Clotting and Fibrinolytic Changes following Firefighting Activities

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ABSTRACT

Approximately 45-50% of all duty-related deaths among firefighters are due to sudden cardiovascular events and a disproportionate number of these fatalities occur following strenuous fire suppression activities.

**Purpose:** To evaluate the effect of strenuous firefighting activities on platelets, coagulation and fibrinolytic activity and to document the extent to which these variables recovered 2 h after completion of the firefighting activity.

**Methods:** Firefighters performed 18 min of simulated firefighting activities in a training structure that contained live fires. Following firefighting activities, firefighters were provided with fluid and allowed to cool down and then recovered for 2 h in an adjacent room. Blood samples were obtained pre-firefighting, post-firefighting, and 2-h post-firefighting.

**Results:** Platelet number, platelet activity and coagulatory potential increased immediately post-firefighting and many variables (platelet function, PTT, FVIII) reflected a procoagulatory state even after 2 h of recovery. Fibrinolysis, as reflected by tPA, also was enhanced immediately post-firefighting but returned to baseline values by 2-h post-firefighting. In contrast, inhibition of fibrinolysis, as evidenced by a reduction in PAI-1 was depressed at 2-h post-firefighting.

**Conclusions:** Firefighting resulted in elevated coagulatory and fibrinolytic activity. However, 2-h post-firefighting, tPA returned to baseline and coagulatory potential remained elevated. The procoagulatory state that exists following firefighting may provide a mechanistic link to the reports of sudden cardiac events following strenuous fire suppression activities.

**Key Words:** hemostasis, coagulation, fibrinolysis, platelet activity
INTRODUCTION

Large multicenter studies have reported a relative risk of 2.1 to 5.9 for suffering an acute myocardial infarction within one hour of heavy physical exertion (6 METS or higher) compared with light activity or no exertion. Coronary artery thrombus formation plays a critical role in exercise-induced myocardial infarctions. Changes in blood clotting and fibrinolytic activity influence thrombus formation and an imbalance in these systems alters the risk of a thrombus formation.

Over the past 10 yr, approximately 40–50% of duty-related deaths among US firefighters have been attributed to sudden cardiac events. In addition, a review of the number of cardiac fatalities and the number of cardiac injuries reported by the National Fire Protection Association over the past five years, suggests that there are approximately 25 non-fatal line of duty cardiac events for every fatal cardiac event. Although firefighters spend a small percentage of their time engaged in firefighting activity, a large portion of their cardiac fatalities occur during or shortly after firefighting activity. Retrospective studies indicate that firefighters are 53 to 64 times more likely to suffer a fatal cardiac event during or shortly after fire suppression activity than during station duty. Importantly, these studies also report that firefighters have a relative risk of 5.2 to 7.6 of suffering a fatal sudden cardiac event following physical fitness training compared to firehouse duties - a relative risk very similar to the risk of acute myocardial infarction in the general population (25,39). The much greater relative risk of a firefighter suffering a cardiac event following fire suppression activities than during physical activity alone, suggests that multiple stressors (i.e., exercise, heat, psychological stress) may cause an exaggerated hemostatic response.

Firefighting involves heavy strenuous work while operating in a hostile environment and leads to activation of the sympathetic nervous system, increased cardiovascular and thermal strain, and
dehydration. Catecholamines stimulate platelets and several coagulatory factors suggesting that sympathetic stimulation may lead to a hypercoaguable state that increases the risk of thrombus formation. Firefighting has been shown to increase platelet number and activity.

An acute bout of strenuous exercise is associated with increased platelet number and activity, coagulatory factors, and fibrin formation, thus creating a procoagulatory state. However, this increase in coagulation is normally accompanied by a parallel increase in endogenous fibrinolysis leading to balanced hemostatic state even during strenuous exercise in normal healthy adults. There is evidence, however, suggesting that coagulatory potential may remain elevated into recovery, whereas fibrinolysis quickly returns to baseline values following very strenuous physical work. The imbalance between coagulation and fibrinolysis may contribute to the increased susceptibility to sudden cardiac events in the period following strenuous activity. It is unknown how heat stress affects the restoration of hemostatic balance following exertion.

While limited data suggest that heat stress can independently promote a procoagulatory state very little work has been done to characterize the coagulatory and fibrinolytic responses to firefighting despite the very high rate of duty-related cardiovascular events associated with fire suppression activity. Burgess and colleagues have recently reported an increase in coagulatory factors immediately following 12 min of firefighting but did not report changes in fibrinolytic factors.

Given (a) the importance of the hemostatic system in acute coronary syndromes, (b) the knowledge that sudden cardiac events are the leading cause of line-of-duty deaths among firefighters, (c) the evidence that among firefighters fire suppression is associated with a greater relative risk of sudden cardiac event than physical fitness training, (d) the strong evidence of hemostatic disruption following strenuous physical activity, and (e) the recognition of multiple stressors faced by firefighters, several of which have been shown to alter hemostasis, it is
important to examine how firefighting affects hemostasis and to investigate hemostatic changes during recovery from firefighting. Therefore, the purpose of this study was to evaluate the effect of strenuous firefighting activities on platelets and clotting and fibrinolytic activity, and to document the extent to which these variables recovered 2 h after completion of the firefighting activity. We hypothesized that firefighting would lead to increased platelet number and function, increased coagulatory potential and enhanced fibrinolysis. Further, we hypothesized that platelet activity and coagulatory potential would remain elevated after 2 h of recovery while fibrinolytic potential would return to baseline levels.

METHODS

Subjects
Eighteen male firefighters (mean ± SD: age 25.4 ± 4.8 yr; height = 1.82 ± 0.08 m; body mass = 85.8 ± 13.5 kg; body mass index (BMI) = 25.7 ± 2.9 kg·m⁻²) participated in testing. Participants were not specifically heat acclimated for our study, but all participants were firefighters who were familiar with performing drills (or live fire activity) in their protective equipment. Prior to testing, participants completed a health history questionnaire. Exclusion criteria included a history of atherosclerotic cardiovascular disease, medications for high blood pressure or cholesterol, or medications known to affect hemostasis (aspirin, acetaminophen, ibuprofen, cold or asthma medication).

Study Design
Participants were fully informed of the purpose of the study, the requirements of participation, and were given an opportunity to ask questions of the investigators. Participants signed an informed consent document indicating that they understood the risks and benefits of participation
and that their participation was voluntary. This study was approved by the University of Illinois Institutional Review Board.

This study investigated alterations in platelet function, coagulatory variables and fibrinolytic variables immediately after strenuous firefighting and following 2 h of recovery. Participants performed prescribed live firefighting drills wearing full personal protective equipment (PPE), including self-contained breathing apparatus (SCBA), in a training structure that contained live fires. The average weight of the PPE and SCBA was approximately 20 kg. Following the firefighting activity, firefighters participated in incident-scene rehabilitation (~ 20 min), where they removed their helmet, hood, gloves, and bunker coat and sat in a cool room (approximately 20°C). During rehabilitation, participants were provided with water *ad libitum* and cooled passively, to reflect what is recommended at a fire scene.

Following the rehabilitation period, participants changed into dry clothes and walked to an adjacent building (fire station) for a 2-h recovery period. During recovery, firefighters were engaged in classroom activities, reading, or viewing television to mimic what may occur at a fire station following a fire call.

The firefighting drills lasted 18 min (requiring approximately one cylinder of air for most participants) and consisted of nine 2-min periods of alternating rest and work. The work cycles included stair climbing, simulated forcible entry, a simulated search, and simulated hose advance. The live fire drills were completed on the second story of a training building that contained live fires. Throughout the firefighting drills, trained personnel controlled the temperature in the training structure by monitoring thermocouple readings and adding small fuel packages to the fire sets sequentially and controlling the ventilation conditions in the room. The temperatures at 1.2 m above the floor were maintained at roughly 70–82°C and the floor...
temperatures were maintained at approximately 35–41°C. The prescribed firefighting activities (other than stair climbing) required participants to work almost exclusively in the space within 1 m of the floor.

Descriptive characteristics were obtained prior to participating in firefighting drills. Height was measured (to the nearest 0.01 m) using a stadiometer and body mass was measured (to the nearest 0.5 kg) using a digital beam balance platform. Body mass index was calculated as the body mass in kilograms divided by the height in meters squared. A cholesterol profile from a finger stick sample (Cholestech, Hayward, CA) was also assessed prior to the live-fire training. An activity questionnaire was administered to determine activity status.

**Heart Rate and Core Temperature**

Body temperature was measured continuously throughout the protocol using a monitor and a silicone-coated gastrointestinal (GI) core temperature capsule (Mini Mitter, VitalSense; Philips Respironics, Bend, OR). Participants swallowed a small disposable core temperature sensor capsule the night before the study was conducted. Heart rate was measured using a heart rate monitor (Vantage XL; Polar Electro, Inc., Lake Success, NY).

**Blood Sampling and Laboratory Methods**

Venous samples were drawn from the antecubital vein using a 21 gauge needle by a trained phlebotomist. Samples were drawn before firefighting activity and again immediately post-firefighting and 2-h post-firefighting. Hemoglobin and hematocrit were assessed via complete blood count (CBC) and plasma volume changes were calculated based on the Greenleaf method.

**Platelet number and function.** Platelet count was assessed at a local clinic from venous whole blood as part of a CBC analysis, using the electrical impedance method (with an instrument such as the COULTER® LH 700 Series; Beckman Coulter, Inc., Fullerton, CA).
Platelet function was assessed by epinephrine-induced and adenosine 5’-diphosphate -induced platelet aggregability using a platelet function analyzer (PFA-100; Dade Behring, Deerfield, IL). Venous blood samples were collected in a Vacutainer containing 3.2% sodium citrate, maintained at room temperature and analyzed within 2 h of collection. Blood was pipetted (800 μl) into the disposable cartridges and then aspirated under high shear rates (5,000–6,000 sec\(^{-1}\)) through an aperture cut into the membrane coated with collagen and adenosine 5-diphosphate (ADP) and a membrane coated with collagen and epinephrine (EPI). Time to occlusion was reported.

**Coagulatory and fibrinolytic variables.** Blood samples were drawn into tubes containing 3.2% sodium citrate for measurements of all coagulation and fibrinolytic factors, except for the assessment of tissue plasminogen activator (t-PA) activity, in which those samples were drawn into Stabilyte tubes (Biopool, Wicklow, Ireland). All samples were centrifuged at 2,300 rpm for 25 min at 4°C with the plasma removed and placed into aliquots, and stored at \(-70^\circ\text{C}\) for later analysis. Plasminogen activator inhibitor (PAI-1) antigen, t-PA antigen and Tissue Factor (TF) were analyzed in duplicate using an enzyme-linked immunosorbent assay kit (American Diagnostica, Stamford, CT). Antithrombin III (AT-III) was analyzed in duplicate using an Actichrome chromogenic activity kit (American Diagnostica, Stamford, CT). Tissue plasminogen activator and PAI-1 activity were analyzed in duplicate using commercially available chromogenic substrate kits (DiaPharma Group, Inc., West Chester, OH). Factor VIII (FVIII) activity was analyzed in duplicate using a chromogenic assay kit (Chromogenix/DiaPharma Group, Inc., West Chester, OH). Activated partial thromboplastin time (aPTT) was analyzed using STA-PTT Automate 5 (Diagnostica Stago, Inc., Parsippany, NJ).
Data Analysis

Changes in plasma volume were calculated using the Greenleaf method. Blood variables that are expressed as a concentration were corrected for changes in plasma volume and statistical analyses were performed for both the non-corrected and corrected values. Because the uncorrected values represent the relevant physiological state, these values are presented in the tables. However, when the analyses of corrected and uncorrected values produced different statistical results, these are noted in the text. Variables were checked for normal distribution, and those variables not normally distributed were log transformed (natural logarithm) prior to statistical analyses.

A one-way analysis of variance with repeated measures was employed to detect differences over time in hemostatic variables. Following a significant F-ratio, post-hoc tests were used to identify specific time points that differed. Data were analyzed using SPSS version 18 (SPSS Inc., Chicago, IL). Descriptive data are expressed as mean ± standard deviation. Statistical significance was set at $P < 0.05$ for all analyses.

RESULTS

Participants were relatively young, apparently healthy firefighters. Total mean cholesterol (174.8 ± 38.9 mg·dL$^{-1}$) was in the desirable range; mean low density lipoprotein (LDL) cholesterol (107.4 ± 32.5 mg·dL$^{-1}$) was in the near optimal range; and mean high density lipoprotein (HDL) cholesterol (46.4 ± 12.6 mg·dL$^{-1}$) was in the average range. Table 1 presents data on the relative distribution of study participants in different categories based on cardiovascular disease risk factors. Seven firefighters were normal weight ($18.5 \leq \text{BMI} < 25$), 10 firefighters were overweight ($25 \leq \text{BMI} < 30$), and one firefighter was obese ($\text{BMI} \geq 30$). Two of the participants had total cholesterol above 240 mg·dL$^{-1}$, one firefighter had low density lipoprotein cholesterol
above 190 mg·dL⁻¹, and six firefighters had high density lipoprotein cholesterol less than 40 mg·dL⁻¹. Seventeen of the participants reported that they were non-smokers and one individual indicated that he was an “occasional” smoker. Based on activity recall data, 28% of participants met current guidelines for physical activity. Three participants indicated that an immediate relative had died prematurely due to cardiovascular disease.

As seen in Table 2, firefighting activity resulted in an increased heart rate, reaching a mean peak HR of greater than 160 b·min⁻¹ by the end of the evolution. Heart rate returned to below baseline values after 2 h of rest following firefighting activity, suggesting the pre-firefighting values were not true resting values and represented an anticipatory response to activity. Core temperature increased by approximately 0.7°C following the 18 min of firefighting activity and returned to below baseline values following 2 h of recovery. Firefighting activity caused an average loss of plasma volume of approximately 4%. By 2-h post-firefighting, plasma volume was restored to baseline levels.

**Platelet number and function.** As seen in Table 3, platelet number increased significantly immediately post-firefighting (P < 0.001) and returned to baseline values 2-h post-firefighting. When the blood was exposed to collagen and EPI, platelet closure time decreased significantly immediately post-firefighting (P < 0.05) and then returned toward baseline, although it remained significantly lower than baseline at 2-h post-firefighting (P < 0.05). Platelet closure time when the blood was exposed to collagen and ADP decreased significantly following firefighting activity (P = 0.018) but was not different from baseline 2-h post-firefighting.

**Coagulatory and fibrinolytic variables.** Some blood samples were not analyzed due to hemolysis, thus the number of samples is noted for each blood variable. Activated partial thromboplastin time (aPTT) decreased significantly immediately post-firefighting (P = 0.025) and remained significantly below baseline at 2-h post-firefighting (P = 0.032, Table 4). Tissue
factor, uncorrected for changes in plasma volume, was significantly increased immediately post-firefighting compared to 2-h post-firefighting ($P = 0.018$). However, when corrected for changes in plasma volume, TF did not vary significantly with time suggesting that hemoconcentration was primarily responsible for this change. Factor VIII increased significantly immediately post-firefighting ($P < 0.001$) and remained elevated at 2-h post-firefighting ($P = 0.002$). Antithrombin III did not change significantly over time ($P = 0.055$), although there was a strong trend for ATIII to be higher after firefighting and in the recovery period.

Several fibrinolytic factors reflected a transient increase in fibrinolysis (Table 5). Tissue plasminogen activator (t-PA) activity and antigen were elevated immediately post-firefighting ($P = 0.007$, $P = 0.005$, respectively), but there were no significant differences between pre and 2-h post values ($P = 0.664$, $P = 0.947$, respectively). There was also a significant effect of time on PAI-1 activity. Post-hoc analysis revealed that PAI-1 activity was significantly lower both immediately post and 2-h post-firefighting than pre-firefighting ($P = 0.027$, $P = 0.020$, respectively). PAI-1 antigen did not differ significantly between pre- and immediately post-firefighting but was significantly lower 2-h post-firefighting than pre-firefighting ($P < 0.05$). When PAI-1 antigen levels were corrected for changes in plasma volume, there was no significant time effect.

**Discussion**

We documented significant changes in platelet number and function, coagulation and endogenous fibrinolysis immediately following live firefighting in a group of young, apparently healthy firefighters. The increase in heart rate and modest elevation in body temperature ($0.7^\circ C$) in this study is consistent with results of other studies investigating short-term firefighting activity. These findings suggest that a short bout of live firefighting activities activates the
coagulatory cascade and enhances fibrinolysis. By 2-h post-firefighting activity, endogenous t-PA had returned to baseline although PAI-1 remained depressed, and platelet function and coagulation remained elevated. These data support the hypothesis that firefighters experience a hemostatic imbalance that is primarily prothrombotic during recovery from firefighting activity.

Epidemiological studies provide convincing evidence that strenuous physical exertion increases the risk of thrombotic events in individuals with underlying cardiovascular disease. Experimental evidence has shown that strenuous physical activity activates both coagulation and fibrinolysis. Acute exercise also leads to an increase in platelet number and platelet function that is dependent on the intensity of exercise. Wallen and colleagues showed that maximal exercise (on a bicycle ergometer), mental stress and epinephrine resulted in significant increases in platelet number and aggregation, which returned to baseline following 60 min of recovery. In our study, we found a significant increase in platelet number and activation following a brief bout of strenuous, but intermittent, firefighting activity. The increase in platelet number was evident before corrections were made for changes in plasma volume and persisted after corrections were made, thus demonstrating that the increase in platelet number reflected more than just hemoconcentration. Catecholamine release as a result of sympathetic nervous system stimulation is likely involved in the release of platelets from the spleen and reticular tissue. The combination of heat stress and exercise results in increased sympathetic drive and catecholamine release compared to physical exertion alone. While platelet number returned to baseline values by 2-h post-firefighting, platelet activation, evidenced by a shorter EPI closure time, remained significantly elevated 2-h post-firefighting. The discrepancy in the recovery data presented here compared to the data reported by Wallen et al. raises the possibility that the combination of exercise and heat stress during live-fire fighting activities may lead to a more prolonged platelet activation than exercise alone.
Strenuous physical activity activates the coagulatory system in healthy subjects to a greater extent than moderate activity. Accompanying the increase in coagulatory variables, strenuous exercise also activates the fibrinolytic system as evidenced by elevated levels of tPA activity and a decrease in PAI-1 activity and these responses are dependent upon the intensity of the exercise. However, strenuous exercise may produce an imbalance in hemostasis. Strenuous exercise has been shown to increase thrombotic tendency but this change is not evident after moderate exercise.

Although many authors maintain that there is a balanced hemostatic response (increased coagulation coupled with increased fibrinolysis) to exercise in healthy individuals; several reports have shown that coagulatory variables remain elevated 1–2 h after strenuous exercise, whereas fibrinolytic variables quickly return to baseline levels. Hegde et al. reported that 30 min of running at 70% of VO$_{2\text{max}}$ resulted in an increase in FVIII activity that was sustained during a 1-h recovery period. In contrast, the tPA activity was significantly elevated post-running but gradually declined during the 1-h recovery period. Lin et al. have reported that elevated coagulatory potential (increased Factor VIII) persisted at 2-h and 6-h of recovery whereas fibrinolytic activity fell sharply. The discrepancy in coagulatory and fibrinolytic potential during recovery from strenuous activity may account for an increased vulnerability to myocardial infarction following strenuous activity. In the present study, we found that markers of enhanced coagulation (elevated TF and FVIII activity) persisted after 2 h of recovery. In contrast, tPA, a maker of fibrinolysis, had returned to baseline by 2-h post-firefighting. Furthermore, the shortened aPTT time at 2 h post FF provides evidence of a shift toward a procoagulatory state following 2 h of recovery.

Firefighters are a unique occupational group who perform strenuous work under stressful conditions. Strenuous firefighting activities can lead to attainment of maximal heart rate,
elevated core temperature, dehydration, decreased stroke volume, increased arterial stiffness, and alterations in myocardial function. Burgess et al. reported that short-term (12 min) firefighting leads to an increase in Factor VIII, but these authors did not report on fibrinolytic factors. Our current data provide evidence of a procoagulatory state following firefighting activities and persisting for 2-h post-firefighting, which may contribute to an increased likelihood of a thrombotic event. These findings support the theory that alterations in blood clotting and fibrinolysis may play a mechanistic role in the increased risk of sudden cardiac events during and following firefighting. However, the relative contribution of multiple stressors (strenuous physical work, heat stress, emotional stress) that are encountered during firefighting on the hemostatic response is unclear.

It is well documented that coagulatory and fibrinolytic responses to exercise differ between healthy individuals and those with underlying cardiovascular disease or risk factors for cardiovascular disease. In the current study we tested a relatively young group of firefighters and excluded firefighters who had a known history of cardiovascular disease, and those who used medications known to alter hemostatic variables. Our participants had lower prevalence of cardiovascular disease risk factors than has been reported in the US Fire Service, and thus our results may underestimate hemostatic disruption that is evident in older firefighters with more risk factors or those who possess clinical or subclinical cardiovascular disease. Furthermore, immediately following firefighting, our participants were provided with planned cooling and rehydration (incident rehabilitation) and placed into a relatively controlled, relaxed environment to recover. Hence, our results may not represent the full magnitude of changes that occur under actual firefighting conditions in which firefighters work for longer periods or operate in more extreme environments, thus increasing core temperature to a greater degree. Another limitation of our study is the relatively small sample size. However, the findings of this study demonstrate
considerable hemostatic disruption following short-term firefighting activity and provide a potential link between the physiological stress of firefighting and high number of sudden cardiac events during and after fire suppression activities. Additional research including more severe firefighting activity, individuals with a range of cardiovascular risk profiles, and a larger population of firefighters is needed to better elucidate the hemostatic responses that occur during and after firefighting activity.

Acknowledgments

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The authors have no conflicts of interest to declare.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.
REFERENCES


Table 1. Classification by risk factors.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Frequency (n (%))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index^a (BMI)(kg·m⁻²)</td>
<td></td>
</tr>
<tr>
<td>Underweight (&lt; 18.5)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Normal weight (18.5–24.9)</td>
<td>7 (39%)</td>
</tr>
<tr>
<td>Overweight (25.0–29.9)</td>
<td>10 (56%)</td>
</tr>
<tr>
<td>Obesity (≥ 30)</td>
<td>1 (6%)</td>
</tr>
<tr>
<td>Total cholesterol^b (mg·dL⁻¹)</td>
<td></td>
</tr>
<tr>
<td>Desirable (&lt; 200)</td>
<td>15 (82%)</td>
</tr>
<tr>
<td>Borderline high risk (200–239)</td>
<td>1 (6%)</td>
</tr>
<tr>
<td>High risk (≥ 240)</td>
<td>2 (12%)</td>
</tr>
<tr>
<td>Low density lipoprotein^b (LDL)(mg·dL⁻¹)</td>
<td></td>
</tr>
<tr>
<td>Optimal (&lt; 100)</td>
<td>9 (50%)</td>
</tr>
<tr>
<td>Near optimal/above optimal (100–129)</td>
<td>7 (39%)</td>
</tr>
<tr>
<td>Borderline high (130–159)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>High risk (160–189)</td>
<td>1 (6%)</td>
</tr>
<tr>
<td>Very high risk (≥ 190)</td>
<td>1 (6%)</td>
</tr>
<tr>
<td>High density lipoprotein^b (HDL)(mg·dL⁻¹)</td>
<td></td>
</tr>
<tr>
<td>Low (&lt; 40)</td>
<td>6 (33%)</td>
</tr>
<tr>
<td>No category per reference (40–59)</td>
<td>9 (50%)</td>
</tr>
<tr>
<td>High (≥ 60)</td>
<td>3 (18%)</td>
</tr>
<tr>
<td>Physical activity (PA)^c</td>
<td></td>
</tr>
<tr>
<td>Does not meet PA guidelines</td>
<td>13 (72%)</td>
</tr>
<tr>
<td>Meets/Exceeds PA guidelines</td>
<td>5 (28%)</td>
</tr>
<tr>
<td>Smoker</td>
<td></td>
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<tr>
<td>Yes</td>
<td>1 (6%)</td>
</tr>
<tr>
<td>No</td>
<td>17 (94%)</td>
</tr>
<tr>
<td>Family history of CVD</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3 (18%)</td>
</tr>
<tr>
<td>No</td>
<td>15 (82%)</td>
</tr>
</tbody>
</table>

^a Classification based on established categories (29)  
^b Classification based on established criteria (28)  
^c PA guidelines: 30–60 min·d⁻¹ (150 min·wk⁻¹) of purposeful moderate exercise, or 20–60 min·d⁻¹ (75 min·wk⁻¹) of vigorous exercise, or a combination of moderate and vigorous exercise (13)
Table 2. Mean ± SD heart rate, core temperature and change in plasma volume variables pre, post, and 2-h post-firefighting (N = 18).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pre</th>
<th>Post</th>
<th>2-h Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (b·min⁻¹)</td>
<td>78 ± 11</td>
<td>162 ± 15ᵃ</td>
<td>67 ± 10ᵇ</td>
</tr>
<tr>
<td>Core temperature (°C)</td>
<td>37.14 ± 0.06</td>
<td>37.89 ± 0.09ᵃ</td>
<td>36.92 ± 0.05ᵇ</td>
</tr>
<tr>
<td>Change in plasma volume (%)</td>
<td>----</td>
<td>-3.99 ± 4.92</td>
<td>1.54 ± 4.92ᵇ</td>
</tr>
</tbody>
</table>

ᵃ Significantly different than Pre (P < 0.05).
ᵇ Significantly different than Post (P < 0.05).
Table 3. Mean ± SD platelet variables pre, post and 2-h post-firefighting (N = 18).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pre</th>
<th>Post</th>
<th>2-h Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet count</td>
<td>256.9 ± 61.7</td>
<td>300.8 ± 81.2</td>
<td>254.4 ± 77.6</td>
</tr>
<tr>
<td>EPI closure time (s)</td>
<td>117.1 ±13.8</td>
<td>94.2 ± 20.5</td>
<td>107.6 ± 18.6</td>
</tr>
<tr>
<td>ADP closure time (s)</td>
<td>86.2 ±16.2</td>
<td>73.9 ± 10.9</td>
<td>79.7 ± 14.6</td>
</tr>
</tbody>
</table>

ADP, adenosine 5-diphosphate induced; EPI, epinephrine induced.

\(^{a}\) Significantly different than Pre (P < 0.05).

\(^{b}\) Significantly different than Post (P < 0.05).
Table 4. Mean ± SD coagulatory variables pre, post, and 2-h post-firefighting.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pre</th>
<th>Post</th>
<th>2-h Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>aPTT (s)((n = 13))</td>
<td>33.9 ± 4.93</td>
<td>31.9 ± 5.5(^a)</td>
<td>31.8 ± 6.0(^a)</td>
</tr>
<tr>
<td>Tissue factor (pg·mL(^{-1}))((n = 17))</td>
<td>67.2 ± 36.7</td>
<td>72.4 ± 30.0</td>
<td>62.1 ± 32.9(^b)</td>
</tr>
<tr>
<td>Factor VIII (IU·mL(^{-1}))((n = 14))</td>
<td>90.1 ± 15.4</td>
<td>138.6 ± 36.0(^a)</td>
<td>127.4 ± 43.2(^a)</td>
</tr>
<tr>
<td>AT-III activity (%((n = 17))</td>
<td>98.5 ± 8.7</td>
<td>102.7 ± 8.4</td>
<td>103.9 ± 8.0</td>
</tr>
</tbody>
</table>

\(^a\) Significantly different than Pre \((P < 0.05)\).

\(^b\) Significantly different than Post \((P < 0.05)\).
Table 5. Mean ± SD fibrinolytic variables pre, post, and 2-h post-firefighting.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pre</th>
<th>Post</th>
<th>2-h Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>t-PA activity (IU·mL⁻¹) (n = 8)</td>
<td>0.59 ± 0.38</td>
<td>2.37 ± 1.49&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.65 ± 0.33&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>t-PA antigen (ng·mL⁻¹) (n = 18)</td>
<td>6.34 ± 3.46</td>
<td>11.8 ± 9.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.24 ± 2.85&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>PAI-1 activity (AU·mL⁻¹) (n = 14)</td>
<td>3.75 ± 3.04</td>
<td>2.63 ± 2.65&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.29 ± 2.28&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>PAI-1 antigen (ng·mL⁻¹) (n = 17)</td>
<td>24.4 ± 16.9</td>
<td>26.1 ± 15.2</td>
<td>21.8 ± 14.8&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

t-PA, tissue plasminogen activator; PAI-1, plasminogen activator inhibitor.

<sup>a</sup>Significantly different than Pre (P < 0.05).

<sup>b</sup>Significantly different than Post (P < 0.05).