

CORRESPONDENCE



Heart Disease Deaths among Firefighters

TO THE EDITOR: It is somewhat surprising that the report by Kales and colleagues (March 22 issue)¹ on emergency duties and deaths from heart disease among firefighters in the United States does not cite the possible influence of carbon monoxide as a causative factor in deaths due to coronary heart disease among firefighters suppressing a fire. There is no doubt about the role of moderate and severe carbon monoxide intoxication in myocardial injury or long-term mortality of cardiac origin.² We performed a study that showed that even in the case of mild carbon monoxide intoxication (carboxyhemoglobin level, <25%), 4% of patients have ischemic changes.³ It has been reported that firefighters who are non-smokers and who do not use self-contained breathing apparatus correctly may have carboxyhemoglobin levels of up to 14%.⁴ Even when such apparatus are used correctly, the carboxyhemoglobin level can reach 9.1%.⁵ We suggest that the increase in cardiovascular demand during fire suppression reported by Kales and colleagues may be partially due, on the one hand, to elevated carboxyhemoglobin levels and, on the other hand, to the union of carbon monoxide with mitochondrial cytochrome oxidase, which directly interferes with cellular respiration.²

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moderate to severe carbon monoxide poisoning. *JAMA* 2006;295:398-402.

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TO THE EDITOR: In the absence of evidence, one wonders whether the task-related deaths from coronary heart disease reported by Kales et al. could be reduced by identifying firefighters at high risk with the use of exercise stress testing for those over 40 years of age, as recommended by the National Fire Protection Association.¹ Although ST-segment depression has poor sensitivity for identifying those at high risk, the Duke treadmill score, heart-rate recovery, functional capacity, chronotropic index, and presence or absence of ectopy after exercise stress testing can greatly

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Table 1. Tympanic and Sublingual Temperatures before and after Maximal, Symptom-Limited Treadmill Testing with Electrocardiography (Bruce Protocol).*

Length of Treadmill Test <i>minutes</i>	Tympanic Temperature			Sublingual Temperature		
	Before Test	At Completion of Test	6 Min after Completion of Test	Before Test	At Completion of Test	6 Min after Completion of Test
11.8±2.9	98.5±1.0	99.0±1.2	99.2±1.2	97.9±0.8	97.5±1.1†	98.3±0.7

* Plus–minus values are means ±SD. To convert values for degrees Fahrenheit to degrees Celsius, subtract 32 and multiply by 5/9.

† The transient fall in the temperature reflects exercise hyperpnea.

enhance diagnostic accuracy.^{2,3} However, typical exercise stress testing does not induce heat stress, which often occurs during fire suppression, including that imposed by thermally restrictive protective gear. In an observational study of 61 healthy candidates for hazardous materials duty (mean [±SD] age, 34±8 years; mean body-mass index [the weight in kilograms divided by the square of the height in meters], 28.5±4.3), we found little sweating, subjective thermal stress, or change in body temperature (Table 1), which rose by only 0.06±0.07°F (0.03±0.04°C) per minute of treadmill exertion. If treadmill testing is performed as part of the medical evaluation of firefighters, it should incorporate heat stress as well as exertion.

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THE AUTHORS REPLY: We agree with Dueñas-Laita and colleagues that carbon monoxide is a likely contributing factor in some deaths from coronary heart disease that occur during fire suppression. Smoke exposure may occur at structure fires, despite the use of self-contained breathing apparatus, and during brush and forest fires, when little or no respiratory protection may be worn. Of 17 firefighters who died from coronary heart disease and for whom data on postincident carboxyhemoglobin levels could be determined, 4 (24%)

had carboxyhemoglobin levels of 3 to 10%.¹ Post-incident determinations of carboxyhemoglobin levels usually underestimate peak exposures because of oxygen therapy and the time that has elapsed between the incident and blood sampling. In addition, we found that more than 40% of on-duty firefighters who died from coronary heart disease were smokers.¹ Exposure to carbon monoxide from tobacco use is additive to that from exogenous smoke. Increased blood carboxyhemoglobin levels are expected to have an adverse effect on aerobic capacity, anaerobic threshold, and exercise tolerance. Finally, smoke from a fire may contain other cardiotoxins, such as cyanide and particulate matter.^{2,3} However, the important message — and the common denominator — of the deaths in our study is that various stressors (physical, psychological, and chemical) in different combinations can trigger cardiac events in on-duty firefighters who have underlying coronary heart disease.

We strongly agree with Raymond and colleagues that exercise stress testing should be useful in identifying firefighters at high risk for on-duty cardiovascular events. Likewise, we concur that including abnormal heart-rate recovery, chronotropic incompetence, certain abnormal blood-pressure responses, and dysrhythmias as criteria for an abnormal result on exercise stress testing would probably increase the sensitivity of the procedure. However, the results of such tests must be interpreted along with a comprehensive risk-factor profile for coronary heart disease⁴ to enhance their predictive value. Moreover, given the lack of direct evidence to support exercise stress testing in screening public safety personnel,⁵ it is premature to conclude that exercise stress testing in firefighters should include induction of exogenous heat stress. Further research conducted in public-safety cohorts is necessary to determine

the best risk-stratification strategies to use in the selection of firefighters for such testing and to develop effective screening and diagnostic protocols.

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Vancomycin-Induced Immune Thrombocytopenia

TO THE EDITOR: In the report by Von Drygalski and colleagues on vancomycin-induced immune thrombocytopenia (March 1 issue),¹ the positive human platelet factor 4 enzyme-linked immunosorbent assay (PF4 ELISA) was used to rule out the diagnosis of heparin-induced thrombocytopenia. Although this test is highly sensitive, it has been recommended² that the test not be used alone but be combined with a functional assay, such as a ¹⁴C-serotonin release assay, which is the gold standard. Furthermore, the authors report that platelet counts increased after the withdrawal of vancomycin. However, in the patient with vancomycin-induced thrombocytopenia whose platelet counts are shown in Figure 1 of the article, intravenous immune globulin was administered, and her platelet count rose 3 days later. The timing of the administration of immune globulin was too close to the withdrawal of vancomycin to conclude that vancomycin was the only cause of the thrombocytopenia.

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TO THE EDITOR: Von Drygalski et al. report convincing data demonstrating that vancomycin-depen-

dent antiplatelet antibodies cause vancomycin-induced immune thrombocytopenia. The authors observed that in most patients platelet levels returned to baseline after vancomycin was discontinued. However, they do not present data showing how long vancomycin-dependent antiplatelet antibodies remained in serum after vancomycin was discontinued. Is the persistence of vancomycin necessary for the maintenance of these antibodies?

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TO THE EDITOR: Von Drygalski et al. report that vancomycin-induced immune thrombocytopenia caused severe bleeding in 34% of patients (10 of the 29 for whom clinical follow-up information was obtained). Did the authors observe an increased incidence of venous thromboembolic or ischemic events in the patients?

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THE AUTHORS REPLY: In response to Panesar, although a positive result on PF4 ELISA is not specific for the clinical diagnosis of heparin-induced