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Hypothermia in Critically Ill Dogs and Cats

Animal Emergency Center, Glendale, Wisconsin
Andrea K. Oncken, DVM
Rebecca Kirby, DVM, DACVECC, DACVIM
Elke Rudloff, DVM, DACVECC

KEY FACTS

- The four basic mechanisms of heat loss from the body are convection, conduction, radiation, and evaporation.

- Primary hypothermia occurs as a result of exposure to a cold environment; secondary hypothermia occurs with an alteration in the body's normal thermoregulatory response.

- In cases of severe hypothermia, it is recommended to replace intravascular volume and rapidly rewarm patients to a temperature of 37°C (98.5°F) while constantly monitoring for complications and supporting vital organ function.

ABSTRACT: Primary hypothermia occurs as a result of exposure to low environmental temperatures. Causes of secondary hypothermia are multifactorial and include underlying disease, trauma, toxins, immobility, surgery, and anesthesia. We have classified secondary hypothermia as mild, moderate, severe, or critical, with the severity of clinical signs relating to the degree of hypothermia. Potential complications of hypothermia include coagulopathy; acid–base, electrolyte, and central nervous system disturbances; and cardiovascular and respiratory depression. Controlled volume resuscitation and aggressive rewarming are recommended. Rewarming modalities include passive surface, active surface, and active core rewarming. Potential rewarming complications should be anticipated.

Hypothermia is a common complication in emergency rooms and intensive care units that can hinder resuscitative, therapeutic, and diagnostic efforts. Hypothermia can be defined as a subnormal body temperature in a homeothermic organism.[1] Primary hypothermia occurs in the presence of normal heat production and usually results from exposure to a cold environment. Secondary hypothermia occurs from illness, injury, or drug-induced alterations in heat production and thermoregulation.[2] Severe or critical secondary hypothermia can increase morbidity and mortality in critically ill animals.

CLASSIFICATION OF HYPOTHERMIA

Primary and secondary hypothermia in dogs and cats have been previously classified as mild (32°C to 37°C or 90°F to 99°F), moderate (28°C to 32°C or 82°F to 90°F), severe (20°C to 28°C or 68°F to 82°F), or profound (less than 20°C or 68°F; Table 1).[3] This classification is more accurate for primary hypothermia. In a recent retrospective review of 55 dogs and 77 cats with secondary hypothermia,[4] a different correlation was found between body temperature and clinical signs than reported above. With secondary hypothermia, adverse effects were reported at higher temperatures than were previously published. From this data, a new classification of secondary hypothermia in dogs and cats is presented (Table 1). Whether hypothermia is primary or secondary,

TABLE 1		
Classification and Clinical Signs of Primary and Secondary Hypothermia		
Category	*Temperature*	*Clinical Signs*
Primary hypothermia		
Mild	32°C–37°C (90°F–99°F)	Shivering, peripheral vasoconstriction, increased metabolic rate, increased cardiac output, piloerection, heat seeking
Moderate	28°C–32°C (82°F–90°F)	Decreased heart rate, decreased respiratory rate, weak pulse, muscle stiffness, hypotension, CNS depression
Severe	20°C–28°C (68°F–82°F)	Peripheral vasodilation, atrial and ventricular arrhythmias, coagulopathies, severe CNS depression or coma, absent pupillary light responses, absent corneal reflex
Profound	<20°C (<68°F)	Asystole
Secondary hypothermia[a]		
Mild	36.7°C–37.7°C (98°F–99.9°F)	Normal (dogs and cats) to increased (dogs) heart rates, normal MAP, normal respiratory rate, normal level of consciousness
Moderate	35.5°C–36.7°C (96°F–98°F)	Decreased MAP, decreased heart rate (cats), increased heart rate (dogs), mental dullness
Severe	33°C–35.5°C (92°F–96°F)	Decreased heart rate, decreased MAP, respiratory depression, severe CNS depression
Critical	<33°C (<92°F)	Moribund, may appear dead, high mortality rate

[a]Oncken AK, Kirby R, Rudloff E: Effects of secondary hypothermia in dogs and cats admitted to the emergency room. Unpublished data, Animal Emergency Center, Glendale, WI, 2001.
CNS = central nervous system; *MAP* = mean arterial pressure.

the basic mechanisms that lead to heat loss from the body are the same.

MECHANISMS OF HEAT LOSS

There are four basic mechanisms of heat loss from the body (Figure 1):

- **Convection** is the transfer of heat from the body surface to air moving past the animal.
- **Conduction** is the transfer of heat from the body surface to colder objects (e.g., cold table surfaces, cage floors) in contact with the skin.
- **Radiation** is the exchange of heat between the body and objects in the environment that are not in contact with the skin, independent of the temperature of the intervening air.
- **Evaporation** occurs when moisture (e.g., surgical preparation solutions) that is in contact with the skin or respiratory tract dissipates into the air, pulling heat with it; animals rely on evaporative heat loss through the respiratory tract when they pant.

Cutaneous heat loss is largely a function of body surface area, with a greater exposed surface area leading to greater conduction of body heat into the environment. Because metabolic heat production is a function of body mass, a larger body mass results in increased heat production.[5,6] Small dogs, cats, neonates, and cachectic animals have a higher surface area:body mass ratio, which makes them more susceptible to rapid heat loss and less efficient at compensatory heat production.

Any impairment of an animal's ability to mount an adequate behavioral response to hypothermia will also lead to increased heat loss. This is a problem for geriatric and neonatal animals and animals with severe debilitating disease or injuries because they may be less able to escape from a cold environment. The ability of the animal to compensate for hypothermia is determined by the degree of hypothermia, size and mobility of the animal, presence of underlying disease, and the success of the animal's thermoregulatory mechanisms.

THERMOREGULATION

Hypothermia occurs from increased heat loss, decreased heat production, or a combination of both. A homeothermic animal's core body temperature remains nearly constant at all times, whereas peripheral body temperature changes constantly with alterations in the temperature of the immediate environment.[7] The temperature regulatory center in the posterior hypothalamus controls the body's compensatory responses, regulating core body temperature (Box 1). Heat production is largely a by-product of cellular metabolism, and body tissues
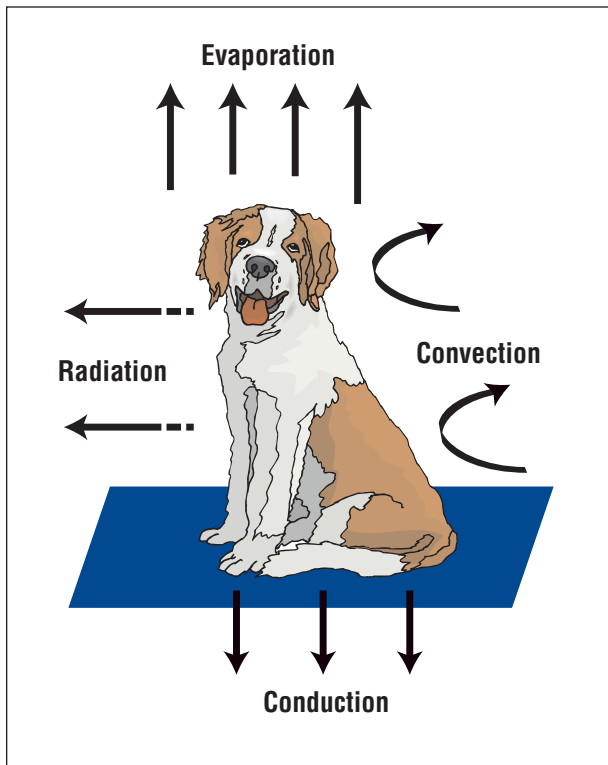
Figure 1—Methods of heat loss from the body include conduction, convection, radiation, and evaporation.

produce heat in proportion to their metabolic rates.[5,8,9] Heat is lost when there is a transfer of core heat into the skin with subsequent loss into the environment. Heat loss is also related to the conductive properties of the tissues. Fat is an effective insulator, conducting only one third as readily as other tissues; therefore, it slows the transfer of body heat.

Heat is conducted through the blood vessels in the skin and subcutaneous tissues (Figure 2). Blood supply to the skin and subcutaneous tissues consists of venous plexuses that are supplied by skin capillaries and muscular arteriovenous anastomoses.[10] Vasoactivity of these vessels is under the control of the hypothalamus through the autonomic nervous system. The rate of blood flow through these arteriovenous anastomoses can vary from less than 1% up to 30% of cardiac output, depending on the degree of vasoconstriction or vasodilation desired. Increased blood flow leads to increased heat loss, whereas decreased blood flow conserves heat within the core. There can be an eightfold increase in heat conductance between the fully vasoconstricted state and the fully vasodilated state.[11]

The hypothalamus has a set point (critical temperature level) that all temperature control mechanisms work to maintain. This set point can be decreased as an

adaptive mechanism to protect the body from severe external stress or systemic illness, leading to secondary hypothermia (Figure 2).

CAUSES OF SECONDARY HYPOTHERMIA

Mild to moderate secondary hypothermia is often endogenously triggered in severe metabolic disease as a protective mechanism to decrease energy expenditure and oxygen use. This conserves energy required by the body's vital systems (e.g., brain, heart) during times of extreme energy deficit.[12–14] This degree of hypothermia is protective only as long as there is no impairment in thermoregulation. Severe metabolic disease can cause cachexia and decrease metabolic rate, which can impair the ability of the thermoregulatory center to regulate compensatory heat production. Consequently, heat loss can predominate, resulting in severe or critical secondary hypothermia.

Thermoregulation can also be impaired by other factors, including toxins and anesthetic agents. Some of these agents cause direct peripheral vasodilation, further increasing heat loss (Box 2). In controlled situations, hypothermia can be beneficial.

METABOLIC BENEFITS OF HYPOTHERMIA

Hypothermia has been used clinically for its protective effects in head trauma, hypovolemic shock, and cardiothoracic surgery. The brain can sustain 5 to 6 minutes of ischemia in the normothermic state, and this time doubles with each 5°C (9°F) drop in core body temperature.[15] Therefore, during periods of low blood flow, the brain is less likely to suffer the effects of hypoxia during hypothermic conditions.[14] Short periods of hypothermia may be advantageous in some cases of trauma-induced hemorrhage by protecting the heart and brain against ischemia until blood volume and hemostasis can be restored.[16] Although hypothermia can have protective effects during low-flow states, it can also have many deleterious effects in critically ill animals.

COMPLICATIONS OF HYPOTHERMIA

When core body temperature falls below 34°C (94°F), thermoregulation becomes impaired. Animals with this degree of hypothermia will cease to shiver or seek heat. Peripheral vasoconstriction is replaced by vasodilation, and core heat continues to be lost. Heat production decreases because the rate of chemical heat production in cells is depressed due to the decreased metabolic rate. Severe hypothermia also depresses the central nervous system (CNS), causing the hypothalamus to become less responsive to hypothermia. When the body core temperature drops below 31°C (88°F), thermoregulation ceases.[11] Metabolic consequences can be reflected

BOX 1
The Thermogenic Response

Hot and cold receptors are located in the preoptic and anterior hypothalamic nuclei. Temperature receptors are located in the skin, spinal cord, abdominal viscera, and surrounding the great veins. When the receptors sense decreasing temperatures, sympathetic centers in the posterior hypothalamus are stimulated, resulting in vasoconstriction of the peripheral arterioles, piloerection to trap heat next to the body, and increased heat production. Increased heat production is accomplished by increased muscle activity (i.e., shivering), sympathetic stimulation of cellular metabolic rate (chemical thermogenesis), and thyroxine secretion, which further increases the cellular metabolic rate.[11]

The set point of the hypothalamus can be influenced by external factors (e.g., fever, systemic illness, external stressors). The normal thermogenic response can be disrupted at the hypothalamic level or the peripheral level by toxins, metabolic disease, endocrine disease, central nervous system (CNS) disorders, certain drugs, and severe hypothermia. Severe secondary hypothermia will lead to an increased sympathetic response. There is, however, a cold-induced decreased receptor responsiveness to catecholamines, resulting in vasodilation; CNS depression; and decreased heart rate, mean arterial pressure (MAP), respiratory rate, and heat production. These clinical signs are worse with critical hypothermia because baroreceptors become nonresponsive to decreasing MAP and no sympathetic response occurs.
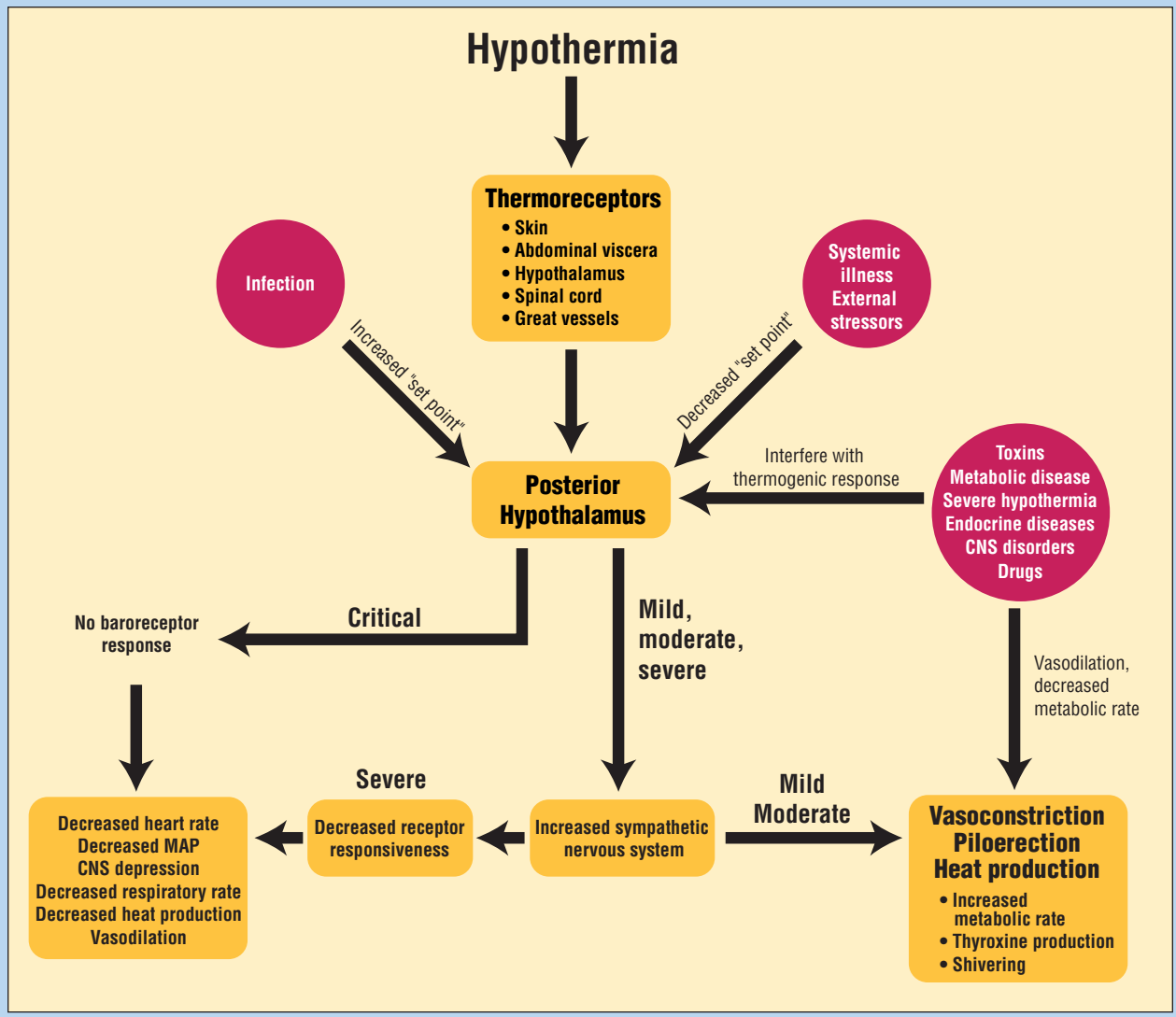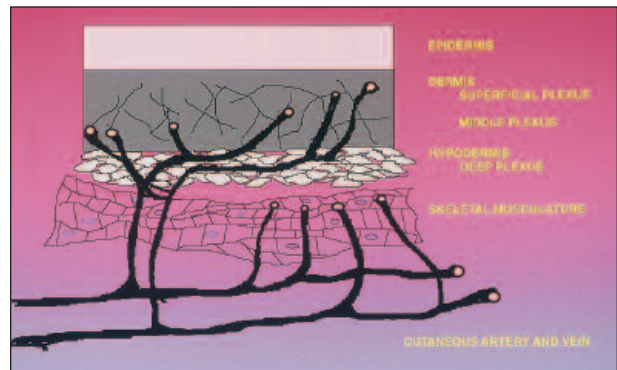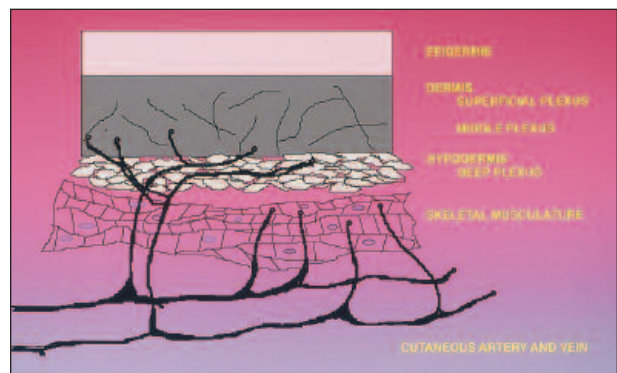
Figure 2A—Normal blood supply.

Figure 2B—Arteriolar vasoconstriction.

Figure 2C—Dilation of vessels.

Figure 2—(**A**) Normal blood supply to the canine skin. (**B**) In mild to moderate hypothermia, the thermogenic response leads to arteriolar vasoconstriction, preserving heat in the core. (**C**) As hypothermia progresses, the vessels dilate, leading to hypotension and further heat loss.

in the serum biochemistries or organ function tests. The cardiovascular, respiratory, and immune systems will become depressed and compromised (Table 2).

CLINICAL PATHOLOGY CONSEQUENCES

Serum biochemical changes induced by hypothermia

BOX 2
Causes of Hypothermia

Heat Loss
- Exposure to a cold environment
  - Cold water immersion
  - Cold table or surface
- Inadequate behavioral response
  - Geriatrics
  - Neonates
  - Injury/trauma
  - Debilitating diseases
  - Surgery/anesthesia
- Decreased body fat
- High surface area:body mass ratio
- Burns
- Open wounds
- Evaporation of antiseptic agents
- Inhalation of cold gas
- Administration of cool intravenous fluids
- Peripheral vasodilation
  - Anesthetic agents
  - Toxins

Decreased Heat Production
- Toxins
  - Ethylene glycol
  - Alcohol
- Anesthetic agents
  - Barbiturates
  - Phenothiazines
- Endocrine diseases
  - Hypothyroidism
  - Hypoadrenocorticism
  - Panhypopituitarism
  - Insulinoma
- Central nervous system disorders
- Cachexia
- Cardiac disease
- Decreased metabolic rate
  - Severe metabolic disease
  - Anesthesia
  - Shock
- Immobility
  - Unconsciousness
  - Trauma/debilitation

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can vary widely depending on the severity of hypothermia and the underlying condition of the patient. Hypoglycemia can occur with severe and critical hypothermia and further decrease metabolic activity, enhancing hypothermia. Potassium alterations can worsen or interfere with treatment of arrhythmias or perfusion abnormalities in patients with hypothermia and hypotension.

Coagulation abnormalities can be overlooked in the clinical setting because most coagulation tests are conducted at 37°C (98.6°F). This may prevent identification of the coagulopathy present at hypothermic temperatures. Coagulopathy and platelet dysfunction can present a serious complication in surgical or posttraumatic patients at risk for hemorrhage. Hypothermia-induced coagulation disorders will rapidly reverse with return to normothermia.[36]

CARDIOVASCULAR CONSEQUENCES

α_1-Adrenergic responsiveness decreases in dogs and cats with decreasing core temperatures.[23,24] With decreasing temperatures there is an early temperature-dependent increase in α_1-receptor binding to norepinephrine, followed by a decreased receptor affinity for norepinephrine at lower temperatures and a subsequent decrease in contractile response.[25] This may indicate a temperature-dependent change in receptor conformation, leading to decreased arterial responsiveness to cate-

cholamines. Therefore, normal thermoregulatory induced vasoconstriction is lost at lower temperatures, and arterial vasodilation occurs.

A study that evaluated baroreceptor responses in rabbits during hypothermia induced by direct blood cooling showed an increase in sympathetic nervous system activity as core temperatures dropped to 28°C (82°F) with little change in the baroreceptor reflex.[27] As temperatures dropped below 25°C (77°F), there was a decrease in the sympathetic nervous system response and a corresponding decrease in heart rate and blood pressure. When temperatures reached 22°C (71.6°F), the baroreceptor reflex was almost completely abolished.

It appears that both decreased receptor responsiveness and decreased catecholamine release are responsible for the cardiovascular changes that occur with hypothermia. The decreased catecholamine release may occur with more severe temperature decreases, due to reduced baroreceptor response. The increased blood viscosity and metabolic acidosis that accompany hypothermia can also decrease myocardial function.[30]

Cardiac function in dogs with experimentally induced hypothermia showed an initial period of increased contractility followed by decreased contractility at low temperatures (less than 20°C or 68°F).[37] Ventricular fibrillation was noted in 50% of the dogs with temperatures below 23.5°C (74°F).

RESPIRATORY CONSEQUENCES

Ventilatory depression can occur when the core body temperature drops below 28°C (77°F), leading to a decreased respiratory rate, minute ventilation, and tidal volume.[14,31] This, combined with alveolar hypoventilation and impaired capillary blood flow secondary to increased blood viscosity, can result in hypoxemia. Pulmonary edema, pneumonia, and acute respiratory distress syndrome may further compromise the respiratory system during advanced hypothermia.[17]

NEUROLOGIC CONSEQUENCES

For controlled periods of cardiopulmonary arrest, hypothermia can be protective to the brain. Severe CNS depression and even coma, however, may occur with progressive hypothermia.

Mild hypothermia disturbs cerebral autoregulation,

TABLE 2
Potential Complications of Hypothermia and the Mechanisms Involved

Variables Affected	Anticipated Change	Mechanism
Clinicopathologic		
Glucose		
Mild hypothermia	Increased glucose level	Increased catecholamine/cortisol production[17]
Moderate hypothermia	Increased glucose level	Decreased metabolic rate, increased insulin rate
Severe hypothermia	Decreased glucose level	Glycogen depletion, impaired gluconeogenesis
Potassium		
Mild hypothermia	Decreased potassium level	Redistribution intracellularly, catecholamine production
Severe hypothermia	Increased potassium level	Deficiency in sodium-potassium–ATPase pump, release from injured cells[18,19]
Acid–base		
Metabolic	Acidosis	Increased lactic acid production, decreased tissue perfusion, increased muscle activity
Respiratory	Acidosis	Respiratory depression
Coagulation		
Activated partial thromboplastin time/prothrombin time	Prolonged coagulation	Decreased enzymatic activity[2,20,21]
Platelets		
Mild hypothermia	Increased aggregation	Increased catecholamine release
Severe hypothermia	Decreased aggregation	Decreased enzymatic activity, decreased thromboxane A_2[20–22]
	Thrombocytopenia	Splenic/hepatic sequestration, dilutional, consumption
Metabolic		
Pancreas	Decreased endocrine/exocrine function	Decreased metabolic rate
Hepatic	Decreased metabolism	Decreased enzyme activity
Renal	Cold diuresis	Increased GFR, vasoconstriction, decreased sensitivity to antidiuretic hormone
	Acute tubular necrosis	Decreased renal blood flow, decreased GFR, blood sludging, ischemic/cold tubular damage
Cardiovascular		
Mild/moderate hypothermia	Increased HR, increased MAP	Increased catecholamine release
Severe/critical hypothermia	Decreased HR, decreased MAP, decreased cardiac output, vasodilation	Decreased catecholamine release, decreased catecholamine responsiveness, decreased potassium responsiveness, decreased baroreceptor responsiveness[14,24–28]
	Conduction disturbances,[23] arrhythmias[29,30]	Cold-induced myocardial irritation, altered myocardial microcirculation[31]
Respiratory	Decreased respiratory rate, decreased minute ventilation, decreased tidal volume,	Decreased cellular metabolism, decreased carbon dioxide, decreased stimulation of respiration[14,32]
	Hypoxia, pulmonary edema, acute respiratory distress syndrome, pneumonia	Shift in oxygen-hemoglobin dissociation curve, blood sludging, decreased alveolar ventilation
Neurologic		
Mild/severe hypothermia	Decreased mentation	Disturbed cerebral autoregulation, decreased cerebral blood flow[32,33]
Critical hypothermia	Central nervous system depression, coma	Disturbed cerebral autoregulation, decreased cerebral blood flow[32,33]
Immune system	Decreased wound healing, increased incidence of infection[34–36]	Decreased phagocytosis, decreased neutrophil migration, granulocytopenia, impaired lymphocyte response, decreased production of cytokines

GFR = glomerular filtration rate; *HR* = heart rate; *MAP* = mean arterial pressure.

and cerebral blood flow (CBF) decreases by 6% to 7% for each 1°C drop in core temperature.[17] This was confirmed in a report that studied the effects of hypothermia (33°C or 91.5°F) on regional CBF in rats in which CBF decreased in parallel with decreasing mean arterial pressure (MAP).[32] In contrast, CBF was maintained by autoregulation in the normothermic rats down to a MAP of 50 mm Hg. This is a serious consideration in posttraumatic or postsurgical patients who present hypothermic and hypotensive. Rapid rewarming is vital for preserving CBF and neurologic function.

IMMUNE SYSTEM CONSEQUENCES

The overall result of hypothermia on the immune system is impaired wound healing and a reported increased incidence of infection.[33–35] It is likely that the length and degree of hypothermia determine the degree of immune system dysfunction.

ANESTHETIC AND SURGICAL CONSEQUENCES

Primary and secondary hypothermia commonly occur during surgery or general anesthesia. Intubation prevents nasal warming of inspired air, and cold, dry air is delivered directly to the lungs. Application of antiseptic agents will increase heat loss as they evaporate, and cold table surfaces and open body cavities will cause further heat loss via conduction and radiation.

Anesthetic agents (e.g., propofol, isoflurane) widen the thermoregulatory threshold; therefore, thermogenic responses are not initiated until low temperatures are reached.[38,39] Anesthetic agents inhibit centrally mediated thermoregulatory vasoconstriction, resulting in peripheral vasodilation and redistribution of core heat to the cooler periphery.[40,41]

The significance of redistribution of blood flow during anesthesia is demonstrated in a study[42] on the use of different methods to maintain core body temperature in anesthetized dogs. Heat applied to the extremities maintained mean body temperature more effectively during anesthesia than heat applied to the thorax and abdomen. This is most likely a result of decreased redistribution of blood flow and decreased heat loss in the extremities because the core-to-periphery temperature gradient was abolished as the periphery was warmed.

Anesthesia also decreases the basal metabolic rate by 15% to 40% and inhibits muscular activity, leading to decreased heat production.[5] Hypothermia delays anesthetic recovery and increases the potential for anesthetic and surgical complications such as arrhythmia, hypotension, respiratory depression, bradycardia, coagulopathy, blood sludging, and anesthetic drug overdose.[43] The duration of surgery and anesthetic procedures should be minimized to prevent secondary hypothermia.

BOX 3
Rewarming Complications

Acute respiratory distress syndrome	Pancreatitis
	Pneumonia
Cerebral edema	Pulmonary edema
Cerebral ischemia	Reperfusion injury
Core temperature afterdrop	Rewarming shock

CONSEQUENCES FOLLOWING TRAUMA

Hypothermia is a common complication following trauma, and a high correlation exists between hypothermia and mortality in human trauma victims.[2,44,45] Hypothermia in trauma patients is often proportional to the degree of shock and severity of tissue damage. This leads to questions regarding whether hypothermia exerts an independent morbidity on trauma patients, or if hypothermia is only a marker for the severity of the injury.[46,47]

Rewarming before resuscitation can decrease an animal's ability to withstand hemorrhagic shock. A study[48] was conducted comparing two groups of rats in which hemorrhage was used to induce hypotension to a MAP of 30 mm Hg. The control group was allowed to autoregulate body temperatures during the shock period. The experimental group was warmed to temperatures between 34°C (93°F) and 36°C (97°F) throughout several hours of shock, before the onset of resuscitation. The MAP postshock was significantly higher in the control group. Survival during shock and postresuscitation was significantly higher in the control group compared with the experimental group.

Another study[49] observed the effect of hypothermia during experimentally induced hypovolemic shock in pigs. Shock was induced by removal of 50% of blood volume. The pigs were then randomized to normothermic and hypothermic (30°C or 86°F) groups through 4 hours of shock. These animals were not volume resuscitated during this study. Arterial oxygen content, potassium, and catecholamine concentrations were monitored during this period. Results showed increased arterial oxygen content and stabilization of serum potassium and serum catecholamine levels during the shock period in the hypothermic animals when compared with the normothermic group.

Both of these studies emphasize the beneficial role of hypothermia during low perfusion states. Rapid rewarming of human trauma patients in combination with fluid resuscitation, however, has a marked effect on decreasing the mean intensive care unit stay, decreasing blood loss and blood product requirements, decreasing fluid requirements, and reducing mortality.[50,51] Despite
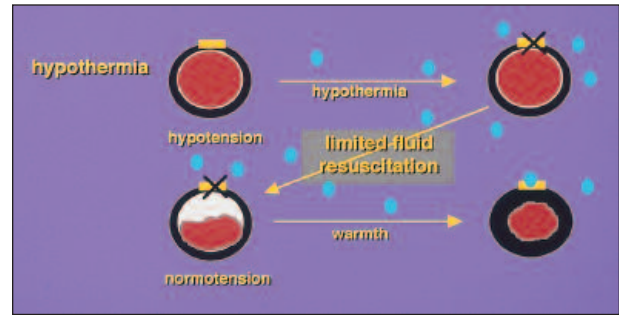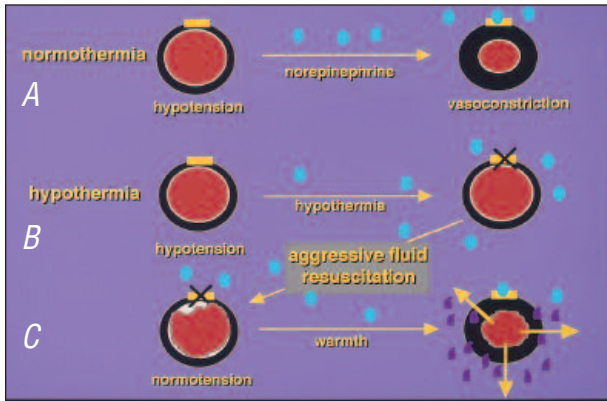
Figure 3A

Figure 3B

Figure 3—(A) Blood vessels in a cat during hypotension and hypothermia and after resuscitation. *(A)* With hypovolemia without hypothermia, the receptors on the blood vessel walls respond to norepinephrine, causing the vessels to constrict. *(B)* With moderate to severe hypothermia, a temperature-dependent decrease in receptor responsiveness to catecholamines occurs; therefore, the vessels become vasodilated. *(C)* Aggressive resuscitation to bring the blood pressure within normal limits without rewarming can lead to volume overload and vascular leakage once vascular tone returns. **(B)** Moderate fluid replacement with simultaneous active rewarming allows the vasoconstriction to occur without hydrostatic-induced vascular leakage.

the importance of aggressive rewarming of hypothermic patients, complications should be anticipated.

COMPLICATIONS OF REWARMING

A patient's core body temperature can continue to drop for a period of time after the onset of rewarming (Box 3).[52] This condition, referred to as the "afterdrop," is caused by the return of cold peripheral blood to the body core and movement of blood from the warmer core to the periphery.

A second important complication to anticipate is the development of rewarming shock. Rapid rewarming will cause a great metabolic burden on patients as well

as significant vasodilation, which can overwhelm an already compromised circulatory system.

Cats with hypovolemic shock will frequently present with a triad of clinical signs (i.e., hypothermia, hypotension, bradycardia). Each component will exacerbate the other. In our experience, aggressive fluid resuscitation to restore the blood pressure to within normal limits in hypotensive, hypothermic cats will often lead to interstitial fluid overload and pulmonary edema (Figure 3). We have found that giving conservative fluid support concurrent with aggressive rewarming is an essential part of shock resuscitation therapy in cats and will often help avoid pulmonary edema.

	TABLE 3		
	Examples of Warming Modalities, Indications for Use, and Methods of Action		
Type of Rewarming	*Methods Used*	*Goal*	*Indications*
Passive surface	External covers (e.g., blankets, foil, bubble wrap)	Prevent further peripheral heat loss while the animal generates heat	Mild hypothermia with adequate circulatory volume
Active surface	Warm water bottles, heating pads, radiant heaters, forced air blankets, heat lamps, dryers	Apply heat to the surface of the body (monitor for overheating, tissue drying, tissue burns)	Moderate, severe, or critical hypothermia (provide volume support during rewarming)
Active core	Heated, humidified, inhaled air; warm peritoneal/pleural lavage; extracorporeal rewarming; warm intravenous fluids; warm water bladder lavage; warm water enemas	Provide heat centrally to rapidly warm the core	Severe or critical hypothermia to prevent dilation of peripheral arterioles

Our recommendations are to actively rewarm the patient until a rectal temperature of 37°C (98.5°F) is reached, restoring coagulation and cardiovascular functions without overwhelming the circulatory system. This also may help prevent the afterdrop phenomenon by reducing the core-to-periphery temperature gradient. Once intravascular volume is replaced and the major consequences of hypothermia are reversed, passive surface rewarming should be sufficient to allow slow return of normothermia as the patient's cardiovascular and thermoregulatory systems recover. It is vital to monitor the patient carefully for hypotension, arrhythmia, acid–base and electrolyte abnormalities, CNS depression, and pulmonary complications during and immediately following rewarming. Any ongoing abnormalities should be treated aggressively.

THERAPY

A strong bias seems to exist among physicians and veterinarians that the deleterious effects of moderate, severe, or critical hypothermia outweigh the benefits. Therapeutic efforts are aimed at rapidly rewarming these patients during fluid resuscitation as well as reducing additional heat loss. Resuscitative efforts should not contribute to the hypothermia.

A study using pigs with induced hemorrhagic shock evaluated body temperature during fluid resuscitation versus no resuscitation at all.[53] Those resuscitated with room temperature fluids had an overall core temperature decrease, whereas those with no resuscitation showed no change in core temperature. This suggests that intravenous fluids warmed to body temperature rather than room temperature fluids should be administered during hypovolemic resuscitation to avoid further decrease in core temperature. A fluid warmer can be used, or fluids can be stored in an incubator warmed to 42°C (108°F). The fluid line can be run through a bowl of hot water, or it can be wrapped around a warm bottle to prevent the fluid from cooling as it is transferred into the patient.

It has been recommended to warm the animal by at least 1°C to 2°C per hour; however, faster rates may be necessary.[28] Moderate intravascular volume support (warm crystalloids [10 to 15 ml/kg] and colloids [e.g., 5 ml/kg hetastarch for dogs and cats; 5 ml/kg Oxyglobin® {Biopure, Cambridge, MA} for dogs]) is recommended during active rewarming in hypovolemic shock. This will support MAP and resolve most cases of hypothermia-induced hypotension, bradycardia, hypoventilation, and coagulopathy while avoiding volume overload. Electrolytes and acid–base status should be monitored and alterations addressed. The electrocardiogram, MAP, and blood gases should be monitored

closely in severely hypothermic patients.

Rewarming hypothermic animals can be accomplished by several different methods, including passive surface, active surface, and active core rewarming (Table 3). The choice of method depends on the degree of hypothermia, the status of the patient, and equipment availability. Surface rewarming should always accompany active core rewarming to reduce core-to-peripheral temperature gradients. During external heating, care must always be taken to prevent skin burns by controlling the temperature of the external heating devices or placing a barrier between the heat source and the patient.

External heating devices can be constructed out of fabric filled with uncooked dried beans or rice. These packets should then be warmed in a microwave. To avoid surface burns, the temperature of these packets should be tested before placing in contact with the animal's haircoat or skin and periodically during the rewarming period. Heat from the core of the bean- or rice-filled packet can redistribute to the surface with time, resulting in a surface temperature that is higher than when the packet was first placed in contact with the animal. Smaller animals can be placed inside heated pediatric incubators. A tent can be constructed out of blankets to trap heat and warm air from hot water bottles or warm air blowers near the animal. Warm water bottles must be removed when they reach body temperature to prevent heat loss to the bottles by conduction.

When using aggressive surface rewarming in hypothermic, hypovolemic animals, the heat source should be applied to the thorax and abdomen, and the extremities should remain cool to prevent peripheral vasodilation. In addition, warming the extremities during hypothermia can decrease neuronal feedback to the thermoregulatory center, thereby decreasing the thermoregulatory response.

Peritoneal and pleural lavage are examples of aggressive core rewarming methods. When using these techniques, no difference has been found in efficacy between warming the thoracic versus the abdominal cavity.[54] A pleural or peritoneal catheter should be placed aseptically. Isotonic crystalloid solutions should be warmed to 45°C (113°F), and six to eight exchanges may be needed to warm the core. The fluids should flow in rapidly at a volume of 10 to 50 ml/kg/exchange and then should be rapidly removed. The process must be stopped before reaching normothermic temperatures (37°C or 98.5°F) to avoid causing rebound hyperthermia.[53]

SUMMARY

Most veterinarians are conscious of the dangers of primary hypothermia and the importance of rewarming; however, secondary hypothermia and its consequences for critically ill patients are often overlooked. Without detection and immediate correction of secondary hypothermia, complications can occur that interfere with the clinician's ability to resuscitate, increasing morbidity and mortality.

In the approach to a postsurgical, posttrauma, or metabolically ill animal, decreased body temperature should be viewed as an important co-morbid factor. Attempts to maintain normothermia should be included in the resuscitation plan for critically ill animals. Early and aggressive treatment will decrease the incidence of hypothermic complications and aid in successful resuscitation. Complications secondary to rewarming should be anticipated. Intravenous volume support should be given, and clinicians should actively rewarm patients to only 37°C (98.5°F). Efforts should be made to support vital organ function. Close monitoring should be done during and following the rewarming process until the patient is stable.

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ARTICLE #1 CE TEST

The article you have read qualifies for 1.5 contact hours of Continuing Education Credit from the Auburn University College of Veterinary Medicine. *Choose the best answer* to each of the following questions; then mark your answers on the postage-paid envelope inserted in *Compendium.*

1. Secondary hypothermia occurs with
   a. systemic disease, injury, or altered heat production.

b. exposure to a cold environment.
c. a normal thermogenic response.
d. frostbite.

2. Heat loss can result from
   a. decreased core-to-periphery temperature gradient.
   b. increased blood flow through the skin.
   c. vasoconstriction of skin arterioles.
   d. increased metabolic rate.

3. The temperature regulatory system is located in the
   a. spinal cord.
   b. parietal cortex.
   c. preoptic and anterior hypothalamic nuclei.
   d. posterior hypothalamus.

4. Severe hypothermia results in
   a. vasoconstriction.
   b. CNS depression.
   c. shivering.
   d. increased metabolic rate.

5. The protective effect of hypothermia is from
   a. increased energy expenditure and oxygen consumption.
   b. destruction of infectious agents.
   c. increased blood flow to the vital organs.
   d. decreased energy expenditure.

6. Coagulopathies may occur in hypothermic patients, most likely as a result of
   a. decreased enzymatic activity.
   b. increased enzymatic activity.
   c. thrombocytosis.
   d. vasculitis.

7. The cardiovascular consequences of severe hypothermia are caused by
   a. increased catecholamine responsiveness.
   b. decreased catecholamine responsiveness.
   c. increased catecholamine release.
   d. hypothermia-induced alterations in catecholamine function.

8. Anesthesia alters thermoregulation by
   a. narrowing the thermoregulatory threshold.
   b. causing vasoconstriction.
   c. increasing cellular metabolic rate.
   d. widening the thermoregulatory threshold.

9. Aggressive rewarming should
   a. accompany moderate intravascular volume support.
   b. begin immediately with mild to moderate hypothermia.
   c. never be attempted until passive rewarming has been attempted for 3 to 6 hours.
   d. be attempted only in patients weighing over 30 kg.

10. Rewarming shock
    a. results from the cold peripheral blood reaching the blood vessels.
    b. results from cold blood entering the kidneys and decreasing urinary output.
    c. results from an increased metabolic burden.
    d. is rarely serious.