) | 4 (40) | 6 (31) | 5 (25) | 36 (72) | < 0.001 |
| $PC_{20} \leq 4$ mg/mL, n (%) | 29 (22) | 3 (15) | 0 (0) | 3 (16) | 3 (15) | 20 (40) | 0.0005 |
| AHR, n (%) | 74 (56) | 10 (50) | 6 (60) | 7 (37) | 10 (50) | 39 (78) | 0.0003 |
| Previous diagnosis of asthma, n (%) | 32 (24) | 7 (35) | 1 (10) | 4 (21) | 7 (35) | 10 (20) | 0.56 |
| No confirmed diagnosis of asthma after testing, n (%) | 9 (7) | 3 (15) | 1 (10) | 0 (0) | 3 (15) | 1 (2) | 0.32 |
| New diagnosis of asthma after testing, n (%) | 34 (26) | 3 (15) | 2 (20) | 4 (21) | 3 (15) | 20 (40) | 0.06 |
| Atopy, n (%) | 97 (73) | 16 (80) | 8 (80) | 12 (63) | 16 (80) | 39 (78) | 0.78 |
| Exercise-induced respiratory symptoms, n (%) | 31 (23) | 14 (70) | 8 (80) | 12 (63) | 14 (70) | 30 (60) | 0.81 |
| Family history of asthma, n (%) | 82 (62) | 2 (10) | 4 (40) | 4 (21) | 2 (10) | 12 (24) | 0.75 |

**Abbreviations:** AHR, airway hyperresponsiveness; EVH, eucapnic voluntary hyperpnea; $FEV_1$, forced expiratory volume in 1 second; FVC, forced vital capacity; $PC_{20}$, concentration of inhaled methacholine causing a 20% decrease in forced expiratory volume in 1 second.

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noses of cardiovascular diseases were found in athletes and 41 (87%) reported exercise-induced cardiovascular symptoms (ie, palpitations, near syncope, and chest pain). However, no differences regarding new diagnoses of cardiovascular diseases, family history of cardiovascular disease, or exercise-induced cardiovascular symptoms were found between sports. Two athletes fulfilled the diagnosis for systemic hypertension (SBP $>$ 135 mm Hg and/or DBP $>$ 80 mm Hg) and began drug treatment. Twelve (26%) male athletes had exaggerated BP response to exercise (SBP $\geq$ 220 and/or DBP $\geq$ 100 mm Hg measured at 100% of the $VO_2$max). All had exaggerated BP response to exercise based on high SBP, while DBP remained within the normal range. In 1 cross-country skier, the RAMP test and 24-hour Holter monitoring revealed an idiopathic supraventricular tachyarrhythmia (8 heart beats at 235 bpm), for which electrophysiology laser ablation was unsuccessfully performed, because the ectopic heart beat could not be stimulated. This athlete had exercise-induced symptoms of palpitations, chest pain, near syncope, and family history of cardiovascular disease.

A second cross-country skier sustained vascular claudication. The symptoms began with left hip pain, occasional cramps in both hips, and left buttock pain during exercise, resulting in decreased performance. Abdominal aorta and iliac arteries magnetic resonance angiography showed a short 20% to 30% stenosis in the middle third of the left external iliac artery, suggestive of endofibrosis of the left external iliac artery. Angiography with intravenous drug stimulation and measurement of intravascular pressure was normal. However, vascular Doppler and pressure measurement immediately after maximal exercise treadmill, suggested abnormal Doppler waves with important turbulence to the left common femoral artery and a significant pressure decrease in the lower left limb. Vascular surgery with vein patch on the left iliac artery was performed. A resting period of 6 weeks and mild training for another 6 weeks were

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**Table 3.** Cardiovascular Characteristics

| Characteristic | All | Cross-Country Skiers | Biathletes | Triathletes/ Cyclists | Long Track Speed Skaters | *P* Value |
|---|---|---|---|---|---|---|
| **Subjects, n** | **47** | **15** | **9** | **12** | **11** | |
| Resting HR, bpm, mean ± SD<br>Normal: 60–90 bpm | 39 ± 5 | 39 ± 4 | 38 ± 6 | 37 ± 3 | 40 ± 6 | 0.53 |
| Total (24-hour) SBP, mm Hg, mean ± SD<br>Normal: $<$ 130 mm Hg | 109 ± 12 | 110 ± 14 | 102 ± 5 | 113 ± 12 | 112 ± 4 | 0.04 |
| Total (24-hour) DBP, mm Hg, mean ± SD<br>Normal: $<$ 80 mm Hg | 58 ± 7 | 56 ± 6 | 54 ± 5 | 61 ± 9 | 61 ± 5 | 0.22 |
| Daytime SBP, mm Hg, mean ± SD<br>Normal $<$ 135 mm Hg | 120 ± 10 | 120 ± 11 | 117 ± 8 | 121 ± 13 | 123 ± 7 | 0.48 |
| Daytime DPB, mm Hg, mean ± SD<br>Normal $<$ 85 mm Hg | 71 ± 8 | 68 ± 6 | 68 ± 4 | 73 ± 11 | 75 ± 5 | 0.05 |
| Nighttime SBP, mm Hg, mean ± SD<br>Normal $<$ 115 mm Hg | 119 ± 10 | 118 ± 11 | 115 ± 7 | 112 ± 13 | 122 ± 9 | 0.36 |
| Nighttime DBP, mm Hg<br>Normal: $<$ 65 mm Hg | 69 ± 6 | 66 ± 6 | 66 ± 4 | 70 ± 7 | 74 ± 5 | 0.01 |
| Maximal HR, bpm, mean ± SD<br>Normal: 200 ± 12 bmp | 190 ± 8 | 188 ± 12 | 194 ± 3 | 191 ± 6 | 186 ± 7 | 0.56 |
| Maximal exercise SBP, mm Hg, mean ± SD<br>Normal: 220 mm Hg | 202 ± 29 | 202 ± 38 | 189 ± 25 | 208 ± 24 | 204 ± 23 | 0.04 |
| Maximal DBP exercise, mm Hg, mean ± SD<br>Normal: 100 mm Hg | 70 ± 10 | 70 ± 9 | 68 ± 14 | 74 ± 5 | 69 ± 12 | 0.52 |
| $VO_2$max, mL/kg/min, mean ± SD [a](males: 55.1;females: 49.0) | 61 ± 9 | 66 ± 7 | 60 ± 11 | 64 ± 8 | 53 ± 6 | $<$ 0.0001 |
| Previous diagnosis of cardiovascular condition, n (%) | 3 (6) | 1 (3) | 0 (0) | 1 (5) | 1 (5) | 0.90 |
| New diagnosis of cardiovascular condition, n (%) | 7 (9) | 3 (9) | 1 (10) | 1 (5) | 2 (10) | 0.29 |
| Family history of cardiovascular disease ($<$ 50 years), n (%) | 11 (23) | 4 (12) | 1 (10) | 1 (5) | 5 (25) | 0.29 |
| Exercise-induced cardiovascular symptoms, n (%) | 41 (87) | 15 (44) | 5 (50) | 10 (53) | 11 (55) | 0.26 |

[a]Normal values in healthy subjects: 90th percentile (age, 20–29 years).
Adapted from La Société Québécoise D'hypertension Artérielle[62] and the American College of Sports Medicine.[63]
**Abbreviations:** DBP, diastolic blood pressure; HR, heart rate; SBP, systolic blood pressure; $VO_2$max, maximal oxygen consumption.


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necessary for recovery. Normal training resumed 3 months postoperatively without any remaining symptoms.

Another cross-country skier had a diagnosis of systemic hypertension following 24-hour ABPM (mean, 140/72 mm Hg) and maximal BP exercise test, which showed BP of 320/85 mm Hg at 100% of the $VO_2$max. This cross-country skier had a family history of systemic hypertension, and blood and imaging investigations (ECG and magnetic resonance imaging of the renal arteries) showed no secondary cause of hypertension. He experienced exercise-induced symptoms (ie, nose bleeding, severe headache, and fatigue) when he was training at a high altitude. This athlete was treated with angiotensin II receptor antagonists, but reported difficulties performing at his usual level; he expressed the feeling of not having as much power as he had before starting the drug regimen. Finally, a treatment with doxazosin 2 mg, a selective inhibitor of the α-1 subtype of α-adrenergic receptors was initiated, and this feeling of being limited during exercise disappeared, while allowing BP to remain at normal levels. One triathlete had a diagnosis of hypertension; he had no family history of cardiovascular disease and no symptoms during exercise. The investigation showed no secondary cause of hypertension. Medication of another triathlete who already had a diagnosis of systemic hypertension was modified because APBM showed that BP was not optimally treated. A swimmer with a previous diagnosis of systemic hypertension who was treated with β-blockers showed no evidence of systemic hypertension during ABPM after cessation of the medication. He had no family history of cardiovascular disease. In 1 female long track speed skater, physical examination showed systolic murmurs and ECG revealed an aortic bicuspid valve. She never experienced any exercise-induced cardiovascular symptoms, but she had a family history of cardiovascular disease. Two female athletes, 1 long track speed skater and 1 biathlete, had severe hypotension following exercise and frequently experienced near-syncope during exercise. Insufficient hydration seemed to be the best explanation, as no other abnormalities were

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**Table 4.** Metabolic Parameters

| | All | Cross-Country Skiers | Biathetes | Triathletes/ Cyclists | Long Track Speed Skaters | Swimmers | *P* Value |
|---|---|---|---|---|---|---|---|
| **Subjects, n** | **47** | **15** | **9** | **12** | **11** | **50** | |
| Blood glucose, mmol/L, mean ± SD<br>Normal: 3.6–5.8 mmol/L | 4.5 ± 0.4 | 4.6 ± 0.4 | 4.2 ± 0.2 | 4.6 ± 0.5 | 4.6 ± 0.3 | N/A | 0.25 |
| TC, mmol/L, mean ± SD<br>Normal: 4.1–6.2 mmol/L | 4.3 ± 0.7 | 4.4 ± 0.6 | 4.1 ± 0.6 | 4.3 ± 0.9 | 4.5 ± 0.6 | N/A | 0.61 |
| TGs, mmol/L, mean ± SD<br>Normal: males, 0.5–2 mmol/L; females, 0.4–1.6 mmol/L | 1.0 ± 0.4 | 1.0 ± 0.5 | 1.0 ± 0.3 | 0.8 ± 0.2 | 1.1 ± 3.0 | N/A | 0.39 |
| HDL-C, mmol/L, mean ± SD<br>Normal: > 1 | 1.5 ± 0.3 | 1.4 ± 0.3 | 1.5 ± 0.3 | 1.5 ± 0.3 | 1.5 ± 0.4 | N/A | 0.97 |
| LDL-C, mmol/L, mean ± SD<br>Normal: < 4.10 | 2.4 ± 0.6 | 2.5 ± 0.7 | 2.1 ± 0.4 | 2.5 ± 0.6 | 2.5 ± 0.7 | N/A | 0.46 |
| TC/HDL-C, mean ± SD | 3.0 ± 0.7 | 3.2 ± 0.7 | 2.8 ± 0.4 | 2.9 ± 03 | 3.2 ± 0.9 | N/A | 0.45 |
| apoB, g/L, mean ± SD<br>Normal: males, 0.5–1.3 g/L; females, 0.5–1.2 g/L | 0.7 ± 0.2 | 0.7 ± 0.2 | 0.7 ± 0.1 | 0.6 ± 0.1 | 0.8 ± 0.2 | N/A | 0.02 |
| apoA1, g/L, mean ± SD<br>Normal: males, 1.20–1.60 g/L; females, 1.30–2.10 g/L | 1.5 ± 0.2 | 1.5 ± 0.2 | 1.5 ± 0.2 | 1.6 ± 0.3 | 1.5 ± 0.2 | N/A | 0.42 |
| apoA1/apoB, mean ± SD | 0.5 ± 0.1 | 0.5 ± 0.1 | 0.4 ± 0.1 | 0.4 ± 0.1 | 0.5 ± 0.2 | N/A | 0.006 |
| Ferritin level, (μg/L), geometric mean (range)<br>Normal: 30–400 μg/L | 55 (9–323) | 53 (9–184) | 80 [33–176] | 61 [20–169] | 71 [19–323] | 41 (9–194) | 0.05 |
| Low ferritin levels (< 35 μg/L), n (%) | 25 (21 F:3 M) (19%) | 4 (3 F:1 M) (12%) | 1 (1 F) (10%) | 3 (2 F:1 M) (16%) | 4 (4 F) (20%) | 13 (12 F:1 M) (26%) | 0.07 |
| Other diagnoses, n (%) | 5 (4%) | 2 (2%) (overtraining)<br>1 (<1%) (anemia) | 1 (<1%) (VCD) | N/A | N/A | 1 (< 1%) (anemia) | 0.11 |

**Abbreviations:** apoA1, apolipoprotein A1; apoB, apolipoprotein B; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; N/A, not available; TC, total cholesterol; TG, triglyceride; VCD, vocal cord dysfunction.

found and no family history of cardiovascular disease was reported. After consultation with a sport nutritionist, the symptoms disappeared.

## Metabolic Parameters

All athletes (N = 133) had blood samples taken, except swimmers for whom blood glucose and lipid measurements were not performed. Higher apoB levels were observed in long track speed skaters and cross-country skiers compared with triathletes ($P = 0.002$). Low ferritin levels ($< 35$ µg/L) were observed in 25 (19%) athletes, including 22 (88%) female athletes. Two (8%) of these athletes had a diagnosis of anemia. Results are presented in Table 4. One ($< 1\%$) cross-country skier had a diagnosis of type 1 diabetes mellitus after repeated measurements of high blood glucose level and a positive oral glucose tolerance test. This skier had no exercise-induced symptoms and no family history of diabetes. One athlete reported family hypercholesterolemia. Two (2%) athletes had overtraining syndrome confirmed by a sport physician. Overtraining diagnosis criteria were essentially based on chronic fatigue, decrease in athletic performance, and increased resting HR.

# Discussion

This study is the first to report the prevalence of cardiorespiratory disorders in Canadian athletes. A high prevalence of cardiorespiratory diseases was observed in this population. This screening allowed the identification of a significant number of previously undetected conditions. These data may be the first step in making recommendations to establish the basis of cardiorespiratory screening programs in Canadian athletes.

To date, athlete health-screening programs have focused on cardiovascular diseases that cause sudden death,[25,26] as well as orthopedic[27] and head injuries.[28] However, other health conditions are very important in the context of high-performance sport. Among these are hematological and iron-related conditions,[29] pulmonary diseases,[30] and the female athlete triad (ie, disordered eating, amenorrhea [cessation of menses], and osteoporosis).[31]

Increased expiratory flow rates in athletes possibly result from adaptation mechanisms, but this remains to be determined. Airway hyperresponsiveness was the most common pathology observed in these athletes. Fifty-six percent had AHR and the highest prevalence was found in swimmers (78%); this is much higher than the prevalence found in the general Canadian population (10.7%).[32] Our results are comparable with those found in other athlete populations, with prevalence being between 10% and 76%, depending on the sport.[33,34] The high-minute ventilation sustained during exercise and the great number of training hours in specific environments increase airway exposure to many allergens, pollutants, and irritants that are probably responsible for the high prevalence of AHR in athletes. Sixty-two percent of athletes reported exercise-induced cardiorespiratory symptoms, the highest prevalence being observed in outdoor winter sports athletes due to the repeated inhalation of dry, cold air.

The wide variation in the reported prevalence of EIB (10%–76%)[33,34] in previous reports may be related in part to the poor correlation between subjective symptoms and objective tests. Some studies of EIB have reported prevalence rates according to symptoms alone; consequently, the prevalence may be inaccurate, particularly in high-ventilation sports.[35] This is of particular clinical relevance because physicians commonly diagnose and treat suspected EIB empirically according to symptoms alone, without objective measurement. Athletes therefore often experience cardiorespiratory symptoms without AHR or respiratory tract infection. Differential diagnosis of asthma, such as vocal cord dysfunction, can easily mimic EIB symptoms. Although the nonspecific nature of the symptoms can lead to overdiagnosed EIB, it can also lead to missed diagnoses of EIB because symptoms may be mistaken for exhaustion or lack of physical conditioning compared with other teammates, attributing EIB to normal manifestations of intense exercise. As a result, in many athletes, EIB may be under-recognized.[35] Untreated EIB can affect athletes' training and performance, while treating athletes without EIB can also have adverse effects. Some asthma medications still require a therapeutic-use exemption, while tachyphylaxis and tachycardia can occur after $\beta_2$-agonists use, and inhaled corticosteroids can produce dysphonia and oropharyngeal candidiasis. Moreover, regular treatment with short- or long-acting $\beta_2$-agonists diminishes the bronchoprotective effect,[36–41] and there is also evidence to suggest that regular $\beta_2$-agonist use increases the underlying severity of EIB.[38,42]

In our population of athletes, atopy was more prevalent than (73%) in the general Canadian population (62.7%)[43] or other studies' athlete populations (48%–59%).[44–46] This observation could be explained by the fact that: 1) the Canadian population has a higher prevalence of atopy compared with European (36.8%)[47] and US (54.3%) population,[48] and 2) there is an increased prevalence of atopy and AHR in the French-Canadian population, probably due to genetic factors.[49] Allergic rhinitis often leads to snoring,

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sleep disturbance, headaches, fatigue, and impaired quality of life, including athletic performance. Moreover, untreated rhinitis can be responsible for chronic cough or contribute to poor asthma control.[50]

We found a 4% prevalence of resting systemic hypertension and a 26% prevalence of exaggerated BP response to exercise in athletes. Two athletes were diagnosed with resting systemic hypertension, 1 had family history of hypertension, and none had secondary cause of hypertension. Although the prevalence of systemic hypertension is approximately 50% lower in athletes compared with the general population, hypertension is the most common cardiovascular disease in athletes.[51] It is well known that systemic hypertension is a silent disease and subjects usually experience no symptoms. For these reasons, screening becomes the only way to detect hypertension, as is the case for many other silent cardiovascular conditions. The increased skeletal and cardiac muscular mass found in athletes and the high cardiac output that they generate may contribute to the increased systemic pressure. After the retirement of sports competition, left ventricular mass decreases to approximately 7% to 15%[52]; however, no data are available on BP. Treatment of hypertensive athletes is important to prevent future damage to target organs. Exercise hypertension was the most common cardiovascular condition found in our cohort of athletes. Twenty-six percent had an SBP $>$ 220 mm Hg. Although the ABPM values were within the normal range in athletes with hypertensive response to exercise, this may reflect subtle pathophysiological features of a preclinical stage of hypertension. However, the meaning of this finding will remain unclear until "normal" values of exercise BP in high-level athletes is better defined. Nevertheless, it has been observed that healthy, nonathlete subjects showing hypertensive response to exercise have an increased risk of future resting systemic hypertension.[53–55] Moreover, Tanji[56] observed that 80% of athletes found to have systemic hypertension in sport preparticipation screening developed hypertension later in life, which demonstrated the importance of screening for hypertension in otherwise seemingly healthy athletes. Longitudinal studies are needed to assess the outcomes of exercise hypertension in athletes.

Comparison between studies is difficult due to nonstandard exercise testing protocols and definitions of exaggerated BP response to exercise. This methodological variability may make the comparability between the prevalence of exercise-induced hypertension and the prediction of the risk of future hypertension difficult. Moreover, studies in young adults have been rare, and these young adults often have risk factors that are not present in athletes, such as high body mass index ($>$ 25 kg/m$^2$), smoking, and inactivity.

A study in healthy young men (mean age, 25 years) reported a 10.6% prevalence of exaggerated BP response to exercise. These subjects who showed exaggerated BP response after ergometer exercise were at increased risk of hypertension 5.8 years later.[53] The Coronary Artery Risk Development in Young Adults (CARDIA) study in young adults (mean age, 25 years) reported a prevalence of exaggerated SBP response to exercise of 18%, and exaggerated SBP response was more common in men and in black subjects. The authors reported that BP elevation during treadmill exercise in white and black men and women was related to elevation of SBP after 5 years.[57] A 12-year follow-up study of young adults (mean age, 19 years) demonstrated that BP immediately after ergometer exercise, BP in men and resting BP in women showed a strong correlation with resting BP at follow-up.[58]

Exercise-induced cardiovascular symptoms were also highly reported by athletes in the current study. Eighty-seven percent of them reported palpitations, near syncope, or chest pain during exercise. However, only 10 athletes had a cardiovascular condition that could possibly explain the symptoms. In several athletes who reported these symptoms, we observed a poor hydration state. Inadequate intake of water and electrolytes could be responsible for their symptoms.

Low ferritin levels ($<$ 35 µg/L) were found in 19% of athletes of whom 88% were women and 8% had anemia. Training, particularly endurance training, decreases iron reserves, and female athletes are more prone to have low ferritin levels because of menses.[59] Low ferritin levels with low hemoglobin levels may lead to anemia, with a consequent decreased work capacity.[60] Although the prevalence of anemia associated with iron deficiency is low (3%) in both the general and athlete populations, low ferritin levels alone may impact sport performance negatively.[61] However, ferritin levels required for the introduction of iron supplementation are still controversial. Studies proposed different target levels between 12 to 20 µg/L, but the highest level is 35 µg/L.[14] Fogelholm[61] reviewed 6 studies of iron deficiency in women (limit of 12–20 µg/L); 37% of female athletes and 23% of female nonathletes were iron deficient.

## Limitations

Our study may have a selection bias. Athletes who were included in the screening may have decided to participate because they experienced symptoms during exercise.


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## Conclusion

The majority of athletes with AHR did not have a history of asthma. Our results imply that empirically diagnosing and treating athletes for EIB without objective testing likely leads to inaccurate diagnoses and may eventually lead to unnecessary morbidity. Further studies of athletes with exaggerated BP response to exercise are needed to investigate the clinical significance and to guide recommendations regarding which athletes should be screened with objective tests before participation in competitive sports. Finally, 26% of athletes had diagnoses other than asthma and arterial hypertension that have interfered with their athletic performance. Treatment and follow-up for these conditions can help to improve sport performance. However, the cost benefit of such programs remains to be determined.

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## Conflict of Interest Statement

Julie Turmel, PhD, Paul Poirier, MD, PhD, Valérie Bougault, PhD, Evelyne Blouin, BSc and Mireille Belzile, MD disclose no conflicts of interest. Louis-Philippe Boulet, MD discloses conflicts of interest with 3M, Altair, Amgen, Asmacure, AstraZeneca, Boehringer-Ingelheim, Genentech, GlaxoSmithKline, Merck Frosst, Novartis, Pharmaxis, Schering, and Wyeth.

### References

1. Irvine C, Taylor NF. Progressive resistance exercise improves glycaemic control in people with type 2 diabetes mellitus: a systematic review. *Austr J Physiother*. 2009;55(4):237–246.
2. Manfredini F, Malagoni AM, Mandini S, et al. Sport therapy for hypertension: why, how, and how much? *Angiology*. 2009;60(2):207–216.
3. Nici L, Donner C, Wouters E, et al. American Thoracic Society/European Respiratory Society Statement on Pulmonary Rehabilitation. *Am J Respir Crit Care Med*. 2006;173(12):1390–1413.
4. Donnelly JE, Blair SN, Jakicic JM, Manore MM, Rankin JW, Smith BK. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Med Sci Sports Exerc*. 2009;41(2):459–471.
5. Franklin BA, Whaley MH, Howley ET, et al; American College of Sports Medicine. *Guidelines for Exercise Testing and Prescription*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2000.
6. Thompson PD, Franklin BA, Balady GJ, et al; American College of Sports Medicine. Exercise and acute cardiovascular events placing the risks into perspective: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism. *Circulation*. 2007;115(17):2358–2368.
7. Turcotte H, Langdeau JB, Thibault G, Boulet LP. Prevalence of respiratory symptoms in an athlete population. *Respir Med*. 2003;97:955–963.
8. Vancamp SP, Bloor CM, Mueller FO, Cantu RC, Olson HG. Nontraumatic sports death in high school and college athletes. *Med Sci Sports Exerc*. 1995;27(5):641–647.
9. Corrado D, Basso C, Thiene G. Sudden cardiac death in athletes: what is the role of screening? *Curr Opin Cardiol*. 2012; 27(1):41–48.
10. Corrado D, Basso C, Schiavon M, Pelliccia A, Thiene G. Pre-participation screening of young competitive athletes for prevention of sudden cardiac death. *J Am Coll Cardiol*. 2008;52(24):1981–1989.
11. Mahler DA. Exercise-induced asthma. *Med Sci Sports Exerc*. 1993;25 (5):554–561.
12. American College of Sports Medicine. *Guidelines for Exercise Testing and Prescription*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.
13. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care*. 2011;34(suppl 1):S62–S69.
14. Nielsen P, Nachtigall D. Iron supplementation in athletes. Current recommendations. *Sports Med*. 1998;26(4):207–216.
15. Gardner RM, Hankinson JL. Standardization of spirometry: 1987ATS update. *J Occup Environ Med*. 1988;30(3):272–273.
16. Knudson RJ, Lebowitz MD, Holberg CJ, Burrows B. Changes in the normal maximal expiratory flow-volume curve with growth and aging. *Am Rev Respir Dis*. 1983;127:725–734.
17. Argyros GJ, Roach JM, Hurwitz KM, Eliasson H, Phillips YY. Eucapnic voluntary hyperventilation as a bronchoprovocation technique—a development of a standardized dosing schedule in asthmatics. *Chest*. 1996;109(6):1520–1524.
18. Anderson SD, Brannan JD. Methods for "indirect" challenge tests including exercise, eucapnic voluntary hyperpnea, and hypertonic aerosols. *Clin Rev Allergy Immunol*. 2003;24(1):27–54.
19. Juniper EF, Cockcroft DW, Kolendowicz R. *Histamine and Methacholine Inhalation Tests: Tidal Breathing Method; Laboratory Procedure and Standardization*. Lund, Sweden: Astra Draco AB; 1994.
20. World Anti-Doping Agency. Medical information to support the decision of TUECs Asthma. Montreal, QC, Canada: World Anti-Doping Agency; 2011.
21. Lougheed MD, Lemiere C, Dell SD, et al. Canadian Thoracic Society Asthma Management Continuum—2010 Consensus Summary for children six years of age and over, and adults. *Can Respir J*. 2010;17(1):15–24.
22. Myers J, Buchanan N, Smith D, et al. Individualized ramp treadmill. Observations on a new protocol. *Chest*. 1992;101(suppl 5):S236–S241.
23. Pescatello LS, Franklin BA, Fagard R, et al. American College of Sports Medicine position stand. Exercise and hypertension. *Med Sci Sports Exerc*. 2004;36(3):533–553.
24. Chobanian AV, Bakris GL, Black HR, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA*. 2003;289(19):2560–2572.
25. Corrado D, Pelliccia A, Bjornstad HH, et al. Cardiovascular preparticipation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol: consensus statement of the study group of sport cardiology of the working group of cardiac rehabilitation and exercise physiology and the working group of myocardial and pericardial diseases of the European Society of Cardiology. *Eur Heart J*. 2005;26(5):516–524.
26. Beckerman J, Wang P, Hlatky M. Cardiovascular screening of athletes. *Clin J Sport Med*. 2004;14(3):127–133.
27. Garrick JG. Preparticipation orthopedic screening evaluation. *Clin J Sport Med*. 2004;14(3):123–126.
28. McCrory P. Preparticipation assessment for head injury. *Clin J Sport Med*. 2004;14(3):139–144.
29. Fallon KE. Utility of hematological and iron-related screening in elite athletes. *Clin J Sport Med*. 2004;14(3):145–152.
30. Holzer K, Brukner P. Screening of athletes for exercise-induced bronchoconstriction. *Clin J Sport Med*. 2004;14(3):134–138.


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31. Rumball JS, Lebrun CM. Preparticipation physical examination—selected issues for the female athlete. *Clin J Sport Med*. 2004;14(3):153–160.
32. Manfreda J, Sears MR, Becklake MR, et al. Geographic and gender variability in the prevalence of bronchial responsiveness in Canada. *Chest*. 2004;125(5):1657–1664.
33. Langdeau JB, Turcotte H, Bowie DM, Jobin J, Desgagne P, Boulet LP. Airway hyperresponsiveness in elite athletes. *Am J Respir Crit Care Med*. 2000;161(5):1479–1484.
34. Rundell KW, Slee JB. Exercise and other indirect challenges to demonstrate asthma or exercise-induced bronchoconstriction in athletes. *J Allergy Clin Immunol*. 2008;122(2):238–246.
35. Parsons JP, Kaeding C, Phillips G, Jarjoura D, Wadley G, Mastronarde JG. Prevalence of exercise-induced bronchospasm in a cohort of varsity college athletes. *Med Sci Sports Exerc*. 2007;39(9):1487–1492.
36. Gibson GJ, Greenacre JK, Konig P, Conolly ME, Pride NB. Use of exercise challenge to investigate possible tolerance to beta-adrenoceptor stimulation in asthma. *Br J Dis Chest*. 1978;72(3):199–206.
37. Ramage L, Lipworth BJ, Ingram CG, Cree IA, Dhillon DP. Reduced protection sgainst exercise-induced bronchoconstriction after chronic dosing with salmeterol. *Respir Med*. 1994;88(5):363–368.
38. Inman MD, Obyrne PM. The effect of regular inhaled albuterol on exercise-induced bronchoconstriction. *Am J Respir Crit Care Med*. 1996;153(1):65–69.
39. Simons FER, Gerstner TV, Cheang MS. Tolerance to the bronchoprotective effect of salmeterol in adolescents with exercise-induced asthma using concurrent inhaled glucocorticoid treatment. *Pediatrics*. 1997;99(5):655–659.
40. Nelson JA, Strauss L, Skowronski M, Ciufo R, Novak R, McFadden ER. Effect of long-term salmeterol treatment on exercise-induced asthma. *N Engl J Med*. 1998;339(3):141–146.
41. Garcia R, Guerra P, Feo F, et al. Tachyphylaxis following regular use of formoterol in exercise-induced bronchospasm. *J Investig Allergol Clin Immunol*. 2001;11(3):176–182.
42. Hancox RJ, Subbarao P, Kamada D, Watson RM, Hargreave FE, Inman MD. B-2-agonist tolerance and exercise-induced bronchospasm. *Am J Respir Crit Care Med*. 2002;165(8):1068–1070.
43. Chan-Yeung M, Anthonisen NR, Becklake MR, et al. Geographical variations in the prevalence of atopic sensitization in six study sites across Canada. *Allergy*. 2010;65(11):1404–1413.
44. Katelaris CH, Carrozzi FM, Burke TV, Byth K. A springtime Olympics demands special consideration for allergic athletes. *J Allergy Clin Immunol*. 2000;106(2):260–266.
45. Katelaris CH, Carrozzi FM, Burke TV, Byth K. Patterns of allergic reactivity and disease in Olympic athletes. *Clin J Sport Med*. 2006;16(5):401–405.
46. Helenius IJ, Tikkanen HO, Sarna S, Haahtela T. Asthma and increased bronchial responsiveness in elite athletes: atopy and sport event as risk factors. *J Allergy Clin Immunol*. 1998;101(5):646–652.
47. Bousquet PJ, Chinn S, Janson C, Kogevinas M, Burney P, Jarvis D. Geographical variation in the prevalence of positive skin tests to environmental aeroallergens in the European Community Respiratory Health Survey I. *Allergy*. 2007;62:301–309.
48. Arbes SJ, Gergen PJ, Elliott L, Zeldin DC. Prevalences of positive skin test responses to 10 common allergens in the US population: Results from the Third National Health and Nutrition Examination Survey. *J Allergy Clin Immunol*. 2005;116(2):377–383.
49. Laprise C, Boulet LP, Morissette J, Winstall E, Raymond V. Evidence for association and linkage between atopy, airway hyper-responsiveness, and the beta subunit Glu237Gly variant of the high-affinity receptor for immunoglobulin E in the French-Canadian population. *Immunogenetics*. 2000;51(89):695–702.
50. Guerra S, Sherrill DL, Martinez FD, Barbee RA. Rhinitis as an independent risk factor for adult-onset asthma. *J Allergy Clin Immunol*. 2002;109(3):419–425.
51. Leddy JJ, Izzo J. Hypertension in athletes. *J Clin Hypertens (Greenwich)*. 2009;11(4):226–233.
52. Pelliccia A, Maron BJ, De Luca R, Di Paolo FM, Spataro A, Culasso F. Remodeling of left ventricular hypertrophy in elite athletes after long-term deconditioning. *Circulation*. 2002;105(8):944–949.
53. Dlin RA, Hanne N, Silverberg DS, Baror O. Follow-up of normotensive men with exaggerated blood-pressure response to exercise. *Am Heart J*. 1983;106(2):316–320.
54. Singh JP, Larson MG, Manolio TA, et al. Blood pressure response during treadmill testing as a risk factor for new-onset hypertension. The Framingham Heart Study. *Circulation*. 1999;99(14):1831–1836.
55. Matthews CE, Pate RR, Jackson KL, et al. Exaggerated blood pressure response to dynamic exercise and risk of future hypertension. *J Clin Epidemiol*. 1998;51(1):29–35.
56. Tanji JL. Tracking of elevated blood-pressure values in adolescent athletes at 1-year follow-up. *Am J Dis Child*. 1991;145(6):665–667.
57. Manolio TA, Burke GL, Savage PJ, Sidney S, Gardin JM, Oberman A. Exercise blood pressure response and 5-year risk of elevated blood pressure in a cohort of young ddults: the CARDIA study. *Am J Hypertens*. 1994;7(3):234–241.
58. Nakashima M, Miura K, Kido T, et al. Exercise blood pressure in young adults as a predictor of future blood pressure: a 12-year follow-up of medical school graduates. *J Hum Hypertens*. 2004;18(11):815–821.
59. Beard J, Tobin B. Iron status and exercise. *Am J Clin Nutr*. 2000;72(2 suppl):594S–597S.
60. Haas JD, Brownlie T. Iron deficiency and reduced work capacity: a critical review of the research to determine a causal relationship. *J Nutr*. 2001;131(2S-2):676S–688S.
61. Fogelholm M. Indicators of vitamin and mineral status in athletes blood: a review. *Int J Sport Nutr*. 1995;5(4):267–284.
62. La Société Québécoise D'hypertension Artérielle. *Hypertension: Guide thérapeutique*. 3rd ed. Montreal, QC, Canada: La Société Québécoise D'hypertension Artérielle; 2007.
63. American College of Sports Medicine. *Guidelines for Exercise Testing and prescription*. 7th ed. Baltimore, MD: Lippincott Williams & Wilkins; 2005.



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