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In this report, we describe the secondary or indirect cardio-
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**Background**

Bioterrorist events can produce a variety of secondary
cardiovascular effects. For example, mass vaccination cam-
paigns to combat such potential bioterrorist agents as

smallpox can lead to cardiovascular symptoms in some of

those who receive the vaccine. The acute and posttraumatic

psychological stress experienced by those affected (directly

or indirectly) by the bioterrorist event may trigger acute
cardiovascular events (such as heart attacks, sudden deaths,

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**Task Force II: Indirect and Secondary Cardiovascular Effects of Biological Terrorism Agents and Diseases**

Dr. Larry M. Baddour declared that his institution (Mayo Clinic) has financial relationships with infectious disease companies. The other authors of this report declared that they have no relationships with industry pertinent to this topic.
Secondary and Indirect Cardiovascular Effects of Bioterrorism

Cardiovascular Effects of Smallpox Vaccination

Variola virus, which causes smallpox, does not produce cardiovascular complications directly, but vaccinia virus, which is used in the smallpox vaccine, has been linked to cardiovascular complications, especially myocarditis and myopericarditis (1,2). The pathogenic mechanism through which the vaccine precipitates myocarditis remains to be determined for 3 reasons. First, endomyocardial biopsy, which is the gold standard for diagnosing myocarditis, has limited sensitivity because the active inflammatory process is frequently patchy in distribution and has a diagnostic yield of only 10% (3). Second, because of the risks associated with endomyocardial biopsy, it is recommended for use only in patients with left ventricular dysfunction and symptoms not controlled by standard medical management. Third, the limited evidence currently available suggests that an abnormal immune mechanism is responsible for the development of myocarditis (4,5).

Several studies have examined the potential cardiovascular effects of smallpox vaccine. For example, Murphy et al. described a case in which endomyocardial biopsy was obtained and primary vaccinia infection was excluded based on negative results of polymerase chain reaction (PCR) screening for the vaccinia genome (4). Histopathologic findings showed a mixed lymphocytic and eosinophilic infiltration, while immunoperoxidase stain demonstrated mainly CO3+ T-cells and the presence of major basic protein staining suggested an aberrant immunologic response with an eosinophil-mediated mechanism of myocyte injury. Examinations of autopsy-proven cases of myocarditis following smallpox vaccination from the 1960s also demonstrated both lymphocytic and eosinophilic infiltrations of the myocardium.

The Department of Defense (DOD) has collected data to determine the frequency of myopericarditis following smallpox vaccination among 230,734 U.S. military personnel who received vaccinia vaccines for the first time between December 2002 and March 2003 (2,4). The DOD study found that the rate of myopericarditis was 1 in 12,819 of those who received the vaccine for the first time. In those who developed acute myocarditis, clinical evidence appeared at 7 to 19 days (mean 10.5 days) following vaccination. However, no cases of myopericarditis developed among 95,622 individuals who received the vaccine but had previously been vaccinated.

Currently, a potential causal link is being examined between smallpox vaccination and ischemic cardiac events. Both fatal and nonfatal ischemic events have occurred in civilian and military populations 4 to 17 days following vaccination (4,6). Analyses of incidence data on current and past cardiac events from a vaccination campaign in New York City in 1947 did not find an increased number of cardiac ischemic events following smallpox vaccination (7). However, new vaccination screening guidelines were adopted to reduce the risk of potential ischemic events by excluding individuals with known cardiac disease or 3 or more known cardiac (ischemic) risk factors (8).

Stress-Related Effects

Natural disasters (e.g., earthquakes and blizzards) and “unnatural” disasters (i.e., caused by humans, such as missile attacks) have been associated with an increased incidence of cardiovascular events related to the sudden stress experienced by the affected population (1–4). On the day of the 1994 earthquake in Northridge, California, for example, the number of sudden cardiac deaths, acute myocardial infarctions (MIs), and all atherosclerotic ischemic heart disease deaths increased.

Increases in emotional and physical stress can stimulate the sympathetic nervous system and increase the release of catecholamines, resulting in increased blood pressure, heart rate, and contractility (which can increase oxygen demand); changes in sheer stress of blood against an atherosclerotic plaque, which may contribute to its rupture; and arrhythmias. At the same time, stimulation of alpha-receptors in the coronary arteries increases vascular resistance and reduces blood flow, leading to a decrease in oxygen supply.

The terrorist attacks of September 11, 2001, and the subsequent anthrax exposures created a very high level of psychological stress in those directly targeted as well as their friends and relatives. The attacks also increased psychological stress among the entire population of the U.S. Although the stress experienced by those who were not directly affected by the attacks may have been less severe than that experienced by those targeted directly, it may have produced far more cardiovascular events because it affected millions of individuals. However, the absolute number of cardiovascular events throughout the U.S. did not produce an unmanageable workload for cardiovascular treatment facilities.

Studies examining the effect of the September 11, 2001, terrorist attacks on cardiac event rates show mixed results. A careful study of the cardiovascular deaths in New York City in the month following September 11, 2001, found no increase in cardiac mortality following the terrorist attack (9). In addition, in 1 analysis there was no significant increase in hospital admissions for cardiac events in a survey of several New York City hospitals (9,10). One potential explanation for these negative results was that patients with cardiac symptoms in New York City may have been directed away from hospitals near the site of attack. Conversely, a study from Brooklyn described an increase in acute MIs and tachycardias following the terrorist attack (11). Studies from New Jersey (12) and Worcester, Massachusetts (13), also suggested that the terrorist attacks increased hospitalizations for MIs in these locations more remote from the site of attack. It is also possible that cardiovascular events occurred at a lower level in New York City and nationwide that was not accounted for in this index. For example, cardiovascular events occurring in individuals within the World Trade Center towers on September 11, 2001, would not have been measurable due to the deaths caused by collapse of the buildings. In addition, it is not yet...
clear whether those who inhaled toxic dust following the collapse of the World Trade Center will experience long-term cardiovascular effects as a result. Certainly some have experienced pulmonary manifestations (14).

Investigators have recently reported an increased firing of implantable cardioverter-defibrillators (ICDs) in the New York City area patients in the month following September 11, 2001. One study reported a 2.3-fold increase in ventricular tachyarrhythmias in the 30 days following the attack compared to the 30 days preceding the terrorist attack (15). Another study by some of the same investigators observed similar findings in Gainesville, Florida (16). However, seasonal variations in such arrhythmias and other cardiac events—which may even occur in mild climates—were not taken into account in these studies (17).

Pathophysiological Processes Through Which Terrorist Attacks Could Trigger Cardiovascular Events

Extensive data on the causes of cardiovascular events by external events provide a basis for understanding the cardiac events that future, potentially more widespread, attacks might cause. The distinct patterns of the onset of many acute cardiovascular conditions (e.g., acute MI, cardiac arrest, stroke) indicate that these events can be triggered by factors external to the atherosclerotic plaque.

Circadian Variation of MI

The onset of MI has a distinct pattern, with peak incidence in the hours after awakening and arising. Serum creatine kinase (CK) measurements obtained from 703 subjects in the MILIS (Multicenter Investigation of Limitation of Infarct Size) study were used to demonstrate a marked circadian variation in the incidence of MI, with a 3-fold increase at 9 AM compared to 11 PM (18). Goldberg et al. (19) and Willich et al. (20) have subsequently refined this evidence by determining that the increased incidence of MI occurs within the first few hours after awakening and onset of activity, independent of time of day. These data indicate that the activities after awakening trigger the onset of a sizeable percentage of acute coronary events. Thompson et al. (21) and others (22) have suggested that in the evening hours (between 6 PM and 12 AM), a secondary peak of MI onset may occur. Infarcts occurring in these hours may result from an evening meal or other triggers concentrated in the evening hours.

A variety of factors both related to and independent of activity level may create the milieu in the coronary plaque that leads to an increased incidence of MI onset in the morning hours. A morning systemic blood pressure surge due to increased cortisol and catecholamine secretion in combination with increased coronary artery tone could promote disruption of a vulnerable plaque (23,24). Increased coronary artery tone alone could worsen flow reduction produced by fixed stenoses (24). Prothrombotic processes—including increased platelet adhesion and aggregability (25,26), increased factor VII, and plasminogen activator inhibitor activity (27), along with increased blood viscosity (28)—have been implicated in the onset of acute cardiac events and have been associated with decreased effectiveness of heparin and thrombolitics in the morning hours (29). Predisposition to plaque disruption and subsequent thrombosis added to reduced fibrinolytic activity in the morning could increase the likelihood that an otherwise harmless mural thrombus overlying a small plaque fissure would propagate and occlude the coronary lumen (30).

Epidemiologic and physiologic evidence suggests that the periodicity of MI onset is probably due to a combination of the true endogenous circadian rhythm and the daily rest-activity cycle. Cortisol secretion, a determinant of systemic blood pressure, is an established endogenous circadian process independent of daily activity (31), while enhanced platelet aggregability (25) and in vitro platelet responsiveness to adenosine diphosphate and epinephrine (26) increase only after the patient wakes and assumes an upright position. The peak morning incidence of MI probably results from the synchronization of adverse pathophysiologic processes.

Weekly Variations in Acute MI

Numerous investigators have reported a circaseptan (weekly) variation in MI onset, with a peak incidence on Mondays (32,33). Willich et al. (32) noted that this increase occurs primarily in the working population, who have a 33% higher relative risk of MI on this day of the week than the nonworking population. However, Spielberg et al. (33) observed a Monday increase in both working and retired subgroups. Some researchers have noted an increased incidence of MI on the weekend (34), while others have identified a weekend nadir (32).

Seasonal Variations in Acute MI and Coronary Artery Disease Deaths

Several investigators have reported a circannual (seasonal) variation in the incidence of acute MI onset, infarct size, and cardiac mortality, with a peak in the winter months (17,35,36). In the 83,541 subjects in the NRMI (National Registry of Myocardial Infarction) database between 1990 and 1993, 10% more acute cardiac events occurred in winter or spring than in summer (p < 0.05) (35). When Spencer et al. (36) reviewed data on 259,891 patients included in the second NRMI study from 1994 to 1996, they noted that at least 50% more cases of MI were reported in the winter (peak in January) than in the summer (nadir in July). Kloner et al. (37) demonstrated larger infarct size during the winter months by retrospective analysis of CK enzyme release in participants from the MILIS and Thrombolysis Myocardial Infarction-4 trial.

Sayer et al. (38) obtained prospective data on 1225 consecutive patients with acute MI admitted to a general hospital. Overall, these investigators noted a winter peak in the incidence of MI onset. However, patients who were
than those who died at rest.

Those who died during exertion were much more likely to demonstrate a plaque with a ruptured cap suddenly (48). Those who died during exertion as a cause of plaque disruption has been further related vessel after thrombolytic therapy. Support for heavy physical exertion was reported at MI onset in 18.7% of patients. Compared to patients whose infarction occurred at rest or during mild activity, those with exertion-related infarction had fewer coronary vessels with greater than 60% stenosis and were more likely to have an occluded infarct-related vessel after thrombolytic therapy. Support for heavy exertion as a cause of plaque disruption has been further strengthened by an autopsy series on men who had died suddenly (48). Those who died during exertion were much more likely to demonstrate a plaque with a ruptured cap than those who died at rest.

Other Cardiovascular Triggers

The circadian, circaseptan, and circannual variations in the incidence of MI onset suggest that the onset of acute cardiac events is not random and can be triggered by endogenous rhythms in combination with external activities and exposures. The MIOS (Myocardial Infarction Onset Study) investigators have identified several of the activities and exposures that can trigger MI onset, including heavy physical exertion, anger, mental stress, sexual activity, cocaine use, marijuana use, and air pollution (41–45). According to these investigators, these triggers account for over 20% of infarctions, totaling more than 250,000 events, in the U.S. each year.

Heavy Physical Exertion

Several studies have identified heavy physical exertion as a trigger of acute MI. The MILIS trial (46), for example, found that 14% of patients engaged in moderate physical activity and 9% engaged in heavy physical activity prior to experiencing an MI. In the TIMI-2 trial (47), moderate or marked physical activity was reported at MI onset in 18.7% of patients. Compared to patients whose infarction occurred at rest or during mild activity, those with exertion-related infarction had fewer coronary vessels with greater than 60% stenosis and were more likely to have an occluded infarct-related vessel after thrombolytic therapy. Support for heavy exertion as a cause of plaque disruption has been further strengthened by an autopsy series on men who had died suddenly (48). Those who died during exertion were much more likely to demonstrate a plaque with a ruptured cap than those who died at rest.

Fifty-four (4.4%) of 1228 patients enrolled in the MIOS trial reported heavy exertion (6 or more metabolic equivalents) within 1 h of the onset of MI (41). The cardiac symptoms often began during the activity. Using the case-crossover study design developed by Maclure (49), the estimated relative risk of MI in the hour after heavy physical activity was 5.9 compared to less strenuous or no physical exertion. Relative risks were 107 among people who usually exercised less than once a week, 19.4 in those who exercised once or twice a week, 8.6 in those who exercised 3 to 4 times a week, and 2.4 in those who exercised 5 or more times per week. Therefore, habitually sedentary individuals were at greatest risk of MI after heavy exertion and increasing levels of regular physical exercise were associated with progressively lower coronary risk. Similarly, exercise appears to protect individuals from sudden cardiac death associated with heavy physical exertion (48,50).

Proposed mechanisms for triggering MI onset by heavy exertion include increased sympathetic nervous system activation leading to increased myocardial oxygen demand (50) and increased platelet activation in sedentary patients and those with prior MI (51). Interestingly, heavy exertion does not cause platelet activation in healthy active volunteers, which may help explain the protective effect of regular exercise. Additional benefits of regular exercise include blunted up-regulation of the sympathetic nervous system during exertion and activation of the fibrinolytic system (52). Thus, a patient’s propensity for developing MI during or after heavy exertion appears to depend, in part, on the degree of sympathetic activity and of balance between prothrombotic and fibrinolytic effects. Despite the proposed importance of sympathetic activation and the potential net prothrombotic effect, it remains unclear whether beta blockers or aspirin decrease the relative risk of MI triggered by exertion.

Anger

The MIOS investigators interviewed 1623 individuals approximately 4 days after they experienced an acute MI to assess the intensity and timing of discrete episodes of anger (and other triggers) during the 26 h before the acute event (42). Anger was objectively assessed by the onset anger scale (a single-item, 7-level, self-report scale) and the state anger subscale of the State–Trait Personality Inventory. Based on the onset anger scale, 39 patients (2.4%) had experienced anger within the 2 h prior to onset of MI. This corresponded to a relative risk of MI of 2.3 in the 2 h following an outburst of anger relative to a control period using the case-crossover method (53). The state anger subscale corroborated these findings with a relative risk of 1.9. Of note, the relative risk of MI within the 2 h following an outburst of anger for patients on aspirin was 1.4, significantly lower than for those not on aspirin (p < 0.05), suggesting a possible role for aspirin in the prevention of anger-triggered MI onset. Reich et al. (54) noted that anger was the probable trigger for 15% of the life-threatening arrhythmias
in the 117 patients they studied. Fear, anxiety, and bereave-
ment have also been implicated in an increased risk of cardiac events.

**Mental Stress**

Acute mental stress may be a trigger of transient myocardial ischemia (53,55), MI (56), and sudden cardiac death (57,58). Bairey et al. (53) noted that 75% of 29 patients with coronary artery disease and exercise-induced myocardial ischemia also demonstrated mental stress-induced wall motion abnormalities by radionuclide ventriculography. Barry et al. (55) performed ambulatory electrocardiographic monitoring supplemented with daily records in 28 subjects with coronary artery disease. The ECG monitoring identified 372 episodes of ST-segment depression over a span of 5 to 6 weeks. At least 22% of the ischemic episodes occurred at high levels of mental stress but low physical activity. In addition, transient ischemia was more likely to occur as the intensity level of mental activity increased.

**Long-Term Cardiovascular Sequelae**

The aforementioned studies indicate that a bioterrorist attack might trigger acute cardiovascular events among those who were not directly exposed to the attack. The kinds of terrorist attacks that have occurred in the past, such as airplane hijackings and car bombings, are unlikely to trigger cardiovascular events long after the event, although any acute events (congestive heart failure resulting from nonfatal MI) triggered immediately after the event could have long-term cardiovascular effects. However, other types of potential terrorist attacks, such as exposing a population to a virus that leads to inflamed plaques or cardiomyopathy might produce a massive number of cardiovascular events long after the attack occurred.

**Preventing and Treating Cardiovascular Events During Terrorist Attacks**

**Pharmacoprevention**

Given the compelling data on the circadian variation of MI onset, most physicians provide pharmacologic protection during the morning hours for patients already receiving on treatment. Most physicians provide pharmacologic protection during the morning hours for patients already receiving anti-ischemic and antihypertensive therapy (18,19,59). Varying levels of evidence show that the 4 classes of agents most commonly administered to prevent acute coronary events (lipid-lowering agents, angiotensin-converting enzyme inhibitors, beta-adrenergic blocking agents, and aspirin) render plaques less likely to be disruptive. Lowering low-density lipoprotein levels to below 60 mg/dl, for example, appears to prevent infarction and death by stabilizing vulnerable plaques, and aspirin use has been associated with a reduced risk of anger-triggered MI onset. However, further study is needed to define the role of additional agents in trigger modulation (42).

The high likelihood that a terrorist attack will trigger acute cardiovascular events has led to questions about the need for preventive therapy in the event of a terrorist attack. The advisability of giving beta blockers or aspirin to the entire at-risk population when news of an attack is publicized must take into account the difference between absolute and relative risk of triggering. While it is true that some potent triggers, such as cocain, increase the risk of an MI 20-fold (relative risk increase), the absolute risk that a 50-year-old, nonsmoking, nondiabetic male will have an MI in any given hour is 1 out of 1 million. The absolute number of infarctions that would result from a terrorist attack, therefore, is likely to be very low. As a result, the potential benefits of mass administration of beta blockers, aspirin, or other agents to millions of individuals are likely to be low, especially when compared to the side effects associated with these agents. The fact that the incidence of cardiovascular mortality did not increase in New York City after September 11, 2001, supports this conclusion (9).

The most promising approach to the prevention of cardiovascular events triggered by a conventional terrorist attack would be to improve methods for preventing such events in general. Improved methods to detect and treat vulnerable atherosclerotic plaques would protect the population against the many potential triggers that they experience daily, as well as the rare trigger of a massive terrorist attack. Furthermore, physical conditioning could also help reduce the likelihood of a stress-induced MI.

The additional threat remains that terrorists might launch a bioterrorism attack using a microorganism or toxin that causes cardiovascular disease, either by worsening atherosclerosis or directly attacking the cardiovascular system. The best defense against such an attack is continued research on the types of agents that might be used in such attacks and the development of drugs and vaccines to counteract their effects.

**Surge Capacity for Emergency Medical Services**

A bioterrorist attack is likely to have a significant, sustained, and harmful effect on the affected community’s ability to manage everyday medical and surgical emergencies, similar to what commonly occurs following natural disasters (e.g., hurricanes, earthquakes, floods) (60–78). For example, in the 1 to 3 weeks following a major hurricane, the emergency department (ED) volume increases 17% to 40%, mostly due to an increase in patient visits relating to lacerations of all types, puncture wounds, stings, and falls (65,67). Unfortunately, sudden peaks in demand for emergency care are becoming increasingly difficult to handle because hospital surge capacity has eroded dramatically in the last decade due to: 1) decreased reimbursement by managed care organizations for inpatient care, resulting in a nationwide reduction of hospital beds, 2) the nursing shortage, 3) a more acute patient mix, and 4) a general deterioration in the health care safety net and an increase in ED visits by uninsured patients who cannot afford routine medical care (79).
Because of these factors, the EDs are overcrowded and ambulances are frequently diverted from their original destinations (80–83). The deleterious effects of ED overcrowding and ambulance diversion are most evident in patients with life-threatening cardiovascular conditions (e.g., cardiac arrest, heart attack, and stroke) for which effective, but highly time-dependent, treatments are available. Schull et al. (82) have shown that ED overcrowding is associated with significant increases in emergency medical services’ ambulance response times and transport intervals for patients with chest pain.

The effect of a terrorist attack on the health care system, and on the care of patients with cardiovascular problems in particular, will depend on the number of victims who survive and the need significant medical care, the nature of the attack, and how long the influx of patients and need for care continues. Following the World Trade Center attack on September 11, 2001, the New York metropolitan area health care system focused its attention on the hundreds of injured survivors, discharging patients with less critical problems and deferring their care. Because the event resulted in only a single burst of patients and did not threaten the hospitals directly, mortality of cardiac patients did not increase in the month following the attack compared to pre-attack control figures (9). However, on the first day of the 1991 Gulf War, the number of deaths from suffocation, asphyxiation, aspiration, MI, cardiac arrest, and cerebrovascular accidents increased abruptly in Israel, as did the number of sudden deaths associated with the use of tight-fitting masks with filters in sealed rooms. Much of the excess risk of death from cardiorespiratory complications during the first air raid alert may have been due to its duration (140 min).

A bioterrorism attack that leads to thousands or more of victims over a period of days, weeks, or months could have a catastrophic effect on health care facilities in the U.S. If the attack involved a highly infectious agent associated with high morbidity and mortality rates, particularly one for which immunization or effective prophylaxis does not exist, health care and public safety personnel (including police, firefighters, emergency medical technicians and paramedics, nurses, physicians) might not be willing to report for duty out of concern for their personal safety or to care for their own families. During such an event, Veterans Affairs and military hospitals, National Guard personnel and disaster management assistance teams, and other resources would probably be deployed, but this would typically take 12 to 24 h unless advance warning of the attack triggers activation and redeployment.

Cardiovascular (and presumably all other) emergency care would be highly compromised under such circumstances. Plans need to be developed to ensure emergency treatment and continuity of care (including supplies of needed medications) during a bioterrorist incident for those with known or emerging cardiovascular disease. Once the acute crisis subsides, more emphasis will need to be placed on managing the mental and physical effects of the attack on the survivors and community at large.

Hospital surge capacity must be increased to accommodate the long-term consequences of a widespread, ongoing bioterrorism attack. The initial focus should be on the large Level 1 trauma center facilities throughout the country, particularly those that serve areas at increased risk of a terrorist attack based on Department of Homeland Security assessment.

The most promising new concept to emerge recently is ER One, a new congressionally funded ED renovation plan developed for the Washington Hospital Center. ER One allows a standard 60- to 70-bed ED to accommodate 4 times that number of patients with a less than 30-min notice and increase its normal patient volume 10-fold with only a few hours’ notice. The design calls for rooms that are larger than standard size and can accommodate multiple patients during surges using collapsible walls to open up work areas. All medical equipment is modular and wired into the ceilings, allowing rapid influx of critical equipment in times of crisis. All public areas (corridors, waiting and registration areas, office spaces) are pre-wired and configured to be turned into patient care areas on short notice. Furthermore, all rooms are negative-pressure capable, allowing the ED to function effectively during a widespread bioterrorism attack.

Data and Health Care Policy Implications

Currently, the U.S. cannot track the incidence and outcomes of common cardiovascular emergency conditions (including heart attack, sudden death, and stroke) or quantify the direct and indirect cardiovascular effects of a bioterrorist event. A sophisticated tracking system is needed to provide early alerts concerning biological attacks, as well as outbreaks (e.g., influenza, SARS) that are not related to terrorism but could have a major impact on a population’s health, including its cardiovascular health. A real-time electronic epidemiological and syndromic surveillance system is critically needed to track the types and numbers of patients with similar symptoms and diseases, beginning in the emergency medical system and continuing through the ED and hospital phases of care.

As more and more emergency medical, ED, and hospital systems convert to electronic medical records, common data elements with standardized definitions should be linked. Software manufacturers that produce electronic medical record products should be required to collect certain uniformly defined data elements that will support this Department of Homeland Security goal and build links into their program to export these encrypted data into a real-time national surveillance system. The system should also include a method to de-identify patient data early in the process to comply with HIPAA. Although the primary purpose of such a system would be to protect national security, it could
be used to increase the quality of care and support such government programs as Healthy People 2010 (84).

**Recommendations**

1. Additional research is needed on the cardiovascular complications of vaccines to determine whether individuals who react unfavorably can be identified prospectively and whether more effective preventive and therapeutic interventions are available for those who experience such complications.

2. Additional research is needed on the links between psychological stress and acute cardiovascular events, particularly with respect to whether any additional prophylactic measures can be used for individuals at increased risk.

3. Data should be collected from ICDs at the time of a bioterrorism incident to provide critical information on the effects of the associated stress on cardiac arrhythmias.

4. Further research is needed on the chronic, long-term effects of bioterrorist attacks.

5. The federal government should provide funds to help hospitals with trauma centers improve their surge capacity using the ER-One design. Such units must be staffed with adequately trained personnel who can sustain their functions during incidents that last for weeks or months.

6. Health care providers should be trained on how to address the consequences of a bioterrorism attack, including how and when to use personal protective equipment, and what information to report and to whom. Such training is now available through nationally standardized courses in core, basic, and advanced disaster life support (85,86).

7. Provide training to increase the general public’s capability to help manage acute cardiovascular conditions. This should include recognizing acute cardiovascular conditions, calling 911 to report suspected events, and using automated external defibrillators (this is consistent with the Healthy People 2010 objectives). In addition, more large public facilities should be equipped with automated external defibrillators (87).

8. Establish a real-time, electronic epidemiological and syndromic surveillance system that can track the incidence of cardiovascular conditions (heart attack, sudden death, and stroke) that might be affected by a bioterrorist event.

9. Require manufacturers that produce electronic medical record products to collect certain uniformly defined data elements that will support this Homeland Security purpose and build links into the program that will export these encrypted data into a real-time national surveillance system.

10. Increase partnerships among multiple federal, state, local, public health, military, and private organizations to provide comprehensive, coordinated training, and response to a bioterrorist event.

11. Increase emphasis on preventing cardiovascular disease, including modifying risk factors.

**Summary**

In this report, we have discussed some of the secondary or indirect cardiovascular effects of the agents and diseases that are most likely to be used for bioterrorism, including the cardiovascular complications of the smallpox vaccine and the impact of the stress and anger produced by bioterrorism on both short- and long-term cardiovascular health. The pathophysiological processes through which bioterrorist attacks could trigger cardiovascular events include circadian variation of MI. We proposed that hospitals adopt plans, such as ER One, to increase their surge capacity quickly when needed to respond to a bioterrorist attack. In our recommendations, we called for additional research on the cardiovascular complications of the smallpox vaccine, the links between psychological stress and acute cardiovascular events, the effects of stress on cardiac arrhythmias, and the long-term cardiovascular effects of bioterrorism. Other recommendations were to train health care providers in addressing the indirect and secondary cardiovascular effects of bioterrorism, focus on preventing cardiovascular disease to reduce the risk of complications during a bioterrorist attack, and develop a surveillance system to monitor cardiovascular events associated with bioterrorism.

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APPENDIX 1. ACCF/AHA/CDC Consensus Conference Report on Emerging Infectious Diseases and Biological Terrorism Threats: Task Force II—Relationships with Industry

Dr. Robert A. Klonek declared that he received consulting fees and speaking honoraria in excess of $10,000 from Pfizer. Dr. James E. Muller declared that he is a principal at InfraReDx, Inc. The other authors of this report declared that they have no relationships with industry pertinent to this topic.