Hypothermia and Hyperthermia Medicolegal Investigation of Morbidity and Mortality From Exposure to Environmental Temperature Extremes

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• ***Context.*—The determination of the cause of death from exposure to extreme temperatures is a diagnosis of exclusion. Because both clinical and autopsy findings are nonspecific, a thorough investigation of the background and scene, evaluation of temporally relevant environmental conditions, and assessment of the victim's underlying state of health with appropriate laboratory studies, which frequently include autopsy, are essential to establish the cause of injury and/or death with reasonable medical probability. Individuals may encounter environmental extremes in many settings during any season. Both constitutional and external factors exacerbate the stress brought about by extreme temperature.**

***Objective.*—This article reviews guidelines for forensic investigation into environmental temperature extremes that contribute to an important seasonal grouping of morbidity and mortality in the United States.**

***Data Sources.*—Articles on clinical and pathologic aspects of hyperthermia and hypothermia were collected and reviewed.**

***Conclusions.*—Recognition of multiple risk factors predisposing humans to both cold-related and heat-related morbidity and mortality enhances prevention. Awareness of the susceptibility of these exposed at-risk individuals is crucial to investigations by both clinicians and medicolegal death investigators.**

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Without thorough review of circumstances, the diagnosis of environmentally induced hypothermia or hyperthermia is difficult to affirm clinically or at autopsy. Between years 1979 and 2002, 16 555 and 4780 deaths were ascribed to excessive environmental cold and heat exposure, respectively, in the United States.[1,2] Unprotected exposures to severe temperatures alter the usual thermoregulatory processes that function in a temperate environment. Most temperature-related illnesses or deaths from environmental exposure are preventable and often preceded by unwise choices by the victim or caregiver. Even though healthy persons may suffer from various degrees of heat or cold exposure at different times, at-risk populations are relatively predisposed to illness and death from intrinsic thermal dysregulation in extremes of ambient temperature.

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THERMOREGULATION

At normothermia (the neutral environment), the human average baseline core body temperature not only is dependent on the employed thermometric device and the sampled body site but is prone to intersubject, within-subject, and circadian variations. In clinical thermometry, the mean ''normal'' oral temperature of 98.2 ± 0.7°F (36.8 ± 0.9°C) correlates to the energy end product of all enzymatic reactions.[3] Fit humans are able to tolerate a transient variation of about ±7.2°F (4°C) in internal body temperature without impairment of physical and mental performance. Metabolism, the sum of all the cellular reactions of the body, is usually measured as the rate of oxygen consumption. A standarized estimate of metabolism is the basal metabolic rate (BMR), which is dependent on the activity of these physiologic processes to sustain euthermia. The BMR is an estimate of calories (cal, a unit of energy, raises the temperature of 1 g of water by 1°C) expended during a 24-hour period for basic and necessary metabolic functions, and amounts to 1 kcal/h for each kilogram of body weight.[4] The preoptic anterior hypothalamus of the brain functions as the internal thermostat. Through autonomic thermoreceptors, skin impulses stimulate the hypothalamus. Cutaneous vascular changes such as vasodilation or vasoconstriction, sweating or shivering, nonshivering thermogenesis, change in physical activity, and behavioral responses combine to sustain BMR and maintain euthermia. Muscular activity effectively increases heat production. Normal body temperature is the vital balance between (1) heat gain from BMR and the environment, and (2) heat loss by discrete heat-transferring mech-

Table 1. Heat Balance Equations at Equilibrium	
$H_{tot} = \pm H_d \pm H_c \pm H_r \pm H_e$	$S = M \pm C \pm R - E$
H_{tot} = total metabolic heat production	S = heat storage from heat production
H_d = conductive heat exchange	M = metabolic rate or loss
H_c = convective heat exchange	C = convective/conductive heat loss or gain
H_r = radiative heat exchange	R = radiative heat gain
H_e = evaporative heat exchange	E = evaporative cooling

anisms. Heat loss occurs at 2 levels: intrinsic from the core to the skin or respiratory tract and extrinsic from the skin to the environment. The intrinsic pathway is predominantly derived from circulating blood and maintained by subcutaneous tissues. The extrinsic pathway of heat-dissipation occurs from 4 environmentally mediated heat-transferring mechanisms—conduction, convection, radiation, and evaporation. Table 1 shows 2 mathematical heat balance equations at equilibrium.[5,6] Although the total metabolic heat production (H_{tot}) or heat storage from heat production (S) is always positive, heat exchange can be negative or positive, resulting in energy production. Once the internal thermoregulatory controls are compromised by excesses in environmental temperatures, signs and symptoms of hypothermia or hyperthermia ensue.

CONDUCTION AND CONVECTION

Conduction is heat transfer between 2 directly apposed objects with the direction of heat flow from higher to lower temperature. Conduction is 25 times greater in liquid than in air, resulting in rapid heat loss and body cooling in cases of water immersion. Subcutaneous fat in the overweight and obese serves as an insulating factor, which slows the rate of conduction.

Convection, the movement of molecules in a gas or liquid, facilitates conduction. Convection increases with rapid movement of air, exemplified by the mechanics of home appliances, such as convection ovens, fans, or air conditioners. Wind chill, the circulation of cool air, significantly increases convective heat loss and often produces deleterious effects in cold temperatures when protective clothing is ineffective.[5] Table 2 charts the revised wind chill temperature index, which was introduced on November 1, 2001.[7]

RADIATION

Absent the influence of wind and humidity, radiative heat loss via electromagnetic rays occurs when the ambient temperature is below core body temperature. Warmer environmental temperatures result in a net gain of body heat. A substantial amount of radiative heat comes from direct and indirect solar energy.[8] Radiative heat is an extremely important cause of hyperthermia in children trapped in vehicles. Significant interior vehicular temperature elevations often occur during mildly warm days, even with overcast cloudy skies, and usually rise considerably in direct sunlight.[9] Just as clouds insulate the earth from lower temperatures, layered clothing protects an individual from excessive radiative heat loss in environments cooler than core body temperature. In a comfortable, lower humidity environment with still air, most body heat loss occurs via radiation.

EVAPORATION

Evaporation is the change from liquid to gas or vapor form. Evaporative heat loss progresses by 2 mechanisms: insensible and sweating. Insensible heat loss occurs as moisture is lost from the skin and lungs in a nonsweating person at rate of 12 to 16 kcal/h. Under nonexercising conditions, normal blood flow dissipates heat at 80 to 90 kcal/h. During exercise or excessive heat exposure, blood flow increases up to 7-fold, prompting cutaneous vasodilation and perspiration.[10] Evaporation primarily results in dissipation of heat from release of energy by the conversion of liquid to gas on the skin surface. Heat loss from evaporation is limited in higher humidity because of ineffective escape of warm perspiration on the skin surface. Higher temperatures are considered more uncomfortable in humid conditions. The heat index, or the wet bulb-globe temperature, is a calculated ''perceived'' environmental temperature when humidity and heat indices are combined. Table 3 charts the heat index, the temperature the body feels when heat and humidity intermix.[11] Evaporation, occurring as efficient diaphoresis with conservation of electrolytes and, to a lesser degree, insensible heat loss, is an important factor in heat acclimatization, the en-

Table 2. Revised Wind Chill Index: Air Versus Wind Speed Producing ''Perceived'' Temperature (T)*

Wind Speed (mph)	Temperature, °F								
	40	**35**	**30**	**25**	**20**	**15**	**10**	**5**	**0**
5	36	31	25	19	13	7	1	−5	−11
10	34	27	21	15	9	3	−4	−10	−16
15	32	25	19	13	6	0	−7	−13	**−19**
20	30	24	17	11	4	−2	−9	−15	**−22**
25	29	23	16	9	3	−4	−11	−17	**−24**
30	28	22	15	8	1	−5	−12	**−19**	**−26**
35	28	21	14	7	0	−7	−14	**−21**	**−27**
40	27	20	13	6	−1	−8	−15	**−22**	**−29**
45	26	19	12	5	−2	−9	−16	**−23**	**−30**
50	26	19	12	4	−3	−10	−17	**−24**	**−31**
55	25	18	11	4	−3	−11	**−18**	**−25**	*−32*
60	25	17	10	3	−4	−11	**−19**	**−26**	*−33*

* Frostbite times: bold indicates 30 min; lightface italic, 10 min; and bold italic, 5 min.

hanced physiologic tolerance to ambient heat. Often, unacclimated victims of hyperthermia become ill because they continue to exercise in an unaccustomed, hotter environment. Acclimatization to elevated ambient heat may require a week or longer before a person physiologically adjusts to exercise or work in such an environment. Sweat production varies from 700 mL/h to 1.5 to 3 L/h in unacclimated and acclimated persons, respectively.

In summary, conduction results in 3% of total body heat loss at normothermia and 15% in circulating air.[12] Convection and radiation together account for 65% of heat loss, and evaporation contributes an additional 22% to 30%.[13] Increases in air flow, wet clothing, low or high humidity, and extremes in temperatures may potentially result in derangements of these normal thermoregulatory mechanisms.

HYPOTHERMIA

Hypothermia is characterized by the unintentional drop in a core body temperature to less than 95°F (35°C).[14] Even mild hypothermia is a medical emergency because of the rapid failure of thermoregulation and incremental decline of the neuropsychological awareness. An average of 689 deaths per year in the United States results from excessive environmental cold exposure. The highest mortality rates occur in the less temperate states of Alaska, Montana, North Dakota, and New Mexico.[1] It is noteworthy, nevertheless, that deaths because of environmentally caused hypothermia occur equally as frequently indoors as outdoors and are also recorded in temperate climates.[15,16] A debilitated elder may become hypothermic at home in temperatures as high as 70 to 75°F (22°C–24°C). Unlike secondary hypothermia, which results from diseases disrupting hypothalamic thermoregulation, hypothermia from exposure to cold (primary accidental hypothermia) supervenes when heat retention by the protective body mass and excess heat production produced by shivering are insufficient to maintain euthermia.[17] Risk for hypothermia is particularly great during prolonged exposure to subfreezing (<32°F [0°C]) ambient temperatures. The wind chill index, calculated by factoring ambient temperature, wind velocity, and thermal radiation, is an accepted, reliable measure of conditions contributing to cold stress.[18] At above-freezing temperatures, an overriding wind chill factor can hasten hypothermia. For example, at 40°F (4.4°C) in wind and rain, and at 60°F (15.6°C) in hurricane conditions, individuals have experienced hypothermia clinically. The elderly and the very young are especially susceptible to fatal cold exposure. Multiple mechanisms of action put the aged at risk for hypothermia: (1) reduced heat production because of loss of physiologic reserves in chronic disease, (2) increased heat loss from malnutrition and diminished subcutaneous muscle and fat, (3) impaired thermoregulation primarily from primary or secondary pathologies of the central nervous system, and (4) inactivity related to senescence.[13] Increased body surface area–body mass index coexists with underdeveloped thermoregulatory mechanisms in infants.[19]

A variety of commonly prescribed drugs, which include antidepressants, barbiturates, opioids, benzodiazepines, phenothiazines, and reserpine, adversely affect the body's ability to sense cold. Ethyl alcohol prompts rapid cooling by effecting continuous peripheral vasodilation and inhibiting heat production by shivering. Alcohol, whose intoxicating effects cloud appropriate decision making in cold environments, is the chemical most commonly detected in the blood of victims succumbing to primary hypothermia.[20]

Various periods of exposure to extreme cold may result in different forms of cold injury or illnesses. There are 4 interrelated types of tissue cooling—whole body, respiratory tract, cutaneous, and extremity. Whole body cooling may lead to serous injury or death. Inhalational cooling may aggravate intrinsic pathologic conditions such as ischemic heart or respiratory diseases. The last 2 forms may cause mild to serious injury of the affected regions but usually are not fatal. Clinical manifestations of peripheral and cutaneous cold exposure include both freezing and nonfreezing injuries, categorized in Table 4.[21–24]

Progressive whole body pathophysiologic alterations, which may arise secondarily, correspond to ascending grades (mild, moderate, severe, or profound) of hypothermia summarized in Table 5.[25–28] As skin and core temperatures fall to a critical degree in severe exposures, hypothalamic control of body temperature fails, and the warm-blooded individual converts functionally to a poikilotherm. The ensuing peripheral vasodilation results in a sudden rush of warmer blood to the extremities. This exaggerated heat sensation, as perceived by the mentally confused, severely hypothermic individual, ''paradoxically'' leads to the rare volitional act of undressing prior to coma and death.[29] Some investigators postulate that the first stage of hypothermia causes paralysis of the vasomotor center producing the aberrant sensation of a higher

Table 2. Extended

Temperature, °F								
−5	−10	−15	−20	−25	−30	−35	−40	−45
−16	−22	−28	−34	−40	−46	−52	−57	−63
−22	−28	−35	−41	−47	−53	−59	−66	−72
−26	−32	−39	−45	−51	−58	−64	−71	−77
−29	−35	−42	−48	−55	−61	−68	−74	−81
−31	−37	−44	−51	−58	−64	−71	−78	−84
−33	−39	−46	−53	−60	−67	−73	−80	−87
−34	−41	−48	−55	−62	−69	−76	−82	−89
−36	−43	−50	−57	−64	−71	−78	−84	−91
−37	−44	−51	−58	−65	−72	−79	−86	−93
−38	−45	−52	−60	−67	−74	−81	−88	−95
−39	−46	−54	−61	−68	−75	−82	−89	−97
−40	−48	−55	−62	−69	−76	−84	−91	−98

Table 3. Heat Index Chart: Air Temperature (T) Versus Relative Humidity Producing "Perceived" T*

	Relative Humidity, %										
T, °F	50	55	60	65	70	75	80	85	90	95	100
110	**152**										
105	**135**	**142**	**148**								
100	*120*	*126*	**130**	**138**	**141**						
95	*107*	*110*	*114*	*119*	*122*	**130**	**132**				
90	96	98	101	*102*	*108*	*109*	*112*	*117*	*119*		
85	88	89	90	91	93	95	97	99	101	*105*	*108*
80	81	81	83	83	84	86	86	87	88	89	91
75	74	75	75	76	76	77	77	78	79	79	80
70	68	69	69	70	70	70	71	71	71	71	72

* Italics indicate danger; bold, extreme danger.

Table 4. Peripheral Cold Injuries*

Nonfreezing injury
- —T above freezing
- —Repeat exposures

Chilblain (pernio)
- —Mild neuronal/endothelial injury
- —Dry conditions
- —Hands/feet in patients of Raynaud phenomenon
- —Repeat vascular spasticity/inflammation → nodules, ulcers, plaques

Trench foot
- —Immersion foot injury
- —Wet environments
- —Constant vasoconstriction → ischemia, bullae, ulcers, pain, ↓ function, gangrene

Freezing injury
- —Tissue T < 0°C causing ice crystals/cellular architectural disruption
- —Microvascular blood stasis and thrombosis

Superficial frostbite
- —Injury of superficial epidermis/dermis
- —No tissue loss

Deep frostbite
- —Injury involves deeper subcutaneous and musculoskeletal elements
- —Deep frostbite graded like burns

First degree (Grade I)
- —Most commonly involves exposed areas of the face, fingers, and toes
- —Erythema and anesthesia
- —Rewarming generally reverses process

Second degree (Grade II)
- —Cutaneous vesiculation, edema, and erythema
- —The frozen epidermis is white and waxy

Third degree (Grade III)
- —Hemorrhagic vesiculation secondary to microvascular injury and thrombosis

Fourth degree (Grade IV)
- —Subjacent musculature, connective tissue, and bone injury

* T indicates temperature; →, leads to; ↓, loss.

body temperature.[30] A variant of this phenomenon, termed the hide-and-die syndrome, arises when the strange location of the body at the scene suggests to investigators that the deceased attempted concealment.[31]

Only in rare cases are there reports of survival without profound brain injury following extreme hypothermia, most commonly seen in children submerged in frigid water for as long as 30 minutes. If the victim is rewarmed promptly, death may not result. Two individuals survived severe hypothermia despite recorded core body temperature at 57.6°F (14.2°C) and 56.7°F (13.7°C), respectively.[32,33]

In fatal cases, the skin of the extremities may exhibit patches of poorly circumscribed pink to red-brown discoloration in association with bright red livor mortis (frost erythema), which may be confused with carbon monoxide or cyanide intoxication. The red color of the skin and soft tissues is attributed to the increased percentage of oxyhemoglobin in the blood engorging the vasculature.[20,34] Cutaneous vesiculation may accompany hyperemia and edema. Abrasions on the palms of the hands and flexor surfaces of the arms and legs often are associated with stumbling.[35] Other nonspecific findings at autopsy may include multifocal hemorrhages of the gastric and duodenal mucosa (termed *Wischnewski spots*), hemorrhagic pancreatitis, and hemorrhage of the iliopsoas muscle.[36–38] However, in the majority of cases, the gross and microscopic findings are nondiagnostic: only pulmonary edema may be present.

In presumptive hypothermic deaths, the investigator should conduct a scene investigation as early as possible. Determination of the rectal temperature by a nonstandard thermometer specially calibrated from 84°F to 108°F (28.9°C–43.2°C) at this stage may assist in the diagnosis.[39] With regard to forensic investigation of deaths presumed to result from exposure to cold, the most important part of the postmortem evaluation of hypothermia is the assessment of the environmental conditions prevalent during the relevant window of time as part of the scene in-

Table 5. Signs and Symptoms With Grades of Hypothermia*		
Hypothermia Grade	**Core Temperature**	**Signs and Symptoms**
Mild	90°F–95°F (32.2°C–35°C)	—Confusion progressing to impaired judgment and apathy —Unable to perform complex motor functions and ataxia —''Umbles''—stumbles, mumbles, fumbles, and grumbles —Vigorous shivering, tachycardia, tachypnea, bronchospasm, peripheral vasoconstriction, increased cardiac output, hypertension —Cold diuresis
Moderate	82.4°F–90°F (28°C–32.2°C)	—Dazed consciousness, irrational behavior, slurred speech, delirium, hallucinations —Paradoxical undressing; hide and die —Hyporeflexia; rigidity; shivering reduced —Hypoventilation —Progressive decrease in cardiac output and pulse; atrial and ventricular cardiac dysrhythmias; J-wave ECG changes
Severe	72°F–82°F (<28°C)	—Waves of shivering interspersed with lengthening dyspnea followed by apnea —Decreased blood pressure, pulse, and cardiac output; ventricular dysrhythmias —Coma
Profound	<68°F (<20°C)	—Flat encephalogram (EEG) —Bradycardia progressing to asystole

* ECG indicates electrocardiogram.

vestigation, a thorough review of contributing medical and social history, and analysis of postmortem toxicology.[4]

HYPERTHERMIA

Hyperthermia—as distinguished from fever (temperature > baseline euthermia) and hyperpyrexia (temperature >106.7°F [>41.5°C]), which characteristically occur in concert with an increased hypothalamic set point from severe infection or central nervous system hemorrhage—is diagnosed clinically by a core body temperature more than 104°F (>40°C) and occurs when the body's thermoregulatory mechanisms are no longer capable of effectively dissipating heat.[40] In high ambient temperatures, evaporation is the most efficient mechanism for mediating heat loss, yet it is ineffective with humidity levels of more than 75%. Excessive heat retention results in hyperthermia or heat illness of varying degrees of severity.

Well-defined constitutional and environmental factors place persons at risk for hyperthermia. The elderly, the very young, and chronic alcoholics are less likely to mount a proper thermoregulatory response to excessive heat. Underlying medical conditions such as hyperthyroidism, obesity, and burns complicate the usual liberation of heat because of the increased metabolism, decreased activity, increased subcutaneous fat, or inability to sweat properly. Various classes of licit and illicit drugs, among which are anticholinergics, antihistamines, antidepressants (monoamine oxidase inhibitors, tricyclics), antiparkinsonian, antipsychotics (butyrophenones, phenothiazines, thioxanthenes), alcohol, amphetamines, phencyclidine hydrochloride, cocaine, and lysergic acid diethylamide, can raise the metabolic rate and cause hypohidrosis resulting in increased heat production. Diuretics, used often by elders, give rise to hypovolemia. Social and environmental factors, such as bed confinement, living on the top floor of an apartment building, lack of access to air conditioning, and social isolation with the inability for self-care, all predispose to preventable, yet often deadly, hyperthermia.[41]

Lack of heat acclimatization is frequently associated with exercise and work-related hyperthermia in hot conditions. Acclimatization, incremental tolerance to a warmer environment, requires 1 to 2 weeks. At rest, BMR generates 100 kcal/kg per hour. Without proper dissipation of heat, the increased heat production can result in body temperature increase at a rate of 2.0°F/h (1.1°C/h). Unacclimated individuals undergoing strenuous activity generate and retain heat energy up to 1000 kcal/kg per hour, which may result in severe heat illnesses or death. Initially, exposure to excessive heat raises the body temperature because inadequate sweat production or anhidrosis yields inappropriate evaporative heat loss to keep the body normothermic. During small periods of ''protected'' exposure, core temperatures adjust and the individual can gradually tolerate the heat longer. In response to the heat, sweating occurs faster, at lower temperatures, and more voluminously. For example, a fully acclimated worker (body weight = 70 kg) may lose sweat up to 12 L/d (up to 2–3 L/h, as opposed to 1 L/h in the unacclimated).[6]

Heat illness is classified according to increasing degrees of severity outlined in Table 6.[42–44] Mild heat illnesses are generally not life threatening. Heat exhaustion and heat stroke are more serious manifestations of heat illnesses. Heat stroke, subclassified as either classical or exertional type, is a life-threatening illness characterized by core body temperature more than 102°F (>40°C). If the core body temperature is not rapidly reduced in either type of heat stroke, significant morbidity and mortality occurs in 30% to 80% of affected people.[45]

In the United States between 1979 and 2002, 4780 heat-related deaths resulted from environmental weather conditions. Six percent of deaths involved children younger than 15 years; 50% in 15- to 64-year-olds, and 44% in elders older than 65 years.[2] Epidemiologic studies underscore the seasonal pattern of heat-related deaths, which peak predominantly during heat waves. For example, 1094 deaths in Italy were attributed to heat-related causes during the heat wave of summer 2003, constituting a 23% increase in mortality compared to previous years.[46]

Hyperthermia deaths in children younger than 4 years often coexist with vehicular entrapment. Two hundred thirty children younger than 15 years died between 1998 and 2004. From January to August 2005, 28 children left in cars died from complications of environmental hyperthermia.[47,48] In a recent study of 171 fatalities of children dying in vehicles from hyperthermia, 27% gained access to an unlocked car, and 73% were either intentionally or

Table 6. Heat Illnesses*

Heat Illness	Signs and Symptoms
Heat edema	—Swollen hands, feet, and ankles with prolonged sitting or standing in hot weather
Heat rash (miliaria rubria, lichen tropicus, or prickly heat)	—Pruritic vesicular rash resulting from sweat forced through ductal walls into surrounding tissues instead of intraductal pathway from gland to skin surface
Heat cramps and heat syncope	—Fluid and electrolyte loss from profuse sweating without adequate hydration —As pH changes, tetany and painful muscular spasms arise —Heat syncope: a vasovagal response to intravascular volume depletion and peripheral vasodilation —Respond well to prompt rehydration
Heat exhaustion	—Significant fluid and electrolyte depletion with decreased cardiac output and massive peripheral vasodilatation —Flulike symptoms include headache, blurred vision, fatigue, nausea, vomiting, chest pain, anxiety, and confusion —Core body T > 100.4°F (38°C) —Prompt fluid and electrolyte treatment deters progression to heat stroke
Classical heat stroke	—Elderly or chronically ill passively affected in heat wave situations —Symptom triad: hyperpyrexia, anhydrosis, and mental status changes
Exertional heat stroke	—Occurs in unacclimated young people involved in vigorous physical activity in hot weather —Second most common cause of death in high school athletes —Tachycardia, tachypnea, hypotension, and mental status alterations —Persistent sweating in 50% victims —Elevated muscle and liver enzymes and creatinine secondary to rhabdomyolysis, hepatic and renal injury

* T indicates temperature.

Table 7. Exterior and Interior Temperature Range (°F) in Cars at Various Times

	Noon		3:00 PM		5:00 PM	
Location	Outside	Inside	Outside	Inside	Outside	Inside
Passenger compartment						
Blue car	82–92	104–128	87–96	115–132	89–97	117–136
White car	82–92	102–126	87–96	109–128	89–97	114–132
Trunk						
Blue car	89–92	102–126	87–96	112–131	89–97	112–128
White car	86–92	100–108	87–96	100–118	89–97	101–112

accidentally left in cars by an adult.[49] The ambient temperature within a parked car on a temperate, sunny day can reach dangerous temperatures within minutes, resulting in heat stroke and death.[50] A temperature profile analysis of the vehicle under similar environmental conditions of entrapment is a critical component of the investigation. Table 7 illustrates the effective interior temperature increase in cars secondary to solar heating.[9]

As in hypothermia, diagnostic findings at autopsy are absent or nonspecific. With brief survival these consist of serosal (pleural, epicardial, pericardial) and cerebral periventricular petechiae and pulmonary and/or cerebral edema. If survival is prolonged, measured in hours before death, laboratory, macroscopic, and histopathologic findings vary greatly and may consist of acute renal failure with rhabdomyolysis, acute tubular necrosis, and disseminated intravascular coagulation; hepatic failure with centrilobular necrosis; acute pancreatitis; pulmonary edema with diffuse alveolar damage; and cerebral edema with diffuse neuronal injury.[51]

The position paper published by the National Association of Medical Examiners supports the diagnosis of heat-related deaths when the following criteria are met: ''. . . [A] death in which exposure to high ambient temperature either caused the death or significantly contributed to it. . . . [b]ased on a history of exposure to high ambient temperature and the reasonable exclusion of other causes of hyperthermia.''[52] These guidelines allow the diagnosis when the passage of time since death or putrefactive decomposition of the body reasonably precludes the measurement of core body temperature. The National Association of Medical Examiners position paper does not require a complete autopsy for diagnosis if sufficient circumstantial evidence is present and other causes of death are reasonably excluded. However, toxicologic analysis of blood and urine and evaluation of vitreous humor chemistry for electrolytes may aid in determining additional factors contributing to death.

COMMENT

Each year in the United States approximately 182 deaths are attributed to excessive environmental heat and 700 to cold. Roughly one half of the cases of hypothermia resulted from exposure in extremely cold weather conditions.[53,54] Unprotected or prolonged exposure to excessive ambient temperatures deleteriously affects the homeostatic thermoregulatory mechanisms in humans. Although elevated heat irreversibly denatures proteins, extreme cold drastically slows cellular reactions. The difference in these pathophysiologic endpoints partially explains why few

persons are successfully resuscitated from advanced hyperthermia, as contrasted to treatment for hypothermia by prompt rewarming, which may effectuate viable resuscitation. However, a much greater number of people die from hypothermia in the United States each year. The National Association of Medical Examiners position paper defines hyperthermia-related mortality with a view of achieving more accurate death certification. Inasmuch as 4 times as many deaths result from exposure-related hypothermia, which are also distinguished by a lack of specific autopsy findings, criteria in determining the cause of death should be considered in a similar fashion.

On completion of the medicolegal death investigation, the official must issue the death certificate, which documents the fact of death and provides information both for vital statistics in support of public health policy and for the administration of justice. In the United States, the process requires accurate formulation of the cause-of-death statement (Part I and/or Part II) and assignment of the manner of death (§29).[55] Under most circumstances in which there is no investigative evidence to do harm, deaths attributed to environmental hypothermia or hyperthermia are classified as accident (International Classification of Diseases, 9th Revision Codes: E900.0 [excessive heat from weather]; E900.1 [excessive heat from man-made conditions]; E901.0 [excessive cold from weather]; E901.1 [excessive cold from man-made conditions]).[56,57] Homicide, which as a classification listed on the death certificate does not necessarily imply criminal culpability, may be the appropriate manner of death in instances of neglectful supervision by the caregiver of an elder (International Classification of Diseases, 9th Revision codes: E968.4)[58] or a child (International Classification of Diseases, 9th Revision codes: E967). As of August 2005, 9 states have legislation regarding children left in cars, and 9 others are considering proposals to address this form of caregiver neglect.[59] As an example, Texas law defines neglectful supervision as a felony if a child younger than 7 years is left unsupervised in a motor vehicle for more than 5 minutes and injury results.[60]

Accidental and other manners of death from both environment-caused hypothermia and hyperthermia are usually preventable. Sound public health policy designed to reduce morbidity and mortality attributable to environmental extremes should promote better public awareness and education about intrinsic and environmental risk factors that contribute to such deaths. The medicolegal official through comprehensive investigation and accurate death certification plays a necessary role in support of the goal of achieving improved public health.

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