CASE REPORT

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Entrapment in Small, Enclosed Spaces: A Case Report and Points to Consider Regarding the Mechanism of Death

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ABSTRACT: The mechanism of death due to confinement in an enclosed space is usually ascribed to asphyxia from oxygen deprivation. We report the case of the decomposed remains of a 23-year-old man discovered in an unused industrial size refrigerator in which the mechanism of death is heatstroke. The investigation of the death indicates the subject most likely voluntarily entered the refrigerator and for unknown reasons, closed the door. Injuries identified at autopsy and damage to the inside of the structure indicate he struggled to exit the cabinet. The autopsy shows no significant natural disease processes and toxicology studies were negative. The diagnosis of heat stroke typically rests on the evaluation of multiple features, including the age and size of the decedent, the ambient temperature, the medical history of the decedent, whole body hydration, body fat content, alcohol and drug use, medication history, general physical condition, and many other factors. The diagnosis of heatstroke due to confinement in an enclosed container requires evaluation of the heat stress of the container, the heat strain experienced by the individual, autopsy findings suggesting signs of a struggle to exit the container, and other factors. In all such cases, a careful death investigation with correlation of autopsy findings is required to accurately determine the mechanism and cause of death. We suggest that for all such deaths, physiological and environmental factors promoting hyperthermia and heatstroke be considered as a possible mechanism of death, along with those associated with the more obvious danger of asphyxiation.

KEYWORDS: forensic science, forensic pathology, death, heatstroke, heat-related illness, asphyxia, autopsy, mechanism of death, hyperthermia, cause of death

Asphyxia is often assumed to be the mechanism of death when a body without other lethal injuries or natural disease processes is found in a small, airtight, or nearly airtight, space. Sometimes it is.

We present the case of a young man who died for other reasons when he became entrapped in an unused, closed, industrial-size refrigerator. Evidence shows that, rather than asphyxiation, the probable mechanism of death in this case was hyperthermia and heatstroke.

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As in all medicolegal death investigations, a careful scene evaluation in combination with autopsy findings is critical in establishing the mechanism(s) and cause of death. In this death the refrigerator measurements and physical characteristics, the atmospheric conditions, physical evidence of a struggle both within the refrigerator and on the body, and knowledge of oxygen consumption rates and heat production rates by the subject allow us to establish the death resulted from hyperthermia due to entrapment in an enclosed space.

In this death, we classify heatstroke and asphyxia as mechanisms of death with the underlying cause of death being entrapment in an enclosed space. This classification is based upon Hanzlick's (1) definitions of mechanisms and causes of death.

Case Report

The decomposed body of a 23-year-old, black male was found in a closed, inoperative refrigerator in the basement of an unused dormitory kitchen at a university. The man had last been seen alive five days earlier. He had a history of occupying and sleeping in small spaces, such as janitorial closets in campus buildings, which he had been consistently told to vacate by university employees. The young man had earlier been a student at the university, but at the time of his death he was disenrolled and homeless. He had no known mental health disorders. He was apparently alive and fully conscious when he entered the enclosure in which he eventually died. Clothing, a Bible, and a few other personal possessions were found in the refrigerator with the body. When found, he was dressed only in jockey-style underwear. The body was semi-recumbent with hips and knees flexed, the back against a lateral wall, and with feet resting against the opposite lateral wall.

The chamber in which the person died was a thermally insulated cabinet designed for food storage. It was unventilated and airtight when its door, which could not be opened from the inside, was closed. The kitchen area outside the refrigerator showed no evidence of a struggle. Metal supports, which extended across the inside of the refrigerator, were broken and lying across the body when it was found. The metal lining of the cabinet, near his feet, was slightly dented.

Autopsy findings included moderate postmortem decomposition with gaseous distention of the scrotum, abdomen and soft tissues, skin slippage and blistering in dependent areas, and bloody fluid purging from the mouth, nostrils, and rectum. The eyes were

shrunken and the swollen tongue protruded from the mouth. The body weight was approximately 64 kg (141 lbs), and the body was 170 cm (67 in.) long. The weight during life was believed to be about 150 to 155 lbs. Conjunctival or periorbital petechiae were not observed, however, evaluation was limited by decomposition. The tongue showed no evidence of biting, suggestive of a seizure. There were contusions on the plantar surfaces of the heels, the prepatellar regions, the sacral and lumbar regions of the back, and the dorsal surfaces of the hands and forearms. Hemorrhage in all of the contused regions extended into the skeletal muscles. There were no fractures or visceral injuries. No natural disease processes were present. Microscopic evaluation of the brain, lungs, heart, kidneys, liver, spleen, thyroid gland, pancreas, and bone marrow showed marked autolysis and decomposition changes; meaningful interpretation was precluded by these changes. Toxicology screening tests of blood for drugs of abuse and commonly prescribed drugs were negative. Neither vitreous fluid nor urine were available for chemical or toxicological evaluation.

The degree of decomposition was consistent with death occurring about five days earlier, shortly after the subject had last been seen alive. The injuries to the plantar surfaces of the heels, the prepatellar regions, the sacral and lumbar regions of the back, and the dorsal surfaces of the hands and forearms and the damage to the inside of the refrigerator intimate a struggle by the subject to free himself from the refrigerator and possibly to alert others of his entrapment within the enclosure. The police interviews of individuals who frequented the kitchen area and acquaintances of the decedent, the undisrupted kitchen in which the refrigerator was located, and the autopsy findings indicate the subject voluntarily entered the refrigerator. There were no indications that he intended to commit suicide. Why the subject closed the door remains unknown.

The mechanism of death is heat stroke with the underlying cause being entrapment in a small and enclosed space. The manner of death is classified as accident.

Discussion

We considered the following conditions and circumstances in deducing the likely mechanism of death:

1. Inside dimensions of the thermally insulated and airtight refrigerator were 58.5 in. wide by 30.25 in. deep by 49.00 in. tall, which enclosed an air volume of 86 711 cubic in. or 1421 L.
2. Two doors, arranged side-by-side, were on the front of the refrigerator. Both doors were secured shut when the body was found. The refrigerator was not turned on and the doors could not be opened from the inside. The refrigerator was made of stainless steel and the insulated walls were 1.25 to 2 in. thick. In the top half of the refrigerator, metal support struts extended between a central vertical dividing segment between the two doors, to the back of the refrigerator. Some of these were broken and lying across the body.
3. The refrigerator did not contain toxic gases or those that either react with, or displace atmospheric oxygen, so it held about 300 L of oxygen when it was first occupied. The volume of air in the chamber was about 1421 L (82.71 cubic in.), for which oxygen was initially about 21%.
4. Oxygen consumption for a normal, resting adult is about 250 cubic cm per min (15 L per hour). It is about 4000 cubic cm per min (240 L per hour) when someone exercises heavily.
5. Metabolic heat production for a normal, resting adult is about 150 W (512 Btu per hour). During moderately heavy exercise, it is about 600 W (2047 Btu per hour).
6. When the young man entered the cabinet and its door latched, he discovered it could not be opened from the inside, and he struggled violently trying to escape.

Based on these assumptions, we calculated expected rates of change for oxygen content, temperature, and relative humidity during enclosure occupancy, and the rate of rise of body heat content and temperature for the entrapped person.

Our calculations are based on direct measurements of the chamber in which the young man died and on assumptions about his state of consciousness and physical activity during his period of entrapment. No data were collected and there were no direct observations during his struggles.

Accurately evaluating information for this case requires drawing distinctions between the physiological conditions of hyperthermia and heatstroke (2). The term, "hyperthermia" designates a condition in which there is an increase in deep body temperature above about 98.6°F (37°C), a commonly accepted value for normothermia. When heat is added to the body at a rate greater than it can be dissipated, there is first an increase in total body heat content and a saturation of its thermal capacitance. There is secondarily a rise in deep body temperature, inducing graded stages of hyperthermia. Many physiological defenses are marshaled to reduce additional heat gain and to increase the rates of heat loss by thermal conduction, convection, infrared radiation, and evaporation. The person's cardiac output increases, there is a development of maximal peripheral vasodilation, and sweating reflexes bring large volumes of water to the skin surface. In most circumstances, these responses adequately limit the development of hyperthermia and forestall further increases in total body heat content and body temperature.

When deep body temperature reaches about 105°F (40.5°C), thermoregulatory reflexes become impaired, sweating ceases, and heatstroke ensues, which induces irreversible, heat-related, and circulatory damage to major organs. Death is imminent. Heatstroke is commonly recognized to develop in two forms. "Environmental heatstroke" results from prolonged exposure to intolerable environmental conditions characterized by still air with high ambient temperature and humidity. Such were the environmental circumstances faced by the young man described in this report. Also, he most likely developed "exertional heatstroke," which results from net body heat gain from high rates of metabolic heat production associated with exercise and heavy physical work.

The inferences we draw and the conclusions we make about this case also rest on distinguishing between the phenomenon of heat stress and that of heat strain (2). "Heat stress" is defined by the environmental conditions, which challenge thermoregulation. It is typically indexed by measurements of ambient temperature, relative humidity, the intensity of infrared radiation, air movement, and related environmental circumstances. Heat stress is the same for everyone exposed to the same environmental conditions.

"Heat strain" is defined in terms of the physiological cost to the person facing heat stress. It is characterized by deep body temperature, peripheral vasomotor state, cardiac output, sweating rate, and related physiological responses. The intensity of heat strain is unique for each person, even though the challenge of the heat stress is the same for all. It is not uncommon to have some people tolerate well, even enjoy, a level of heat stress, to which others, who are identically exposed, become ill, succumb, and die. Recognizing that anyone's danger in a heat stress comes not from the intensity of the heat stress itself, but from the resultant level of heat strain, clarifies why just environmental measures fall far short in providing important information about heat-induced illness and debilita-

TABLE 1—*Effects of oxygen depletion.*

Stage	Oxygen Volume (%)	Symptoms or Phenomena
…	16 to 21	None; work and exercise completed without distress
1	12 to 16	Breathing and pulse rate increased; muscular coordination slightly disturbed
2	10 to 14	Consciousness continues; emotional upsets; abnormal fatigue upon exertion; disturbed respiration
3	6 to 10	Nausea and vomiting; inability to move freely; loss of consciousness may occur; may collapse and although aware of circumstances, be unable to move or cry out
4	Below 6	Convulsive movements, gasping respiration; respiration stops and a few minutes later, heart action ceases

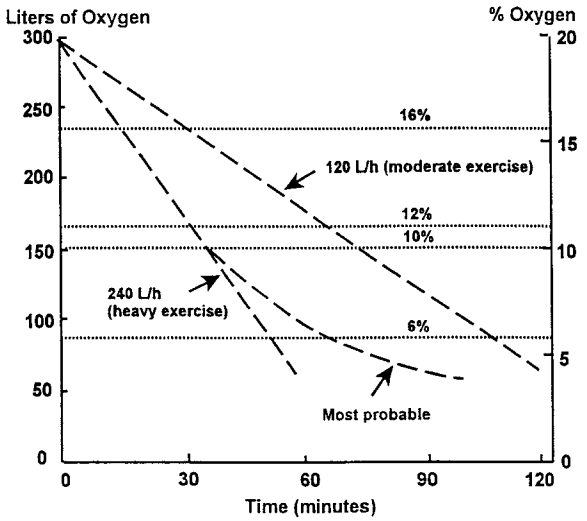

FIG. 1—*Expected changes in the volume of oxygen (left ordinate; Liters) and its percentage (right ordinate) for the chamber are shown as functions of time (abscissa; minutes) when it was occupied. Heavy exercise is predicted to decrease available oxygen more rapidly (about 240 L//h) than would moderate exercise (about 120 L/h). Its rate of depletion would be expected to lessen once oxygen availability impaired physical ability and the level of consciousness (Table 1).*

tion. People are injured and die from heat strain, not from heat stress.

Enclosure Oxygen Content

Table 1 describes the signs and symptoms for a normal person in general good health who is breathing either room air, or when it has diminished oxygen content. Data presented in figures are assumptions about the entrapped person and about his presumed level of activity prior to death. These assumptions are based upon the autopsy findings and scene investigation data. Figure 1 shows the expected time course of oxygen depletion in the occupied enclosure. Beginning with about 300 L of oxygen (about 21% of room air), the amount and percentage of oxygen would be expected to decrease depending on the rate used by the entrapped person. Because the scene investigation and autopsy findings indicate the person was struggling, we assume oxygen consumption was in the order of 240 L per hour. This would deplete oxygen in the enclosure to the point at which he would be unconscious (Table 1) by about 30 min (Fig. 1). Considering how rapidly the arterial partial pressure of oxygen equilibrates with that in alveolar gas, the effects of oxygen depletion shown in Table 1 would be expected to have an effect within seconds of exposure. Functions shown in Fig. 1 are based on reasonable values for oxygen consumption rates during rest and during exercise (3).

Data in Fig. 1 show how chamber oxygen content would be expected first to decrease rapidly during the initial period of intense struggle, but then to slow as the person weakened and consciousness waned. Figure 1 also shows how chamber oxygen consumption would be expected to fall, were metabolism at a level of only "moderate exercise," about 120 L per min. If oxygen deprivation were the only endangering factor in the enclosure, unconsciousness would be expected between 30 and 60 min, and death would likely come within 60 to 90 min.

Not only was the person trapped in the unventilated enclosure depleting chamber oxygen, he was also predictably increasing its partial pressure of carbon dioxide. At rest, its production rate would be expected to be in the order of 200 cc per min, and about 1200 cc per min were he physically active (3). Even small increases in ambient carbon dioxide partial pressure are well known to be a powerful respiratory stimulus [about 2.5 L/(Torr × min.]. The presumed and continuous increase in chamber carbon dioxide concentration was yet another burdening factor for the entrapped person. It added to the effects of the potentially lethal environment inside the chamber, which had a decreasing oxygen content (Fig. 1), increasing ambient temperature (Fig. 2), and increasing relative humidity (Fig. 3) to result in his developing hyperthermia (Fig. 4).

Enclosure Air Temperature

The enclosure in which the person died, a refrigerator, was constructed with thick, thermally insulated walls to reduce inward heat flux. Such insulation is equally effective, though, in reducing out-

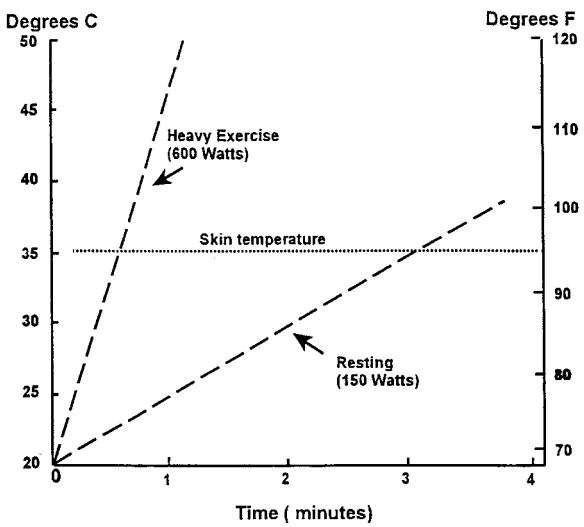

FIG. 2—*Predicted increases in chamber temperature (ordinates) are shown as functions of time (abscissa) when it was occupied. High rates of metabolic heat production associated with presumed violent escape attempts (about 600 W) would cause chamber temperature to increase more rapidly, than were the entrapped person either resting (about 150 W), or unconscious. Calculations are made assuming that the specific heat of air at 80°F (26.7°C) is: 0.2401 cal/(g × °K).*

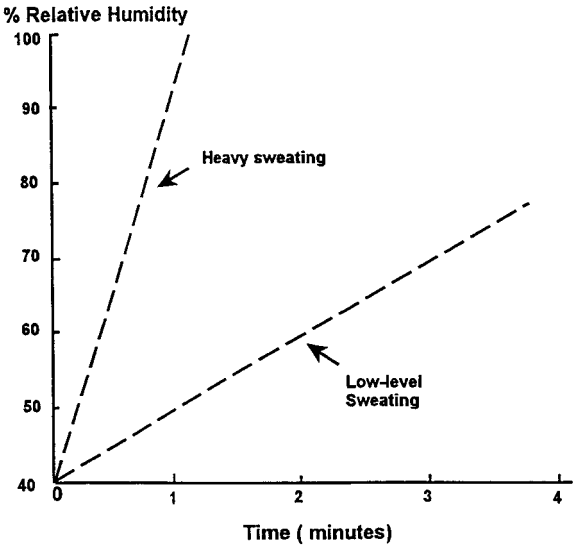

FIG. 3—*Presumed increases in the water content of chamber air (ordinate; % relative humidity) are shown as functions of time (abscissa; minutes) were the entrapped person sweating heavily (about 2L/h; 1.3 ozfl/min) during escape attempts, or sweating less heavily were he either resting or unconscious.*

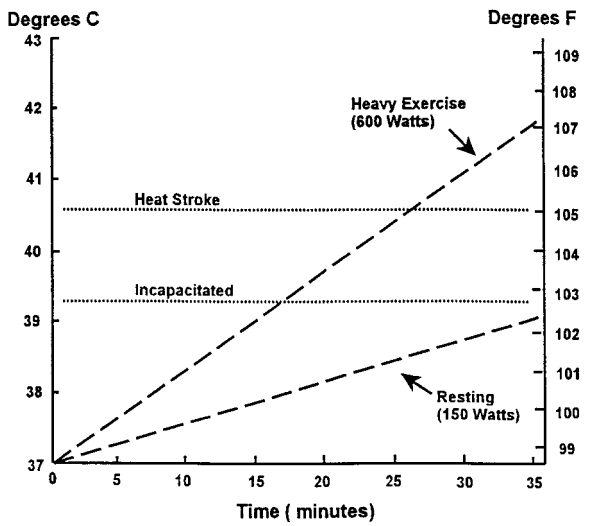

FIG. 4—*Expected increases in body temperature (ordinates) are shown as functions of time (abscissa; minutes) for the entrapped person described in this report. High metabolic heat production rates associated with vigorous escape attempts ("Heavy Exercise") would increase total body heat content rapidly, because chamber air temperature (Fig. 2) and relative humidity (Fig. 3) soon generate an intolerable heat stress, once the sealed chamber is occupied. Body temperature near 40.5°C (105°F) would predictably trigger a lethal cascade of thermophysiological debilitations during heatstroke, leading to death. Calculations are based on then assumptions that the person's body weight was 150 to 155 lbs (68 to 70 Kg), the average specific heat for body tissue is about 0.83 cal/(g × °C) (3.48 Joules/(g × °K), and the reported level of heat production associated with different levels of physical activity.*

ward heat flux. This means that most of the metabolic heat produced by someone trapped in an inoperative and unventilated refrigerator remains in it, where it either increases its air temperature, or is stored to increase body heat content and temperature. The greater the intensity of physical activity, the higher the rate of oxygen consumption, the greater the rate of metabolic heat production, and the faster enclosure air and body temperatures rise.

Data in Fig. 2 show how air temperature is predicted to have increased in the refrigerator of this case, were the entrapped person either resting, or, more likely, struggling vigorously to escape. In either circumstance, enclosure air temperature rose to the level of average skin surface temperature (35°C) within minutes to present a potentially lethal, progressively worsening, unrelenting, and inescapable heat stress. These estimates were calculated on the basis of chamber air volume, air specific heat and density, and on assumptions about the entrapped person's level of oxygen consumption (Fig. 1) and rate of metabolic heat production. It takes only a little heat to increase air temperature, because of its low specific heat and density. Inspired cold air on a winter day, for example, is normally heated to the level of deep body temperature by the time it reaches only the back of the throat.

It is of special thermoregulatory significance when air temperature is about 35°C. There is then no longer a temperature gradient between the skin surface and the surrounding air and objects along which body heat can be dissipated either by conduction, convection, or infrared thermal radiation. This is a serious threat to the protection of normal body heat content and temperature. The only physical avenue for body heat loss is then the evaporation of the water in sweat from the skin surface. If ambient relative humidity increases, this, too, disappears.

Ambient temperature in the room at the time of the scene investigation was 29°C (85°F) and it is believed to have been similar at the time the subject entered the refrigerator. Considering the thick thermal insulation of the chamber in which the young man was trapped, however, it is unlikely that air temperature outside the enclosure played a sizable role in the conditions which led to his death.

Enclosure Relative Humidity

Even though air temperature is high, body temperature can still be stabilized as long as sweat evaporates at the skin surface. Ambient relative humidity must remain below 100% for water to go from a liquid to a gas phase. Data in Fig. 3 show how water vapor partial pressure is expected to have changed in the unventilated refrigerator, once it was occupied and its door was closed. Enclosure air was saturated with water vapor within minutes after the person entered the chamber. Calculations for data in Fig. 3 were based on known factors for ambient water vapor saturation and on estimated sweating rates for someone who is physically active.

There are several reasons to expect that the person in the chamber was sweating heavily. Not only was he exposed to a hot environment (Fig. 2), but also, he was struggling to escape. Heavy sweating starts within seconds after intense exercise begins and can reach rates of 2 L per hour, or more (4), and can reduce total body water by about 10% (5). Also, it is a safe assumption that he was experiencing a growing sense of panic as he came to realize the seriousness of his situation, and as he started to feel the oppressive thermal discomfort of the enclosure. Emotional stress is a powerful stimulus for psychogenic sweating, not only on the hands and feet, as is commonly recognized, but also over the general body surface, especially at times of extreme agitation.

The effects of heavy sweating and the water vapor expelled with each respiration would be expected to rapidly increase the air's relative humidity in the unventilated enclosure (Fig. 3). Its air would reach a maximum level of water saturation within minutes, and relative humidity became 100%. The primary endangering consequence of this to the entrapped person is that he can no longer dis-

sipate body heat by evaporating the water in sweat. Only minutes after the enclosure door closed, he could no longer lose heat either by conduction, convection, infrared radiation (Fig. 2), or by water evaporation (Fig. 3). Thermoregulatory reflexes are now completely ineffective and unprotecting. The unavoidable, thermodynamic consequence is that heat produced by metabolism is now stored to increase total body heat content and temperature.

Body Temperature

Calculations to support predictions shown in Fig. 4 are based on known values for average specific heat and density of body tissue, the person's body weight (mass), and a presumed rate of metabolic heat production for his attempts to escape the enclosure. With heavy exercise inside a small, sealed, hot and humid enclosure, body temperature would be expected to rise to a thermally incapacitating level within no more than about 20 min., possibly much sooner. It would likely have increased to the point of inducing lethal heatstroke within about 30 min., or sooner.

Heat stroke is an imminently life threatening condition. Characteristically, sweating ceases, cardiac output and tissue blood flow increase dramatically to dangerous levels, and the person typically becomes delirious, manic, physically uncontrolled, and lethally hyperthermic. Unless body cooling begins immediately, tissue damage and death are unavoidable. Even if body heat content and temperature are successfully lowered, the person may die within hours to days, because of irreversible damage to organs.

Factors Affecting Heat Strain

Although not germane to the conditions of this case, characteristics of age, whole body hydration, body fat content, alcohol and drug use, medication history, general physical condition and many other factors must be considered in evaluating circumstances in which people die if thermal stress is suspected (2). Although children, for example, cannot produce heat as fast as adults do because of their smaller somatic muscle mass, they still face special thermoregulatory hazards. Because of their relatively larger body surface area-to-mass ratios and their smaller body mass for heat storage, children increase body temperature faster than adults do when exposed to the same high temperature and high humidity environments. They also cool more quickly in the same cold stress for the same reason.

The very old, much like the very young, face heat stress with compromised safety, compared to young, healthy adults. Many people over sixty-five years of age, for example, have diminished thermoregulatory capacities, which contribute to their being overrepresented with heat-induced disabilities during heat waves (6). Some have reduced sweating abilities (7), and others suffer from reduced thirst sensitivity (8), which promotes inadequate hydration. For all too many, physical fitness level is low, which disables the important first line of defense against both heat and cold stresses.

Some over-the-counter and prescription medications affect thermoregulatory ability. For example, anticholinergic drugs, some tricyclic antidepressants, and some antihistamines inhibit sweating (9). Also, some sedatives impair thirst threshold (10), some antidepressants increase metabolic heat production and induce peripheral vasoconstriction (11), and the antidopaminergic action of some antipsychotic medications directly alter thermoregulatory precision (12). Also, it is well recognized that many amphetamine-like drugs produce hyperthermia, even without environmental or exercise-induced heat stress (13).

Mechanism of Death

We conclude that heat stroke was the mechanism of death for our case. There were, however, multiple endangering factors developing in the sealed chamber for its hapless occupant. Not only was the chamber's temperature (Fig. 2), relative humidity (Fig. 3), and body temperature (Fig. 4) most likely increasing, so was its carbon dioxide partial pressure. Each presents its own physiological problems and potentially lethal consequences. Heat stroke would have most likely developed before the other potentially lethal conditions. Hyperthermia and heat stroke may have induced seizures, cardiac arrhythmias, and any number of other pathophysiological events as it progressed. We suggest that heat-induced illness and heat stroke be considered as possible factors in other situations where people die in confined spaces, especially if they are in thermally insulated enclosures, like refrigerators.

The autopsy findings of heatstroke are nonspecific. Individuals surviving longer than 24 h may show lobular pneumonia, acute tubular necrosis of the kidneys, adrenal hemorrhage, subendocardial hemorrhage, degeneration of myocardial fibers, disseminated intravascular coagulopathy, or centrilobular necrosis of the liver. If survival is less than 12 h, petechiae of serosal surfaces, including pleura, epicardium, and pericardium, may be the only abnormalities identified (14). Rhabdomyolysis, more commonly seen in exertional heat stroke, also plays a role in acute renal failure (15). Some of the early complications of heatstroke in the living patient include hypotension, shivering, seizures, coma, electrocardiographic changes, pulmonary edema, congestive heart failure, hypokalemia, hypernatremia, rhabdomyolysis, and diarrhea. Late complications of heat stroke include hyperkalemia, hypocalcemia, hyperuricemia, renal failure, cerebral edema, hyperosmolar coma, persistent neurologic deficits, thrombocytopenia, disseminated intravascular coagulation, hepatic failure, adult respiratory distress syndrome, and gastrointestinal hemorrhage (16).

Conclusion

Anyone trapped in a small, confined, unventilated and thermally insulated enclosure faces a most dangerous situation, for which survival is impossible, unless, of course, there is escape in time. If the person is conscious in the enclosure, high levels of physical activity driven by terrorized and panic-induced attempts to escape would be expected. The heat produced by such activity rapidly increases the enclosure's temperature. In addition, psychogenic and thermoregulatory reflexes, which subserve heavy sweating, and the expelled water vapor in each breath, quickly saturate the enclosure air with water vapor. The person now faces an environment in which hyperthermia and heatstroke quickly develop. Death follows shortly afterward, perhaps in as short a period as 30 min. Were someone unconscious in a small, confined space, physical activity would be low and there would be a greater likelihood for depleted oxygen to be more endangering than the heat stress. Even so, death would be expected in 1 to 2 h.

In the case presented here, the mechanism of death was related directly to the circumstances of confinement. We suggest that for all such environments, physiological and environmental factors promoting hyperthermia and heatstroke be considered as the possible mechanism of death, along with those associated with the more obvious danger of asphyxiation.

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