

## Toxic effects from nitrogen dioxide in ice-skating arenas

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**N**itrogen dioxide, a product of combustion in malfunctioning ice-resurfacing machines, can accumulate in indoor ice-skating arenas that are poorly ventilated. This hazard is rare but potentially lethal. We report a case of nitrogen dioxide poisoning in a hockey player and milder symptoms in other people in the arena.

### Case report

A 43-year-old man presented with a 3-day history of cough with blood-stained sputum, severe orthopnea and cyanosis. His symptoms had begun immediately after he had played in an ice-hockey tournament, peaked 48 hours later and began to lessen in another 48 hours.

The patient was afebrile and tachypneic. Crackles were heard in all lung fields. The findings of a cardiovascular examination were normal. The oxygen saturation in arterial blood ( $\text{SaO}_2$ ), with the patient breathing room air, was 84% and decreased to 75% after mild exertion. The blood pH was 7.45, and the partial pressures of oxygen and carbon dioxide were 46 and 37 mm Hg respectively.

An electrocardiogram appeared normal, and cardiac enzyme levels, the hemoglobin level and the leukocyte count were also normal. A chest radiograph showed bilateral centrifugal airspace disease; the cardiac silhouette was normal. Lung function tests revealed a diffusing capacity of 82% of the predicted norm.

The patient was erroneously treated for left ventricular failure with oxygen and diuretics; treat-

ment was continued with oxygen alone. Two days later his  $\text{SaO}_2$  was 95%, with no desaturation after mild exertion. A chest radiograph showed resolution of the airspace disease; 1 week later the diffusing capacity was 109% of the predicted norm.

Cough developed in all of the other hockey players, the two referees and the timekeeper after the tournament. Two players reported hemoptysis; one visited his physician, who reported that a chest radiograph appeared normal.

The ventilation fans in the arena were not in operation the day of the tournament despite hourly resurfacing for 16 hours. As well, the engine of the ice-resurfacing machine was not functioning properly. The referees reported a haze above the ice that increased in density during the day.

The air in the arena was tested with a gas-detector tube system (Gastec, Newark, Calif.) 30 days after the tournament. With both ventilation fans operating normally, a trace of nitrogen dioxide (less than 0.5 ppm [recommended maximum safe level 1 ppm<sup>1</sup>]) was detected in the air above the ice just after resurfacing, and 2 ppm was detected in the exhaust fumes of the resurfacing machine. The purpose of this measurement was to establish the presence and source of the nitrogen dioxide rather than the concentration during the tournament.

### Comments

The inhalation of toxic gases in arenas has been traditionally linked to carbon monoxide.<sup>2,3</sup> The substitution of propane for other fuels in resurfac-

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ing machines led to the expectation that the inhalation of toxic gases would be eliminated. However, nitrogen dioxide poisoning has emerged as a problem.<sup>2,4-6</sup>

People with carbon monoxide poisoning seek fresh air before the effects become too severe. The symptoms are well recognized, and the cause is detected early. With nitrogen dioxide poisoning the symptoms do not peak until 24 hours or more after exposure. There is no immediate irritation of the upper respiratory tract, especially with concentrations of less than 60 ppm; therefore, the gas can be inhaled for some time without distress.<sup>7</sup> The precise toxic level in humans varies with the concentration and the duration of exposure.<sup>7</sup> People with nitrogen dioxide poisoning present with the features of acute pulmonary edema.<sup>7-9</sup> The difficulty in recognizing the condition frequently results in a delayed or missed diagnosis.

Nitrogen dioxide is poorly soluble and affects mainly the peripheral airways and alveoli.<sup>7,8</sup> The inhaled gas combines with water in the lungs to produce nitrous and nitric acid.<sup>7</sup> The resultant chemical irritation increases vascular permeability in the lower airway mucosa and alveoli and causes bronchitis and pulmonary edema.<sup>8</sup>

In the case we have described, high concentrations of nitrogen dioxide were produced with the frequent resurfacing of the ice, and because the gas is heavier than air it settled directly above the ice. Our patient had the most severe symptoms because he had played at the end of the day and had skated longer and harder than the other players, therefore having had the highest minute ventilation.

Given the popularity of skating and ice hockey, many players, officials and even spectators may be exposed to nitrogen dioxide if the ventilation in arenas is inadequate. The same precautions against exposure to carbon monoxide — battery-powered machines, proper maintenance of engines and adequate ventilation in arenas<sup>2,3</sup> — are recommended to reduce the risk of nitrogen dioxide poisoning. Arenas should end hockey tournaments and close if the ventilation is inadequate and should make mandatory checks of resurfacing-machine exhaust and arena air quality to reduce the risk of accidental exposure. Such a policy is especially relevant to nitrogen dioxide poisoning because of its late-presenting clinical features compared with the early warning symptoms of carbon monoxide poisoning.

The few reports in the literature of nitrogen dioxide poisoning in arenas<sup>1,4,5</sup> likely underrepresent the problem, since the condition is poorly recognized. Given its potential lethal effects, nitrogen dioxide poisoning should be given wider publicity.

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ment of Medicine, and Mr. William L. Wright, senior public health inspector, Saskatoon Rural Health Region, for their technical assistance.

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