ARTICLE

Cardiovascular Strain of Firefighting and the Risk of Sudden Cardiac Events

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[1]Health and Exercise Sciences, Skidmore College, Saratoga Springs, NY; [2]University of Illinois Fire Service Institute, Champaign, IL; [3]Department of Environmental Health, Harvard TH Chan School of Public Health, Boston, MA; [4]Occupational Medicine, Cambridge Health Alliance/Harvard Medical School, Cambridge, MA

SMITH, D.L., J.P. DEBLOIS, S.N. KALES, and G.P. HORN. Cardiovascular strain of firefighting and the risk of sudden cardiac events. *Exerc. Sport Sci. Rev.*, Vol. 44, No. 3, pp. 90–97, 2016. *Approximately 45% to 50% of line-of-duty deaths in the fire service are caused by sudden cardiac deaths, which most often occur during or shortly after firefighting duties. We present a theoretical model linking the cardiac, vascular, and hematological responses of firefighting to the triggering of sudden cardiac death in susceptible individuals.*
Key Words: firefighting, cardiac, vascular, coagulatory, cardiovascular disease, sudden cardiac death.

Key Points

- The leading cause of duty-related death within the fire service is a sudden cardiac event, accounting for approximately 50% of line-of-duty deaths.
- Sudden cardiac events are much more likely to occur after firefighting activities than other duties and may be the result of primary arrhythmias or myocardial infarction.
- Firefighting leads to significant cardiovascular strain, including alterations in cardiac function, vascular function, and hemostasis.
- Most firefighters recover from the stress of firefighting without incident. However, the cardiovascular strain of firefighting may trigger a cardiovascular event in firefighters with an underlying disease.
- Increased cardiac work, vascular dysfunction, tissue ischemia, and a procoagulatory state may be important causal links that increase the risk of sudden cardiac events in the vulnerable firefighter.

Club

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INTRODUCTION

Firefighters respond to multiple types of emergencies, such as fires, vehicle/machinery accidents, medical calls, calls for public assistance, technical rescue, and hazardous materials spills to protect the communities they serve. However, statistics show that firefighting (*i.e.*, those activities directly related to fire suppression) results in the greatest risk of injury or fatality (8). Firefighting activities often are conducted in immediately dangerous to life or health conditions that may expose firefighters to extreme environmental temperatures and to multiple chemical and particulate hazards. These conditions necessitate that firefighters wear heavy, insulated, and restrictive personal protective equipment (PPE). Hence, it comes as no surprise that firefighting results in high levels of cardiovascular strain.

Fire service statistics reveal that despite all the acute traumatic risks that firefighters face (*e.g.*, burn injuries, smoke inhalation, structural collapse), by far, the leading cause of line-of-duty death is cardiac related. In fact, approximately 45% to 50% of all firefighter duty–related fatalities are caused by sudden cardiac death (SCD) — a proportion that is relatively stable and stubbornly high. This point is highlighted in Figure 1, which compares SCD with fatalities from burns and asphyxiation since 1990. The number of cardiac fatalities seems to be trending downward during the past 10 years. This encouraging trend may be caused by efforts by the International Association of Firefighters, the International Association of Fire Chiefs, and the National Volunteer Fire Council, all of which have undertaken rigorous campaigns to increase medical evaluations and to promote firefighter wellness and fitness. However, as the numbers indicate, considerable work remains to further reduce cardiac fatalities in the US Fire Service (Fig. 1).

Importantly, sudden cardiac events are disproportionately more likely to occur during or after a firefighting activity than other duties. Although firefighters spend a small percentage of their time (1%–5%) engaged in fire suppression activities, more

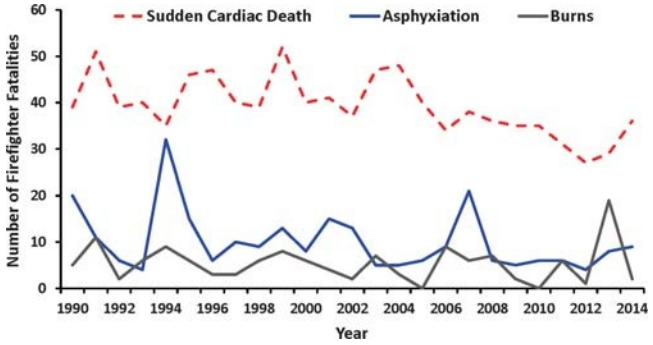

Figure 1. Line-of-duty deaths by major cause since 1990.

than 30% of cardiac fatalities occur during or shortly after firefighting activity, resulting in a 10 to 100 times greater risk of sudden cardiac events after firefighting versus station duties (19). Furthermore, there are approximately 17 to 25 duty-related nonfatal cardiovascular events (heart attacks and strokes) for every fatal event (8,15).

Cardiac events are devastating for individual firefighters and their families, but because firefighting relies on a coordinated team effort, duty-related cardiac events also can jeopardize job performance and the safety of other firefighters and may compromise the ability of firefighters to protect civilians during emergencies. Cardiac injuries likewise often will require significant time away from the fire department at significant cost and burden to the local fire department. Hence, there is an important public safety concern about cardiovascular events in the fire service. We recently have published a review that proposed a theoretical model highlighting the interaction of occupational, medical, and behavioral risk factors in contributing to underlying cardiovascular disease (CVD) (coronary heart disease (CHD) and/or structural heart disease) and where the strenuous duties of firefighting may trigger a cardiovascular event (Fig. 2) (32). In the current review, we extend the previous model by detailing results of translational research documenting the cardiovascular strain of firefighting and further highlighting the potential mechanisms by which cardiovascular responses to firefighting may lead to pathophysiologic changes that can trigger fatal arrhythmias or myocardial infarction, thus leading to a sudden cardiac event (Fig. 3).

FACTORS AFFECTING THE CARDIOVASCULAR STRAIN OF FIREFIGHTING

Firefighting involves performing a complex series of physically demanding tasks under psychologically stressful conditions within arduous environments. As depicted in Figure 3, the cardiovascular strain of firefighting results from multiple interacting factors including 1) sympathetic nervous system activation; 2) the strenuous physical work (aerobic and anaerobic); and 3) exposure to environmental conditions and pollutants contained in fire smoke.

Sympathetic Nervous System Activation

Sympathetic nervous activation begins with the alarm and continues throughout a fire call. The sudden sounding of an alarm increases psychological/mental stress, activating the sympathetic nervous system. While on the call, sympathetic activation continues because the fire scene is a dynamic cluttered environment in which fire behavior can change rapidly and where loud noises, time urgency, and potential danger produce powerful sympathetic arousal. Several studies have shown a fivefold to sevenfold risk of sudden cardiac events *during alarm response* versus nonemergencies (19,20). The increased relative risk of a cardiac event during this period can be attributed largely to sympathetic arousal and a putative surge in catecholamines because there is not yet exposure to the fire environment or the requirements for large amounts of physical work.

Physical Work

Structural firefighting requires a combination of static work and aerobic exertion, such as stair and ladder climbing (while

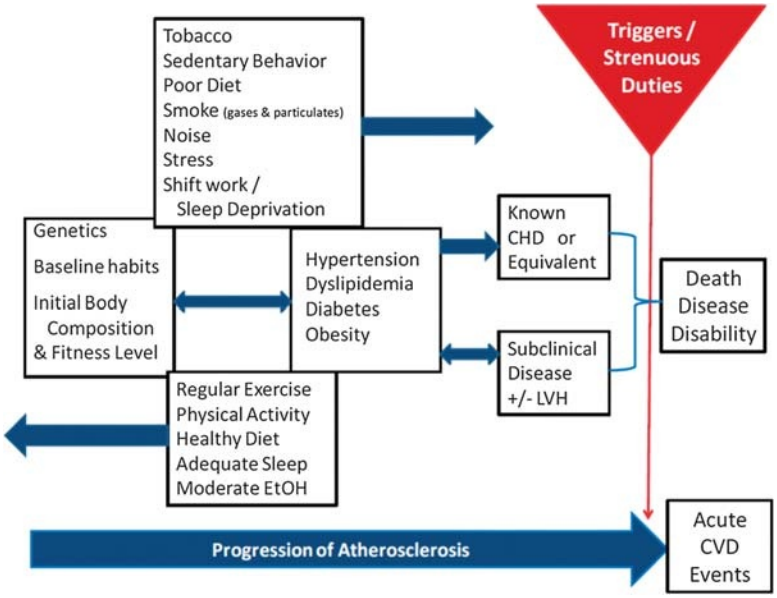

Figure 2. Theoretical model of occupational, medical and behavioral risk factors that contribute to sudden cardiac events following firefighting activities. Note that there is overlap between occupational and lifestyle risk factors. Arrows indicate that factors can be associated with progression or regression of risk factors and subclinical disease. EtOH, alcohol; CHD, coronary heart disease; LVH, left ventricular hypertrophy; CVD, cardiovascular disease. (Reprinted from (32). Copyright © 2011 Wolters Kluwer Health. Used with permission.)

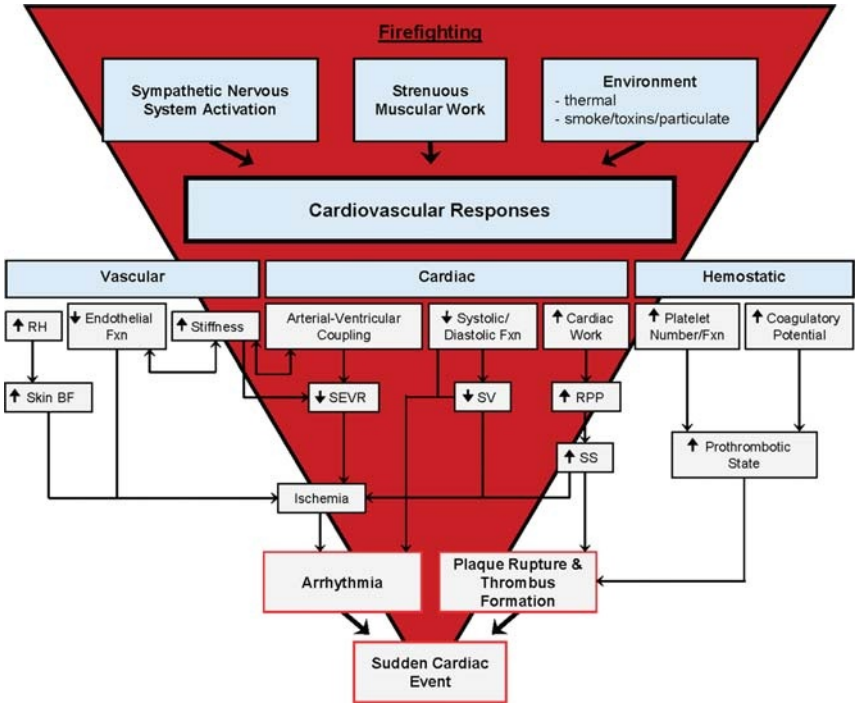

Figure 3. Theoretical interplay between cardiac, vascular, and hemostatic responses to firefighting and sudden cardiac events in susceptible individuals. RH, reactive hyperemia; BF, blood flow; Fxn, function; SEVR, sub-endocardial viability ratio; SV, stroke volume; RPP, rate pressure product; SS, shear stress.

carrying heavy equipment), forcible entry, victim search and rescue, building ventilation, and fire attack and suppression. Although it is known that different firefighting tasks require different levels of energy expenditure, firefighting often results in oxygen consumption of greater than of 40 $mL \cdot kg^{-1} \cdot min^{-1}$ (24). The strenuous work of firefighting is performed while wearing heavy (≥25 kg) and fully encapsulating PPE that provides range-of-motion restrictions, heavy insulation, and limited breathability and adds to the metabolic demands of firefighting. In addition, firefighters perform a great deal of upper body work (lifting and carrying heavy equipment, hose movement and control, chopping, ceiling overhaul), and upper body work (such as wood chopping or snow shoveling) leads to an exaggerated blood pressure response and an increased cardiac work and is associated with an increased risk of a sudden cardiac event (12).

Environmental Conditions

Radiant heat from a fire adds considerably to the heat stress experienced by firefighters. We have reported an increase in core temperature (T_{co}) of approximately 1.5°C after short-term (≤20 min) firefighting activity (30), and the rate of T_{co} change increased with subsequent firefighting activities (17). Both heat stress and dehydration exacerbate cardiovascular strain associated with heavy work (9).

Although firefighters regularly use self-contained breathing apparatus to protect their airways while operating inside the structure, firefighters routinely are exposed to fire smoke (outside of the structure and during overhaul/cleanup operations), which contains toxic and asphyxiant gases such as carbon monoxide, hydrogen cyanide, hydrogen sulfide, and particulate matter among a myriad of other chemicals (5). When inhaled, carbon monoxide reduces the availability of oxygen, thus, potentially resulting in tissue hypoxia. Tissue hypoxemia caused by carbon monoxide and other asphyxiants (*e.g.*, hydrogen cyanide, hydrogen sulfide) may lead to myocardial ischemia in susceptible individuals. Fire smoke also contains particulate matter associated with the promotion of arrhythmias, decreased heart rate variability, and increased blood pressure (23). Such particulates also may increase the formation of free radicals leading to activation of proinflammatory and prothrombotic pathways that may cause endothelial dysfunction and increased blood coagulability.

CARDIOVASCULAR RESPONSES TO FIREFIGHTING

Firefighting operations are complex, and no fire scene is identical to another. The magnitude of the cardiovascular strain is affected by the fuel that is burning (*e.g.*, heat release rate, products of combustion), the size of the fire (amount of fuel involvement), dimensions and layout of the structure, the resources (human and equipment) that are available to fight the fire, the tactics that are used (*e.g.*, interior, exterior, or combination attack), and the firefighter's assigned task. For example, a firefighter who is responsible for operating the pump on the engine at a small fire would have a different level of cardiac strain than a firefighter who is performing search and rescue activities on the interior of a large structure during fire suppression activities. The following sections detail research efforts to characterize systematically the effects of firefighting on the various components of the cardiovascular system — the heart, vasculature, and hemostatic system.

Cardiac Responses

During strenuous fire suppression activities, heart rates (HR) increase and rise to maximal or near maximal levels and T_{co} increases rapidly (18,29). Studies that have focused on long-term firefighting activities that require more than 1 cylinder of air (supporting heavy firefighting work for approximately 12–16 min) have shown that the rate of increase in T_{co} is

augmented in later bouts of activity, further exacerbating cardiovascular strain and leading to higher HR (17). HR responses to firefighting vary tremendously depending on the type of work the firefighter is doing and multiple other factors (ambient temperature, length of time engaged, fitness level, etc.). Furthermore, firefighters' HR vary throughout a given emergency because of the intermittent nature of the work (17).

Although HR is the most frequently documented cardiovascular variable because of its ease of measurement, alterations in other cardiac variables are likely even more important to describe the cardiovascular risk associated with firefighting. We have reported a 35% reduction in stroke volume (seated position) after three short bouts of strenuous firefighting activity that lasted a total of approximately 20 min (29). More recently, we performed a full echocardiographic examination (supine position) before and after a 3-h training period that included multiple training evolutions/drills, each lasting approximately 15 to 30 min (11). The firefighting training resulted in near maximal HR (mean, 192 ± 15 beats min^{-1}) and an increase in T_{co} from 37.1 ± 0.5°C to 38.9 ± 0.6°C. Echocardiographic measurements were obtained within 30 min of firefighting, and mean HR had decreased to 90 ± 13 beats min^{-1} at the time of measurement. In this study, we found a 13% reduction in stroke volume (11). Furthermore, the repeated bouts of training resulted in significant reductions in left ventricular diastolic size and volume, transmitral flow velocities, mitral E/A (the ratio of passive ventricular filling to active atrial filling of the ventricle), and left ventricular shortening fraction and ejection fraction. The changes in left ventricular function observed in this study may reflect changes in preload after the firefighting activity. However, there is some evidence that intrinsic systolic and diastolic function also may be depressed after firefighting activity. TDI-E′, an indicator of rate of ventricular relaxation, decreased by 19% when measured at the lateral wall but was unchanged when measured at the septal wall (11). The decline in the lateral wall TDI-E′ is a marker of lusitropic function that is less load dependent than other measures of diastolic function. Although the clinical significance of the cardiac changes presented above are not fully comprehended, it is important to consider these changes in the context of overall cardiovascular function, especially given that these results were noted in apparently healthy firefighters with no known CVD. Indeed, this study found that, in addition to decreased left ventricular function, there was a decrease in systemic arterial compliance (11). These findings raise the possibility that arterial-ventricular coupling may be altered with firefighting and reinforces the importance of investigating vascular function after firefighting.

Vascular Responses

The myocardium and vasculature work together in a concerted effort to deliver blood to body tissues. Changes in vascular function and/or the interaction between the myocardium and the vasculature may lead to tissue ischemia/hypoxia, precipitating a sudden cardiac event. We have conducted several studies to investigate the effects of firefighting activities and heat stress on the vasculature and its function.

Yan *et al.* (35) reported changes in arterial-ventricular coupling after repeated bouts of firefighting during a 3-h period of live-fire training in a group of young (28 ± 1 yr) firefighters. Arterial-ventricular coupling was measured on the right common carotid artery using wave intensity analysis, a hemodynamic index in which changes in blood pressure and blood flow provide insights into cardiovascular stress (25). In response to live-fire training, wave 1 amplitude, an indicator of cardiac contractility, was reduced by approximately 28%, suggesting that systolic function decreased (35). In a laboratory study designed to further investigate the role of heat stress in changing arterial-ventricular coupling, Smith *et al.* (26) examined 11 young (22 ± 3 yr) men before and after a 100-min exercise/rest treadmill task (alternating 20-min walk/20-min rest in structural firefighting PPE) and found a nearly 32% increase in wave 2 amplitude. This measure of end-systolic/early-diastolic ventricular function from wave intensity analysis indicates increased afterload on the myocardium after heat stress (26).

We also have examined the influence of firefighting on vascular structure and function. Fahs *et al.* (6) examined the acute effects of live-fire training drills on arterial stiffness and limb blood flow in a group of 69 male firefighters (mean age, 28 ± 1 yr). After 3 h of live-fire training, hemodynamic proxies (wave reflection time and augmentation index) influenced by aortic stiffness increased by approximately 3%. Despite a possible increase in aortic stiffness, a concomitant increase in peripheral forearm vascular conductance (80%) and hyperemia (32%) also were observed (6). It is suggested that the unique combination of thermal, metabolic, psychological, and mental stress that occurs with firefighting activities might explain the increased aortic stiffness and forearm blood flow.

As a follow-up to live-fire studies, Lefferts *et al.* (22) conducted a laboratory-based study to isolate the influence of thermal stress on vascular and central hemodynamic changes. Controlling for hydration status, Lefferts *et al.* (22) reported that exercise-induced moderate heat stress (T_{co} of 37.8 ± 0.1°C caused by treadmill walking in firefighting PPE) did not change aortic stiffness (pulse wave velocity, pre: 5.0 ± 0.1 $m \cdot s^{-1}$ vs post: 4.9 ± 0.1 $m \cdot s^{-1}$; $P = 0.698$). However, Lefferts *et al.* (22) did find alterations in myocardial work demands (rate-pressure product increased by 37%) and an indirect proxy of coronary perfusion (subendocardial viability ratio (SEVR) reduced by about 27%). After a short 18-min bout of live-fire activity, Horn *et al.* (18) found a larger increase in rate-pressure product (80%–95%) and similar reductions in SEVR (30%–35%). These results, although they must be interpreted cautiously given limitations with SEVR, suggest a potential mismatch between myocardial oxygen demand and supply, which may result in ischemia during strenuous work in the heat. This potential mismatch may be more pronounced after firefighting activities than exercise-induced heat stress.

Thus, firefighting activity and heat stress may result in vascular dysfunction and arterial-ventricular uncoupling as evidenced by increased central arterial stiffness, decreased vascular function, and a myocardial oxygen supply-demand mismatch. A potential linkage between vascular alterations and the risk of SCD may be related to firefighters' individual characteristics. We have found increased arterial stiffness in a group of obese (≥29.5 $kg \cdot m^{-2}$) and overweight (25.9–29.4 $kg \cdot m^{-2}$) firefighters compared with lean (<25.9 $kg \cdot m^{-2}$) firefighters, where carotid artery β stiffness was greatest in the obese group compared with the overweight and lean groups (5.9 ± 0.3, 5.1 ± 0.3, and 4.9 ± 0.3, respectively) (7). Furthermore, we also have examined vascular changes in older firefighters (40–60 yr) before and after

firefighting activity and found that reactive hyperemia, a measure of microvascular blood flow, and pressure-controlled arterial stiffness (central pulse wave velocity/aMAP) increased significantly after firefighting (21). The cause of increased vascular stiffness after firefighting is not known. The increased central vascular stiffness observed after firefighting activities could be a compensatory mechanism to counteract the profound skin vasodilation in an attempt to facilitate blood flow to the heart and brain or it could reflect a detrimental response to the stressors encountered during firefighting. Additional studies are necessary to better understand the cause and consequences of arterial stiffness and to elucidate what populations of firefighters who, through underlying disease or CVD risk factors, may have a greater risk of experiencing acute vascular changes that may result in SCD.

Because changes in vascular function may be caused by reactive oxygen species or prostaglandin production, we had conducted studies that used antioxidant supplementation or aspirin. We have investigated the effect of vitamin C supplementation on vascular responses to firefighting in young healthy firefighters and found that 2 g of vitamin C did not affect measures of vascular stiffness or microvascular function (6). We also have investigated the effect of aspirin supplementation on vascular changes associated with firefighting in older subjects and found that 2 wk of aspirin supplementation did not affect microvascular (reactive hyperemia) or macrovascular (arterial stiffness/central blood pressure) responses to firefighting (21).

Collectively, research has shown that firefighting and exercise-induced heat stress lead to vascular alterations, specifically, reduced vascular reactivity/endothelial function (6) and increased central arterial stiffness (7,22), which may play roles in arterial-ventricular uncoupling in response to firefighting activity and heat stress (26,35). The uncoupling of the arterial-ventricular system may explain the oxygen supply-demand mismatch observed after firefighting and exercise-induced heat stress (22), leading to ischemia. Increased arterial stiffness after firefighting may be detrimental because of increased cardiac work (increased afterload) or impaired vascular function (compromised vasodilation), and this effect is likely more pronounced in individuals with underlying CVD. Furthermore, overweight/obese and older firefighters may be at increased risk for abnormal vascular responses associated with firefighting and heat stress because the vasculature of these persons is stiffer at rest without the external influence of occupational stressors (7).

Hemostatic Responses

Firefighting leads to an increase in blood pressure and sweating. Accordingly, we have documented a decrease of approximately 15% in plasma volume after an 18-min bout of firefighting (30). In turn, this resultant hypovolemia decreases central venous pressure, causes hemoconcentration, and increases blood viscosity.

Firefighting likewise seems to disrupt hemostatic balance. We conducted a large study (N = 114) to examine the acute effects of firefighting on platelet number and function. Live-fire firefighting drills (~18 min) caused a modest change in T_{co} (0.7°C) and a peak HR of 167 beats min^{-1} and resulted in increased platelet number and decreased platelet closure time (increased aggregation) in young (29 ± 8 yr) apparently healthy firefighters (31). In a follow-up study, we evaluated the effects of firefighting activities on platelets, coagulation, and fibrinolytic activity and documented the extent to which these variables recovered 2 h after completion of the firefighting activity in young (25 ± 5 yr) apparently healthy firefighters (27). Platelet number, platelet activity, and coagulatory potential increased immediately after firefighting, and many variables (platelet function, activated partial thromboplastin time (aPTT) and factor VII) continued to reflect a procoagulatory state even after 2 h of recovery. Fibrinolysis also was enhanced immediately after firefighting but returned to baseline values 2 h after firefighting. Research with athletes has similarly indicated that strenuous physical activity acutely increases platelet number and platelet activation (4) and leads to a hypercoaguable state that is normally offset by simultaneous increases in fibrinolysis (16). This hypercoaguable state persists longer into the postactivity recovery period than does the increase in fibrinolysis, potentially reflecting a "vulnerable period" after strenuous exercise (16). In situations of abnormal hemostatic balance, the exercise-induced procoagulatory state may result in increased incidence in cardiovascular events during and immediately after strenuous physical activity. These data support the hypothesis that firefighting leads to a hemostatic imbalance that is primarily prothrombotic during the recovery period from firefighting activities.

INDIVIDUAL CHARACTERISTICS AND UNDERLYING CVD RISK

As we have summarized in a previous review (32), individual factors may mediate the magnitude of the cardiovascular strain of firefighting. Although firefighting results in significant cardiovascular strain, most firefighters recover from firefighting activities with no untoward event. However, the cardiovascular strain of firefighting interacts with an individual's health and fitness status to determine whether responses to the strain of firefighting are limited to transient physiologic disruptions or result in the activation of pathophysiologic pathways that may lead to sudden cardiac events (32). Underlying cardiovascular health status is determined largely by the prevalence of cardiovascular risk factors. As shown in the Table, such risk factors greatly increase the risk of cardiac death in the fire service, with smoking, hypertension, diabetes, and old age all associated with a greatly elevated risk. However, the greatest risk was associated with previous CHD diagnosis (32). In addition, low fitness also likely contributes to the increased risk of sudden cardiac events (2) because fit individuals can do more work at the same level of cardiovascular strain or they experience less strain at the same level of work. Unfortunately, there is substantial evidence that firefighters often lack high levels of fitness and a large pecentage are overweight or obese (2,7,32).

A potential linkage between vascular alterations and the risk of SCD also may be related to individual factors, including obesity. Obesity (body mass index (BMI), ≥30 $kg \cdot m^{-2}$) is a global epidemic affecting all members of society, including first responders. In the fire service, obesity has been found to increase the relative risk of an on-duty coronary heart disease fatality threefold (32). Obesity is a well-established risk factor for CVD and has been associated with reduced arterial function and increased aortic blood pressures (10). We examined the influence of body weight on vascular structure and function in a group of 110 firefighters (30 ± 8 yr). Firefighters were evenly

TABLE. Relative risk of cardiovascular outcome by risk factor in firefighters

	On-Duty CHD Fatalities, OR (95% CI) (20)	Non-CHD Cardiovascular Retirements, OR (95% CI) (16)	CHD Retirements, OR (95% CI) (16)
Current smoking	8.6 (4.2–17)	2.5 (1.2–5.1)	3.9 (2.5–6.2)
Hypertension	12 (5.8–25)	11 (6.1–20)	5.4 (3.7–7.9)
Obesity, BMI ≥30 $kg \cdot m^{-2}$	3.1 (1.5–6.6)	3.6 (2.0–6.4)	1.4 (0.96–1.93)
Cholesterol ≥5.18 $mmol \cdot L^{-1}$ (200 $mg \cdot dL^{-1}$)	4.4 (1.5–13)	1.1 (0.51–2.24)	2.4 (1.6–3.6)
Diabetes mellitus	10.2 (3.7–28)	7.7 (2.9–20)	13 (6.1–28)
Prior diagnosis of CHD	35 (9.5–128)	NA	30 (9.1–96)
Age ≥45 yr	18 (8.5–40)	26 (13–51)	63 (35–111)

BMI, body mass index; CHD, coronary heart disease; CI, confidence interval; diabetes mellitus, defined as random blood glucose level more than 8.3 $mmol \cdot L^{-1}$ (150 $mg \cdot dL^{-1}$), previous diagnosis, or receiving insulin or hypoglycemic medications; hypertension, defined as resting blood pressure of 140/90 mm Hg or higher, previous diagnosis of hypertension, or receiving antihypertensive therapy; OR, odds ratio. (Reprinted from (32). Copyright © 2011 Wolters Kluwer Health. Used with permission.)

divided into tertiles according to BMI (<25.9, 25.9–29.4, and ≥29.5 $kg \cdot m^{-2}$). We found increased arterial stiffness in the obese (≥29.5 $kg \cdot m^{-2}$) and overweight (25.9–29.4 $kg \cdot m^{-2}$) firefighters compared with that in lean (<25.9 $kg \cdot m^{-2}$) firefighters (7). Furthermore, carotid artery β stiffness was greatest in the obese group compared with the overweight and lean groups (5.9 ± 0.3, 5.1 ± 0.3, and 4.9 ± 0.3, respectively). However, measures of endothelial function did not differ by BMI classification (7).

We also have investigated the effect of obesity on coagulatory response to acute firefighting activity (28). An 18-min bout of live-fire training caused a significant increase in coagulatory and fibrinolytic markers, including an overall shift toward a procoagulatory state, as evidenced by a decrease in aPTT and an increase in platelet activity. We also found that obese firefighters exhibited lower baseline levels of fibrinolytic activity (lower tissue plasminogen activator and higher plasminogen activator inhibitor-1), which is consistent with previous literature. However, contrary to our hypothesis, we did not detect an increase in baseline coagulatory measures in obese firefighters nor did we find a greater coagulatory response among obese firefighters. Additional research is necessary to better understand how age, body composition, fitness, and cardiovascular risk factors affect vascular-hemostatic balance after firefighting because it is known that the vascular-hemostatic responses to exercise differ between healthy individuals and those with underlying CVD or CVD risk factors (34).

TRIGGERING A CARDIOVASCULAR EVENT

Atherosclerosis and structural heart changes, such as left ventricular hypertrophy (LVH), generally develop during a period of many years — even decades. Although CVD may progress with or without symptoms for many years, a plaque rupture or arrhythmia can lead to the rapid onset of severe symptoms and even SCD. Understanding what precipitates, or triggers, such an event is of interest to researchers, clinicians, and those involved in health policy.

Classic studies have temporally linked heavy physical exertion/strenuous work (such as running or snow shoveling) to the onset of acute cardiovascular events, with the risk being concentrated among individuals who are unaccustomed to such levels of exertion (1,12). Sympathetic nervous activation caused by emotional stress, such as excitement and frustration/anger, also is associated with triggering cardiovascular events in individuals with known CHD (32). In addition, environmental conditions such as elevated levels of air pollution have been shown to be associated with increased rates of sudden cardiac events among susceptible individuals (23).

Although a series of complex pathophysiological processes are involved in SCD, pump failure usually caused by terminal cardiac arrhythmias is ultimately the cause of death. Terminal arrhythmias may be caused by a primary electrical conduction problem (primary arrhythmia) or as complications of plaque rupture and thrombus formation that causes a myocardial infarction. SCD can be caused by many underlying conditions; however, a very large percentage of SCD is caused by CHD (*i.e.*, atherosclerosis) and/or cardiomegaly/LVH (13,36).

Firefighting as a Trigger for Sudden Cardiac Events

The work of a firefighter is characterized by long periods of low-intensity work, such as inspection, chores, and public education, unpredictably punctuated by episodes of strenuous work. During these intense intervals, firefighting activity involves heavy muscular work and requires high levels of oxygen consumption. Thus, during firefighting, multiple stressors may function independently or more likely synergistically to precipitate acute CVD events among susceptible firefighters (32).

Multiple studies have provided compelling evidence that firefighting activities can trigger cardiovascular events in susceptible firefighters (19,20). These studies found highly elevated and remarkably consistent odds for SCD and other acute CVD events during emergency firefighting activities compared with nonemergency duties. The largest of these studies (19) investigated line-of-duty deaths (n = 449) attributed to CHD between 1994 and 2004 and found that, although firefighting (*i.e.*, active fire suppression) represents between 1% and 5% of a firefighter's annual working time, firefighting accounted for more than 30% of line-of-duty CHD deaths. This resulted in a relative risk of SCD during fire suppression of roughly 10 to 100 times the risk encountered during nonemergency duties (19).

The aforementioned physiological disruption and cardiac strain associated with firefighting do not normally pose a significant risk in healthy individuals. However, in susceptible individuals, the stress of firefighting can serve as a trigger for SCD. Most sudden cardiac events are likely caused by myocardial infarctions in individuals with CHD or fatal arrhythmias in individuals with CHD or cardiomegaly/LVH.

Coronary Heart Disease

Studies that have examined autopsy findings of firefighters who suffered SCD have found that roughly 90% of victims had evidence of coronary atherosclerosis (13,20). CHD is characterized by atherosclerotic plaque in the arterial wall, which is preceded by endothelial dysfunction as the onset of the atherosclerotic process. As the plaque progresses, it results in stenosis and can cause ischemia. Death from a myocardial infarction frequently involves the rupture of vulnerable plaque, exposing blood to underlying connective tissue that is highly thrombotic. Platelets begin to adhere to the vessel and aggregate to each other to form a plug. Ultimately, this may result in the formation of an occlusive thrombus that causes a myocardial infarction.

Firefighting leads to increased shear stress that may increase the risk of plaque rupture. Furthermore, firefighting increases platelet number and activity and leads to a procoagulatory condition that may make thrombus formation more likely. Thus, although most firefighters recover from the cardiovascular strain of firefighting without incident, an individual with underlying atherosclerotic plaque, especially vulnerable plaque, is at a greatly increased risk of plaque rupture and thrombus formation during firefighting activity.

Cardiomegaly/LVH

Cardiomegaly (increased heart size and mass) and LVH (increased wall thickness and mass) are structural abnormalities that increase the risk of SCD caused by arrhythmia. LVH is a powerful predictor of cardiovascular morbidity and mortality in population-based studies (3), and there is a strong graded association between left ventricular mass and increased cardiovascular risk (14). LVH is frequently associated with fatal arrhythmias (33). Research has found that the increased risk associated with LVH is independent of other factors such as age, sex, smoking status, diabetes, and serum cholesterol. In a majority of cases, LVH is typically a result of hypertension with or without obesity and/or CHD. Obstructive sleep apnea, which also is commonly associated with hypertension and obesity, is another risk factor for LVH. In cases of firefighting SCD, CHD and LVH are frequently comorbid (13,20,36). Myocardial fibrosis is thought to be an important mediator of increased risk of SCD associated with LVH, although the precise mechanisms by which LVH causes cardiovascular morbidity and mortality are not fully understood. However, vascular changes, such as increased arterial stiffness, altered wave reflections and arterial-ventricular uncoupling may lead to the development of LVH.

A recent retrospective study found that cardiomegaly/LVH is a frequent cause of SCD in the general public and is highly associated with obesity and death at a younger age than CHD (33). There is mounting evidence that LVH/cardiomegaly is common among US firefighters and plays a major role in CVD events in the fire service. Kales *et al.* (20) conducted a case-controlled investigation of on-duty CHD fatalities and found evidence for LVH in 76% of the CHD deaths where the autopsy results were available. Subsequently, a larger follow-up case-fatality study was conducted to compare firefighters succumbing to on-duty CHD fatalities with firefighters suffering nonfatal CHD events leading to retirement. Among the fatalities, LVH/cardiomegaly was mentioned in summary reports of almost 60% of the available autopsies (13). In addition, Yang *et al.* (36) studied younger (<45 yr) firefighters and found a greater than 100-g difference in heart weight among SCD cases compared with trauma fatality controls. Furthermore, approximately 66% of cardiac cases had evidence of cardiomegaly (heart weight, >450 g), and this conveyed a fivefold increase in relative risk of SCD. These studies provide convincing evidence that LVH/cardiomegaly plays a role in a large percentage of firefighter fatalities.

As illustrated in Figure 3, firefighting may lead to several cardiac and vascular changes that increase the risk of arrhythmia, particularly in individuals with underlying vascular dysfunction and/or structural heart abnormalities. Obviously, firefighting leads to increased cardiac work. Furthermore, our research suggests that firefighting also leads to increased arterial stiffening and decreased arterial compliance. The combination of increased cardiac work and decreased arterial compliance may lead to ischemia that could provoke an arrhythmia, particularly in individuals with underlying CHD and/or structural heart changes that include myocardial fibrosis, which likely increases susceptibility to electrical abnormalities.

CONCLUSIONS

The proposed model suggests that the cardiovascular strain associated with firefighting may trigger a sudden cardiac event in a susceptible person through several biological pathways. Increases in shear stress may cause rupture of vulnerable plaque, resulting in thrombus formation and the occlusion of coronary arteries, which may be exacerbated by hypercoagulability that is known to increase the risk of thrombotic events. Alternately, acute risks encountered during firefighting activities may increase the risk of fatal arrhythmias. Ischemia (caused by an increase in myocardial oxygen demand that exceeds myocardial supply) may result in electrical, mechanical, and biochemical dysfunction of the cardiac muscle. Exposure to environmental conditions (such as gaseous and particulate toxicants in smoke) also may increase susceptibility to arrhythmias (23), particularly in those with LVH and other forms of cardiomegaly or in the context of ischemia.

Additional research is necessary to better understand how individual characteristics affect the cardiovascular responses to firefighting and the precise mechanisms by which firefighting leads to an increased risk of fatal arrhythmias and plaque rupture. Additional research also is needed to identify the types of physical fitness training programs that provide the greatest potential for reducing the risk of sudden cardiac events in the fire service. Firefighters accept great risks to protect their communities. Exercise professionals have a unique opportunity to help improve the health of this remarkable occupation, which is so important to our public safety.

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