



Exercise-induced hyperventilation: more common than appreciated

Introduction

Exercise-induced respiratory symptoms, especially in adolescents, are common and are often not caused by asthma.¹ Hyperventilation during exercise, breathing in excess of exercise metabolic requirements, is a cause of pseudoasthma,² usually erroneously attributed to exercise-induced bronchoconstriction (EIB). We recently treated an adolescent with exercise-induced hyperventilation and identified 11 more individuals in 18 months with similar clinical features.

A 15-year-old nonsmoking female student (Table 1, #1) had left home to attend ballet school 6 months before presenting to us. She developed breathlessness with strenuous exertion, difficulty getting air in, chest tightness, and associated lightheadedness, headaches, and weakness. Symptoms were nonresponsive to bronchodilators and inhaled corticosteroids. Voluntary hyperventilation while at school reproduced all her symptoms. Despite this, on return to Saskatoon, she was referred for management of refractory asthma. She had no rhinitis or other health problems, was using no medications, and had normal results of physical examination (oxygen saturation, 100%). Chest radiograph, pulmonary function including maximal inspiratory flows, and methacholine challenge³ (provocation concentration causing a 20% fall in FEV₁ [PC₂₀] > 16 mg/mL) were normal; she was non-atopic. Exercise testing failed to produce symptoms, bronchospasm or hyperventilation. A diagnosis of exercise-induced hyperventilation was made based on the history, the exclusion of asthma, and the hyperventilation response.

We subsequently over 18 months identified 11 young athletic individuals (10 female) with similar symptoms (Table 1, #2–12). All had breathlessness on strenuous exertion with a sensation of difficulty on inspiration and either syncope (n = 4) or pre-syncope (n = 8). None of the patients described stridor, none had gastroesophageal reflux disease symptoms, and none had any respiratory symptoms at any other times. Paresthesia accompanied the respiratory symptoms in 4. Three reported symptoms occurring only during competition and not during practice involving equivalent exercise. When the suspected diagnosis was discussed with these patients, none admitted to pre-exercise hyperventilation, a technique believed by some to preload oxygen stores to help improve performance.

Asthma was suspected in all; response to bronchodilators (n = 12) or inhaled corticosteroids (n = 6, Table 1) was absent (n = 9) or equivocal (n = 3). Rhinitis was absent except for 1 with mild perennial rhinitis and 1 with rhinitis on cat exposure. Resting oxygen saturations were uniformly high (98–100%). Three of these patients had elevated room air oxygen saturations (98–99%) in emergency rooms during attacks. Inspiratory and expiratory flows

were normal (Table 1), and methacholine PC₂₀ was normal (>8 mg/mL) in 8, and mildly (n = 1) to borderline (n = 3) reduced in 4. In 3 subjects with borderline airway hyper-responsiveness, the methacholine-induced symptoms did not mimic those that occurred with exertion. Exercise testing was done in 6; none developed EIB or hyperventilation, and symptoms were not reproduced. End-tidal partial pressure of carbon dioxide was low normal (30–35 mmHg) and did not fall after exercise. None of the additional 11 patients was challenged with voluntary hyperventilation.

We present 12 patients with non-asthmatic exercise-induced dyspnea. We suspected exercise hyperventilation but were unable to objectively confirm this in 11 of 12. The clinical clues suggesting hyperventilation include difficulty with inspiration, high resting oxygen saturation, absence of rhinitis or nocturnal symptoms, and poor response to bronchodilators. Paresthesia (n = 4) and syncope (n = 4)/presyncope (n = 8) are particularly suggestive of hyperventilation with acute respiratory alkalosis. Asthma was excluded by a normal methacholine test in most subjects, and in those who did respond to methacholine (mild or borderline) the methacholine-induced symptoms did not mimic the exercise symptoms. Possibly in this latter subset, minor bronchoconstriction might have contributed to the hyperventilation. Symptoms^{4,5} may be more accurate than the hyperventilation test⁶ in identifying hyperventilation. The gold standard for identifying exercise-induced hyperventilation would be an exercise test positive for hyperventilation with low end-tidal CO₂.² However, exercise under supervision may not reproduce symptoms or cause hyperventilation²; we suspect this may be similar to the failure to reproduce the symptom complex during practice compared with competition.

Intermittent laryngeal dysfunction during exercise could produce somewhat similar symptoms,^{1,7} particularly difficulty with inspiration. This could not be entirely excluded in our patients. None had stridor, inspiratory flows were normal, and none developed typical attacks, stridor, or decrease in inspiratory flows during methacholine (n = 12) or exercise (n = 6) testing. However, laryngeal dysfunction can be intermittent, and normal inspiratory flow when asymptomatic does not exclude this possibility; the major diagnostic tool, fiberoptic laryngoscopy during exercise, is not routinely available. Because our subjects did not develop symptoms or inspiratory flow limitation during exercise, laryngoscopy is unlikely to have been abnormal. None of the other differential diagnoses as outlined in the EIB practice parameter⁷ seems plausible in these individuals.

Treatment strategies include education regarding the mechanism of symptom generation secondary to hypocapnea and respiratory alkalosis and reassurance that there is no underlying organic cause of their symptoms. Breathing retraining has been suggested as a treatment for hyperventilation.⁸ This was attempted in 1 patient (#2) with limited success. Pharmacotherapy has limited value.

Disclosures: Authors have nothing to disclose.

Table 1
Clinical characteristics and laboratory data

#	Age sex	Activity	Difficulty inspiring	Presyncope (+) or syncope (++)	Paresthesia	Response to asthma therapy		Rhinitis	Resting oxygen saturation	FEV ₁ (%)	PIF (L/s)	Meth PC20 (mg/mL)	Exercise test	PetCO ₂	
						BD	ICS							Rest	Exercise
1	15F	Ballet	+	+	—	—	—	No	100	91	3.9	>16	Normal	34	36
2	16F	Track	+	++	—	±	—	No	98	94	4.2	6 ^a	Normal	34	35
3	15F	Taekwondo	+	++	+	—	—	No	99	116	5.3	12	Normal	36	37
4	14	basketball	+	++	—	—	—	No	100	98	3.7	1.3	ND	ND	ND
5	18F	Soccer	+	+	—	—	—	No	99	112	6.0	14	Normal	30	38
6	37F	Exercise	+	++	—	±	NT	No	98	121	3.0	>16	ND	ND	ND
7	26F	10 K run	+	+	—	—	NT	No	99	115	5.4	>16	ND	ND	ND
8	15F	Exercise	+	+	—	—	—	No	100	115	5.9	8	ND	ND	ND
9	23F	Swimming	+	+	—	—	NT	No	99	84	5.8	>16	ND	ND	ND
10	16F	Exercise	+	+	—	—	NT	mild	99	104	5.2	4 ^a	ND	ND	ND
11	14F	Track	+	+	—	—	—	Cat exposure	99	82	4.2	5 ^a	Normal	32	34
12	16M	Soccer basketball	+	+	—	±	—	No	98	100	6.9	>16	Normal	38	35
12	16M	Kayak	+	+	+	±	—	No	98	100	6.9	>16	Normal	38	35

Abbreviations: FEV₁, forced expired volume in 1 second (% predicted); PC₂₀, provocation concentration causing a 20% FEV₁ fall; PetCO₂, end-tidal partial pressure of CO₂ (mmHg); ND, not done; NT, not tried; PIF, peak inspiratory flow.

^aSymptoms induced by methacholine did NOT mimic those induced by exercise.

In summary, we were able to identify 12 individuals suspected of exercise-induced hyperventilation. This condition, and other nonasthmatic causes of exercise limitation (eg, laryngeal dysfunction), may be more common than is generally appreciated. We recommend that clinicians dealing with asthma or exercise keep this possibility in mind when dealing with exercise-induced breathlessness.

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