Thermal/Electrical Injuries

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2.1 Burns

Burns to the skin are described by addressing the depth and size, most often described as total body surface area (TBSA) affected. In the human literature, size and depth of a burn are prognostic indicators for survival or post-burn sequelae. The location and shape of a burn or scald may also provide evidence of intentional or unintentional injury.

The morphology of a burn varies depending on the agent of heat and the duration of contact. An open fire burning at 600 °C may produce burns of different depths depending on how close and how long an animal is exposed to it. Prolonged contact at a distance from the flame may produce pathologic changes similar to direct contact and quick exposure. Similarly, water at a sub-boiling temperature of 65 °C can produce deep burns if exposure or submersion is prolonged.

Burns to the skin have been classically described as first, second, third, or fourth degree based on gross characteristics and the depth of affected tissue. However, because heat is not evenly distributed across the skin surface, gross manifestations of heat injury may range from simple erythema to full-thickness necrosis of the skin. Full gross and microscopic descriptions of the lesions seen in a thermal injury case can help dispel any confusion that may arise with the use of classical monikers.

The gross and microscopic appearance of the different depths of burns is described by Tintinalli et al. [1] and shown in Table 2.1. Superficial burns affect only the upper epidermis. They appear hyperemic and are sensitive to the touch. The classic example of a superficial burn is sunburn, in which the skin appears pink and the animal exhibits withdrawal or avoidance when the area is touched. Superficial partial-thickness burns affect the epidermis and the dermal papilla, while sparing deeper layers. The adnexa remain intact and the skin is very painful. Blisters are the hallmark of this depth of burn and the area appears moist and red. Hot, water-based liquids often cause superficial partial-thickness burns.

Deep partial-thickness burns extend into the dermis, affecting adnexa, but spare the panniculus. These burns appear white or pale yellow. Because the nerves in a deep partial-thickness burn have been affected, the area is not painful to the touch. Similarly, blood vessels in the area are cauterized and the skin does not blanch when pressed. These lesions may be caused by exposure to hot objects, such as cigarettes and flames or hot, oil-based liquids.

Full-thickness burns extend into the panniculus adiposus and carnosus. All adnexa in the area become necrotic or are destroyed, depending on the agent of the burn. These wounds may be

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J.W. Brooks (ed.), *Veterinary Forensic Pathology, Volume 2*,
https://doi.org/10.1007/978-3-319-67175-8_2

Table 2.1 Burns of the skin may be classified by the level to which the thermal agent has an effect. Each classification is associated with clinical, gross, and histological characteristics that may overlap or differ based on the portion of the burn that is examined

	Superficial burn	Superficial partial-thickness burn	Deep partial-thickness burn	Full-thickness burn
Depth	Epithelium only	Upper dermis sparing adnexa	Deep dermis affecting some parts of adnexa	Panniculus affecting the entire depth of adnexa
Clinical presentation	Painful to the touch	Very painful to the touch	Discolored area is painless	Painless
Gross appearance	Smooth, erythematous skin that blanches with pressure	Moist, erythematous skin with blisters that blanches with pressure	Erythema with central, static area of white or yellow discoloration (zone of coagulation)	Charred, leathery skin may be black or pale
Histological appearance	Capillary and lymphatic ectasia with cutaneous edema	Separation of the epithelium from the dermis with proteinaceous fluid or cellular infiltrate within the void. Elongation of basilar cell nuclei in intact skin	Coagulative necrosis and smudging of dermal collagen. Loss of differential staining and nuclear pyknosis	Possible tissue loss or metal deposition (depending on thermal agent)
Causes	Radiant heat (sun)	Hot water scalds	Hot water, oil, or grease, steam, flame	Contact with fire, hot metal, hot oil

blackened, leathery, or pale and are painless. Common agents of full-thickness burns include high-voltage electrocution; hot objects, such as soldering irons; or prolonged contact with steam or oil-based liquids.

Heat dissipates with distance from the injurious agent, and the gross and histologic alterations in the skin reflect this temperature differential. In 1953, Jackson described three zones of tissue alteration that result from a deep partial-thickness or full-thickness burn [2]. At and directly surrounding the point of contact with the hot object or electrical current is the zone of coagulation. In this area, the tissue is necrotic and damage is irreversible. In a live animal, the skin appears blanched and the coloring is not responsive to pressure. Surrounding the zone of coagulation is the zone of stasis, which is characterized initially by vasoconstriction and ischemia. Immediately after the burn event, tissue in this zone remains viable, though subject to the effects of inflammatory mediators. Without local and systemic treatment of the burn victim, tissue in the zone of stasis may become necrotic. Between the zone of stasis and unaffected tissue is the zone of hyperemia. Vessels in this area are dilated due to local release of inflammatory mediators. Unless subject to additional insults—such as infection—tissue in the zone of hyperemia remains viable and heals with little to no scarring.

The estimation of the body area affected by burns is well established in the human literature by the "rule of 9 s." In this schema, the arms each represent 9% of a human's surface area; the legs, 18%; the head, 9%; the trunk, 36%; and the genitalia, 1% of the total body area. Because of obvious physical differences between quadrupeds and humans, this rule cannot be directly applied to animals. Additionally, variations in conformation between breeds make a single rule difficult to formulate. A rough estimation of the surface area of different body parts can be made based on a generic dog schematic (Fig. 2.1). In this diagram, the forelimbs represent 10% of the surface area; the hind limbs, 15%; the head and neck, 25%; and the trunk, 50%. Animal tails vary widely in surface area and pelage character, and a description of the effects of thermal damage should be included in the narrative on affected surface area.

The skin reacts to heat consistently, whether the injurious agent is a flame, hot liquid, heating pad, or electric current. The patterns of injury

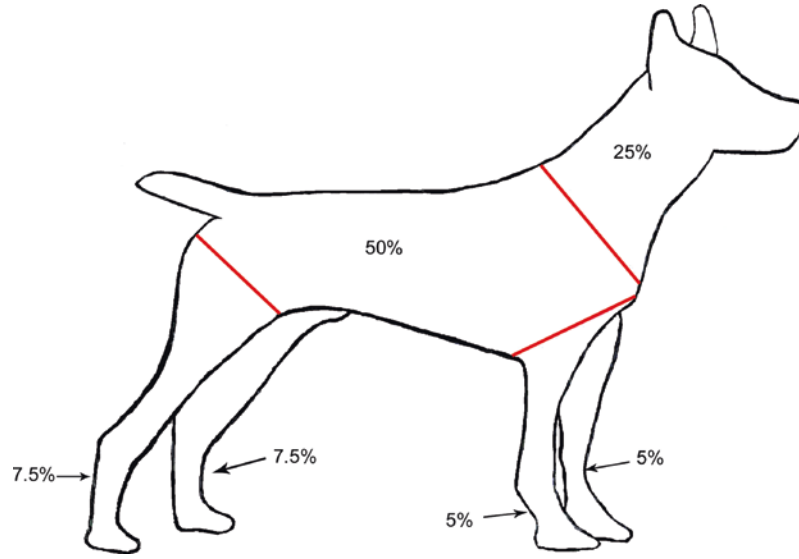

Fig. 2.1 Approximation of body surface area represented in a schematic of a generic dog. In this model, the forelimbs collectively represent 10% of the body surface area; the hind limbs, 15%; the trunk, 50%; and the head and neck, 25%. Estimation of the body surface area affected by thermal or chemical burns can inform predictions for healing and survival

vary depending on the electrothermal cause. Despite the source, protein degeneration begins to occur when the cell temperature reaches approximately 45 °C [1]. Coagulation necrosis is the norm. Additional histologic changes include intraepidermal or epidermal-dermal separation (blisters). Intraepidermal separation is much more common in electrocution events than in thermal burns [3]. The presence of concurrent intraepidermal and subepidermal separation is also more common in electrocuted skin than in flame-damaged skin. Other electrothermal changes in the skin include dermal smudging or homogenization, darkening of the epidermal cell nuclei, and elongation of the nuclei in the basal and hair follicle epidermis.

In animals, the primary barrier to exogenous heat or electricity is the hair or feathers. Often, the effects of heat exposure are limited to this barrier only. As keratin melts, it curls and discolors brown or black [4]. Under a dissecting microscope, there is an abrupt change between the normal, straight hair or feather barb and the curled and darkened outer portion of the hair or barb. Higher temperatures cause the hair or feather to char, turn to ash, and break off. It is not unusual in high-heat burns to have a focal or diffuse absence of plumage or pelage, with only follicles remaining of the external covering.

2.2 Fire

The fires to which domestic animals are exposed are most often present in an enclosed space, such as a house or a room within a house. From a legal perspective, it is important for the veterinary forensic expert to establish whether the animal was alive during the fire or dead prior to ignition of the surroundings. In some house fire incidents, it can be challenging to determine a cause of death if the animal is partially or fully consumed by the flames. However, even in cases in which the entire body surface area has been charred, internal organs often remain only mildly altered. Radiographic, gross, and histologic examination of burned remains still provides valuable information on the condition of the animal at the time of death and the cause of mortality.

Muscles that are exposed to the broad heat of a fire react by contracting. In humans, this contraction of major muscle bundles results in a "pugilistic pose" wherein the limbs assume a crouching and defensive posture: the hands close, the knees bend, and the elbows flex. Animals that are burned may have an arched back and flexed limbs, reflective of contraction of the most powerful muscle bundles.

In a fire situation, the most dangerous elements are inhaled gasses and particulates. Fire-related

deaths are more often attributed to inhalation injury than to physical burning of the skin. The products of combustion are dictated by the objects that are on fire. The smoke that is produced is inconsistent from fire incident to fire incident and may include particulates, aerosolized fluids and solids, superheated air, and gasses in varying quantities and types. In addition to these harmful substances, combustion uses up the oxygen in a confined space, resulting in a low-oxygen environment. Irritants that may be produced by the combustion of objects include ammonia, sulfur dioxide, hydrogen chloride, and phosgene. These may have direct effects on the airways, resulting in edema and inflammation. Asphyxiants displace oxygen both in the environment and physiologically, making this class of agent highly dangerous in an enclosed space situation. The most common fire-derived asphyxiants are carbon monoxide (CO) and cyanide (CN).

Smoke may originate not only from the obvious flames of a fire but also from items that are smoldering [5]. Smoldering occurs when an object is exposed to heat sufficient to cause thermal decomposition, but the relative oxygen concentration in the immediate surroundings is insufficient to support flame. This high-heat decomposition, or pyrolysis, still results in the release of particulates, gasses, and superheated air that can be detrimental to humans or other animals in the area.

Standard evaluation of fire survivors, especially those showing signs of tachypnea, tachycardia, and CNS depression, should include blood gas analysis and CO-oximetry in addition to a thorough history and physical evaluation. Chest radiographs may initially be unremarkable, but edema, inflammation, and cast formation may result in an alveolar or interstitial pattern 2–36 h after the event. Additional delayed effects of smoke inhalation include cerebral edema due to prolonged hypoxia, and stenosis and fibrosis of airways. Similar to burn victims, infection may follow a fire event resulting in bacterial pneumonia.

2.2.1 Pulmonary Irritants

Soot and other fire-related particulates are produced at the upper surface of the fire. In wildfires, these particulates are carried up and away on the wind. In an enclosed space, however, smoke fills a room from top to bottom and may be present below the surface of the flames. Soot on the mucosal surfaces of the trachea, esophagus, or stomach is a sign of vitality and respiration during a fire [6]. The absence of soot in these areas, however, does not definitively indicate that the animal was dead at the time of the fire. Elevated levels of CO and cyanide produced by a fire may induce toxicity, unconsciousness, and death before the animal is exposed to particulate matter in the air. Conversely, when soot is detected in the airways or upper GI tract, toxic or lethal levels of carboxyhemoglobin (COHb) and CN are almost always detected [7].

Grossly, soot and fire-related particulates appear as a black coating on the mucosal surface of the trachea. Swallowing during the fire results in black, smudgy material in the esophagus and stomach. In acute deaths, the material is extracellular and associated with the surface mucus layer. Particles that are smaller than 3 μm in size may reach the alveoli [5].

In a fire situation, animals may be exposed to superheated air. The temperature of this inhaled air declines quickly distal to the larynx. Thus, animals that survive exposure to a burning structure rarely have direct thermal injury caudal to the vocal cords. Damage due to inhalation of steam, however, may occur in the more distal portions of the trachea [1]. Postmortem exploration of whether an animal was alive during the thermal event should include histologic sampling of the trachea from at least three locations. Areas immediately adjacent to the larynx may exhibit loss of epithelial cilia, mucus production, and deposition of soot particles at the surface. These changes decrease and disappear in distal portions of the trachea. Edema of the lower airways may occur up to 24 h after inhaling superheated air. Animals that survive for a period of time after inhalation of hot gasses may develop acute respiratory distress syndrome, and/or pseudomembranous or purulent tracheobronchitis.

In some cases, delayed responses to the inhalation of superheated or smoke-laden air may occur. A common pathologic response to smoke

inhalation in humans is the development of tracheobronchial polyps [8]. Histologically, these polyps are composed of granulation tissue and fibrosis with little inflammation. The presence of polyps may induce a chronic, dry cough and persist for up to 6 months. In animals, asthma or chronic obstructive pulmonary disease may develop weeks or months later. Additionally, inhaled carcinogens produced by the combustion of plastics may eventually cause neoplasia.

2.2.2 Carbon Monoxide

Carbon monoxide is naturally occurring and may be present in the body at base levels around 1%. Animals that live in the environment of a smoker may have slightly elevated levels. Carbon monoxide binds to hemoglobin with approximately 200 times greater affinity than oxygen. Approximately 85% of systemic CO is bound to hemoglobin, forming carboxyhemoglobin (COHb). Circulating COHb is taken up by the tissues in the same manner as oxyhemoglobin but is incapable of providing resources for aerobic respiration.

Normal atmospheric CO concentration in an average home is generally 10 ppm or less. Increased volume fraction of atmospheric carbon monoxide may be a result of combustion of carbon-based materials in an enclosed space. Stoves that use wood, propane, or other petroleum products consume oxygen and produce CO_2. If the environment in which the stove is located is not vented properly, oxygen within the space becomes less available, and consumption of CO_2 will produce CO. OSHA limits human exposure to CO to 50 ppm, averaged over an 8-h period. The peak exposure limit for some operations is 200 ppm.

Animals breathing in an environment with an increased volume fraction of CO develop elevated levels of COHb. The COHb competes with oxygenated hemoglobin for uptake into cells where it interferes with aerobic respiration and production of ATP. The cells become hypoxic and organs throughout the body are affected [9]. Additionally, CO induces the release of NO and guanylate cyclase [1] which acts on the vasculature, causing vasodilation. The combination of vasodilation and low blood oxygen saturation results in ischemia-reperfusion injury. This damage is most commonly seen in cardiomyocytes and neurons, especially those in the basal ganglia. In addition to neuronal cell death, acute myocardial infarction and rhabdomyolysis may be seen in animals that survive the initial event.

Antemortem diagnosis of CO poisoning can be done by measuring the amount of circulating COHb. Blood gas analyzers with CO-oximetry can accurately measure the amount of COHb, as well as oxyhemoglobin and methemoglobin saturation [1, 5]. Routine blood gas analysis without CO-oximetry calculates the amount of these compounds and does not directly measure their levels. Thus, in cases of toxicity, some measurements using this method may be artificially high. The relative amount of blood COHb decreases when the animal is removed from the smoke environment. Thus, measurements of blood gasses upon admission to the clinic may not reflect the peak levels that the animal experienced. Blood gas analysis is inaccurate for animals found dead at the scene. The most accurate measurement modality for postmortem blood samples is the gas chromatography-thermal conductivity detector method (GC-TCD) described by Lewis et al. [10].

Clinical signs of CO toxicity may be seen when COHb rises above 10% [1]. Animals may become dyspneic, cyanotic, and lethargic. Due to decreased oxygen saturation of the blood, animals may be tachycardic. Acute fatality may occur when blood levels of COHb are 50–80%. There is no direct correlation between COHb levels and the presence or intensity of clinical, gross, or histologic signs, though levels above 10% are considered to be indicative of toxicity. Other historical factors, such as evidence of fire, closed windows, and/or combustion materials, can help point toward CO toxicity.

The blood and mucus membranes of animals that have died of carbon monoxide poisoning are unnaturally bright red. Aside from dilation and congestion of vessels, histologic lesions of acute

CO poisoning are not evident. In comparison to pure hypoxic events, toxicity from CO poisoning may have a delayed course. Animals that survive an initial insult may exhibit morbidity and mortality hours, days, or months after an event. Animals that initially show improvement with supportive care may reverse clinical course and develop seizures or other CNS disturbance. The pathophysiology of delayed CO poisoning is not entirely clear, but changes of selective neuronal necrosis are most commonly seen in the pallidum, substantia nigra, cerebellum, and hippocampus. Capillaries in these areas may exhibit plump, reactive endothelial cell nuclei with occasional lymphocytes in the Virchow-Robin spaces [11]. Necrosis of the deep cerebral white matter may also occur in delayed CO toxicity. Computed tomography can also be helpful in delineating symmetrical neuronal loss in the globus pallidus and other basal ganglia [12].

2.2.3 Cyanide

Cyanide also inhibits oxidative phosphorylation by binding to ferric ion cytochrome a_3, part of cytochrome oxidase in the mitochondria. Like CO, cyanide toxicity causes a decrease in ATP production and disrupts cellular aerobic respiration [1].

In a fire situation, hydrogen cyanide (HCN) is produced through the combustion of nitrogenous materials, such as wool, silk, mattresses, and upholstery [7]. The effects of inhaled HCN are immediate and may include dyspnea, seizures, bradycardia, and loss of consciousness. Clinical signs may be seen when concentrations of HCN reach 200 ppm in the air.

Gross necropsy findings are inconsistent and may include a bitter almond smell and cherry red discoloration of the skin and mucus membranes. Due to the rapid onset of death due to cyanide inhalation, histologic effects are rare. There are no delayed effects of cyanide inhalation as are reported in CO toxicity.

Animals that survive a cyanide inhalation event may have metabolic acidosis but normal blood gas and CO-oximetry results. Normal levels of COHb in the blood of animals exposed to smoke or fire and exhibiting clinical signs of hypoxia should alert the veterinarian to the possibility of cyanide inhalation. Concentrations of cyanide in deceased animals can be measured in the heart blood via modified microdiffusion and photometry. Some toxicology labs have specific packaging and sampling requirements for cyanide samples.

Because the production of HCN during a fire depends on the material being burned, there is no strong correlation between measured COHb and HCN in fire- or smoke-inhalation victims [7]. HCN is more commonly detected in closed-space fire events than in other types of fire situations.

2.3 Erythema Ab Igne

Chronic exposure to a heat source that is cooler than the burn threshold (<45 °C) may result is an erythematous and hyperpigmented condition called erythema ab igne [13]. Heat sources may include heating pads, heat lamps, space heaters, or hot water bottles. Chronic heat source exposure in animals may result in localized alopecia and a reticulated or interlacing pattern of hyperpigmentation and erythema. Microscopically, there may be keratinocyte atypia, thinning of the epidermis, single-cell keratinocyte necrosis, and adnexal atrophy [14].

2.4 Scalds

Scalds occur when the skin comes in contact with hot liquids. Scalding is associated with partial-thickness skin damage, and full-thickness burns and charring do not occur. The hair coat of mammals initially provides protection against the hot liquids. However, once the liquid reaches the skin, the hair acts as an insulator, holding the heat to the surface and potentially increasing the injury. Scalds may have an intentional or unintentional etiology [15]. In humans, intentional scalds most often occur when a child is held or suspended in a body of hot water, such as a tub or sink. In these cases, the scalds are of uniform skin depth and degree and have well-defined margins between

the affected and unaffected skin. Accidental scalds happen when hot liquids are dropped or pulled onto the body. In these cases the scald margins and depth of damage are irregular, due to movement of the body at the painful stimulus and cooling of the liquid as it pours over the skin. In veterinary forensics, the pattern of intentional scalds and scalds due to neglect may mimic accidental human scalds as hot liquids may be poured over or splashed onto an animal. Other indications of neglect, such as matted hair and ill-fitting collars, may exist in these cases to assist the forensic veterinarian in their assessment of the case.

2.5 Radiation

Nonionizing radiation has low energy and does not generally have an adverse impact on DNA or other cellular components. Common sources of nonionizing radiation include television sets, cell phones, and radios. Ionizing radiation is capable of eliciting molecular change in the cell, which may be harmful. Humans and other animals are exposed to ionizing radiation every day in the form of sunlight, natural decay of uranium and thorium in the soil, and natural radioactive minerals in food.

The sun produces ultraviolet radiation of wavelengths between 100 and 400 nm that is further subdivided into UV-A (315–400 nm), UV-B (280–315 nm), and UV-C rays (100–280 nm; [16]). The Earth's ozone layer filters out the shorter UV-C rays and most of the UV-B rays before they reach the surface [17]. The 5–10% of UV-B rays that do pass through the ozone layer to affect animals penetrate the skin superficially, with very little reaching the dermis. As the primary instigator of sunburn, these rays may be directly absorbed by DNA, resulting in molecular rearrangements, and also precipitate inflammation by inducing cytokines and vasoactive mediators. This inflammatory cascade results in the visible sunburn or superficial burn. Gross changes and the pain associated with them peak between 24 and 48 h postirradiation. Extended exposure to UV-B rays may result in keratinocyte apoptosis. The epidermal response to chronic exposure involves hyperkeratosis, upregulation of epidermal melanin production, and increased thickness of the epidermal layer as protection against further UV damage.

UV-A rays can penetrate deeply into the dermis and incite reactive oxygen species (ROS), which can then directly damage DNA. ROS may oxidize guanine, resulting in its transformation into 8-hydroxy-2′-deoxyguanosine, which pairs with adenine. This changes a G/C pair into an A/T pair. Cellular DNA repair mechanisms correct these defects in the vast majority of instances, but missed maintenance of certain mutations may result in carcinogenesis.

In contrast to nonionizing radiation, far ultraviolet light, X-rays, gamma rays, and all particle radiation from radioactive decay are regarded as ionizing radiation. Ionizing radiation most notably causes hydrolysis of water, resulting in the formation of reactive oxygen species which can directly harm DNA and other macromolecules [18]. The most common effect of ionizing radiation is carcinogenesis resulting either from direct neoplastic transformation of a cell or inactivation of tumor suppressor molecules, such as p53 and retinoblastoma. The most commonly reported neoplasm related to non-sunlight, ionizing radiation exposure is leukemia, though thyroid tumors [19], hemangiosarcoma, osteosarcoma, and pulmonary adenocarcinoma have also been documented [20].

Reactive oxygen and nitrogen species may continue to be generated in an irradiated cell, even after the ionizing event has ceased. Additionally, the excessive generation of these species can be passed to the cellular progeny through cell division and to adjacent cells through intercellular mechanisms of communication.

High doses of ionizing radiation—such as is given in radiotherapy—and chronic exposure to low-level radiation have been associated with myocardial fibrosis and pericardial adhesions in both humans and animals [21]. Narrowing of vessels in the heart and brain may lead to congestive heart failure and thrombotic or embolic central nervous system infarction. There may also be an effect on epithelial cells at the transitional zone of the lens, resulting in posterior subcapsular

cataracts and cortical cataracts. Nuclear cataracts are not associated with ionizing radiation.

Mitochondrial DNA and proteins are also affected by ionizing radiation. Resulting defects in mitochondrial function can result in aberrations of protein manufacture and transport and accelerated cellular aging.

2.6 Sources of Thermal Injury in Wildlife

Outside of a domestic situation, the dangers of electrothermal injury do not necessarily decrease. Wild animals are subject to wildfires, commercial electricity and heat sources, and lightning. The effects of industrial gas flares and the solar industry are discussed below. Electrocution is addressed later in this chapter.

2.6.1 Gas Flares

Landfill management and the production of energy from fossil fuels often involve the disposal of excess volatile gasses. These gasses are often transmitted up a tall, chimney-like stack and directly released or burned in the upper portions of the stack. Gas flare stacks are at least 30′ tall and burning may be intermittent or constant. Birds and insects may be attracted to the gas flares because of the light emitted from them. Birds may also be pursuing the attracted insects as prey items.

Birds that fly in close proximity to the gas flares may experience temperatures sufficient to singe the feathers. The pattern of singeing is different from that of burns or electrocution in that the feathers are diffusely affected. When birds fly over a gas flare, all the feathers over the ventral aspect of the body and inner aspects of the wings are exposed to heat. The outer edges of the feather barbs are affected first and will curl and darken synchronously. Charring of the skin and burning of the feather shafts are generally not seen because birds do not fly into the flame itself for a prolonged period of time. Damage to the feathers may be sufficient to impair flight, and birds may crash to the ground, sustaining additional blunt force injuries.

2.6.2 Solar Energy Equipment

There are three different types of commercial solar power-generating facilities: photovoltaic, solar power tower, and parabolic trough. Thermally, the most dangerous modality to birds is the solar power tower. These facilities utilize thousands of large mirrors to reflect the sun's rays, measured in solar flux units, onto a centrally located tower. Within the tower is a fluid that is heated by the concentrated rays and, through vaporization and cooling cycles, runs a generator. The concentration of the sun's rays around the top of the tower produces a very high solar flux or solar intensity. This potential energy turns into heat when it comes into contact with an object.

The concentrated solar energy around the tower also emits light, which attracts flying insects, including monarch butterflies. The insects, in turn, attract insectivorous birds, which attract avian aerial predators. As the insects and birds fly into the solar flux field, their bodies are heated by the concentrated rays. Low-intensity exposure results in diffuse feather singeing similar to that in gas flare exposure. In contrast to the frank heat of a gas flare, however, solar energy is absorbed differentially based on the color of the surface being affected. Sun energy is absorbed more by darker feathers or integument than by light feathers or integument. Thus, in low-level solar intensity exposure, dark bands on the feathers may be singed or otherwise affected more than lighter bands. Additionally, only the surface of feather that is exposed to the solar flux will become superheated. Areas of the vane that are shaded by other feathers appear to be less affected or unaffected by the solar flux (Fig. 2.2).

Exposure to high levels of solar flux may cause severe singeing of the feathers or immediate combustion of the whole body. Birds may be completely incinerated mid-flight leaving little to nothing for the pathologist to examine.

Fig. 2.2 Feather from a common raven (*Corvus corax*). This raven flew into and was exposed to solar flux around a solar power tower. In physical position on the wing of the bird, only a strip of the feather adjacent to the shaft was exposed to the solar flux. Because other portions of the feather were shaded by the adjacent flight feathers, only the exposed strip became singed

2.7 Microwave Burns

Microwave ovens (or "microwaves") produce microwave radiation within the electromagnetic spectrum. Units for residential use produce microwaves of around 2.45 gigahertz and wavelengths of approximately 12 cm in length. They heat food via a method known as dielectric heating. Water and other molecules present in food have asymmetrical charges, meaning that one side of the molecule is more positive than the other side. Microwaves act to move these molecules so that the charges are aligned in the same direction. The shifting polarity of the waves then causes the molecules to rotate 180° back and forth. The movement and collisions required to achieve alignment and rotation results in the production of energy in the form of heat.

The effects of microwave radiation are limited in depth due to the resistance of the object being microwaved. The production of thermal energy is lost as the microwaves travel deeper into the tissue. Also, molecules that are more electrically symmetrical (e.g., fats) or tightly packed (e.g., bone) are less able to be moved and aligned by the microwaves, making heating less efficient. The electrical asymmetry of water makes this substance an efficient target for microwave radiation.

In a microwave oven, microwaves are produced in a magnetron that is positioned focally at a top corner of the unit. The microwaves are initially generated unidirectionally but then bounce off the walls of the chamber with continued generation. Eventually, the waves scatter and bounce throughout the chamber, hitting food from many directions.

Humans are generally indirectly injured by microwaves, through scalds or burns from food that has been heated in a microwave oven. Animals, however, may be small enough to fit in the chamber of a microwave and be exposed directly to this radiation. Due to the interfaces between the skin, panniculus adiposus, and muscle, damage from microwaves is limited to the superficial portions of the body. The area of the body affected depends on the size of the animal, the duration of exposure, and the power level setting. Larger animals may prevent or limit the bouncing of microwaves off the walls of the chamber, absorbing the radiation before it contacts the opposite wall of the chamber [22]. This may result in focal or multifocal burns, rather than diffuse damage to the skin.

Microwave radiation causes coagulative necrosis of the skin and a leathery gross appearance. As with direct thermal burns, the depth of damage dictates the sensitivity of the lesions. Penetration of the waves is often deep enough to produce anesthesia in the affected tissue. Unlike thermal burns, however, the transition from normal to damaged tissue in a microwave burn is often abrupt with little tapering of gross and microscopic changes. Due to vascular thrombosis and, perhaps, immunosuppression, inflammation around the burn is delayed and mild.

Reepithelialization is often slow, and sequelae to the event, including septicemia and multiorgan failure, may mirror those of thermal burns.

2.8 Post-Burn Sequelae

Sequelae to major burns may include multiple organ failure, septicemia, insulin resistance, hyperglycemia, and fat and muscle wasting. In adult humans, these conditions are more likely to occur when over 40% of the TBSA has been burned. Serious disorders may occur when over 60% of the TBSA of children has been burned [23].

Immunosuppression in the post-burn period is largely orchestrated by interactions between macrophages and dendritic cells [24]. In the peracute post-burn period, dendritic cells may mediate the humoral immune response to the insult. Later activity results in the stimulation of macrophages throughout the system. Macrophages produce both inducible nitric oxide synthase (iNOS) and prostaglandins. Both PGE_2 and nitric oxide (NO) suppress T cell proliferation, the effects of which may be seen several days after the thermal injury. Additionally, macrophages that, as a result of thermal injury, have been stimulated into producing NO and PGE_2 are resistant to the anti-inflammatory effects of IL-10. Macrophages stimulated by non-thermal injury are normally sensitive to the effects of IL-10. This cytokine activity results in a period of immunosuppression post-burn.

The activity of dendritic cells and NO is also implicated in the breakdown of the barrier function in the gut lining after thermal injury. NO causes instability of the cell membranes of enterocytes and endothelial cells. This disruption allows enteric bacteria access to the circulation. Suppression of the immune system then allows proliferation of bacteria within the circulatory system.

Inflammatory mediators are mobilized immediately after a burn injury. mRNAs are differentially expressed in burn-damaged dermis to coordinate these mediators, with some being upregulated and some downregulated to promote healing. Inflammatory mediators involved in repair of burns include Notch-1 and Notch-2, VEGF-A, TGF-beta, and COL1A2 [25].

Severe burns result in pathophysiologic alterations that negatively impact the structure and function of mitochondria [26]. Mitochondria-rich muscle tissue acts as a reservoir for amino acids and proteins that are required during the healing phase. The combination of increased demand on muscle tissue and the decreased capacity of the energy-generating units results in muscle wasting in the post-burn period, often lasting for over 9 months in humans.

Cardiac functioning and rhythm is altered after severe burn events [27]. Beginning immediately after the burn injury and lasting for up to 72 h, cardiac function is severely depressed in what is referred to as the "ebb phase." Following this initial period, the "flow phase" is characterized by increased energy expenditure and heart rate which may last for more than a year. This process is thought to be mediated by beta-adrenergic receptors.

In studies of human burn victims, females had poorer outcome than males [28]. This increased risk of mortality was not associated with burn size and is contrary to the better outcome exhibited by human female victims of other types of trauma. Similar studies have not been explored in animals.

2.9 Electrothermal Injury

Electrothermal injury may occur when an animal completes a circuit with an object of sufficiently high current and voltage. The movement of electrical charges is dictated by the interactions between current, resistance, and potential difference (Fig. 2.3). The flow of electricity (current) between two points is measured in amperes (A). The difference in electrical potential between those two points is measured in volts (V). Material between the two points has an inherent resistance, measured in ohms, that affects the flow of electricity. Ohm's law states that the electrical current is directly proportional to the difference in electrical potential between two points and inversely proportional to the resistance between the points. Substances with low resistance, and thus amenable to current flow, include water and steel. Rubber and wood offer greater resistance and tolerate less flow of electricity. In

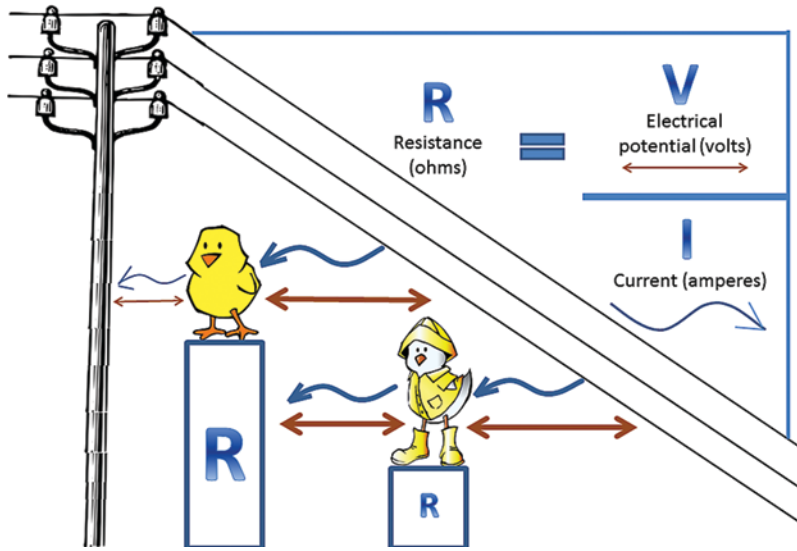

Fig. 2.3 The relationship between electrical potential, current, and resistance with regards to electrocution injury. Electricity flows between two points—in this case a ground wire and a power pole. The birds depicted have an inherent resistance that affects both the current of electricity and the electrical potential between those two points. The greater resistance provided by the dry bird on the left limits the flow of electricity through the body, whereas the wet bird in the right allows greater flow and, thus, greater injury

real-world situations, animals that are wet or in contact with moisture or metal provide relatively little resistance to the flow of electricity. Dry skin or contact with dry, nonmetallic substrate is more protective against the full flow of current.

Electrical currents may be either alternating (AC, cyclically flowing back and forth) or direct (DC, unidirectional flow). Residential electricity is AC, whereas electricity in batteries and lightning is direct. Regardless of whether the electricity alternates or flows in one direction, the path the current takes through the body follows the route of least resistance and is usually the shortest distance between the contact points.

Injuries resulting from contact with an electrothermal source include physiologic disturbance of the cardiac rhythm, physical damage to soft tissues (burns), and/or indirect, mechanical injury as a result of a fall or muscle contraction.

2.9.1 Electrocution

Events involving contact with a source of electricity are classified as either high voltage (>1000 V) or low voltage (<1000 V). Domestic animals may suffer electrical injury due to contact with exposed live wires, Tasers, and jumper cables or contact with water that is then exposed to an electrical current. Wildlife, especially birds, may be exposed to the higher voltage electricity in power lines. Additionally, proximity to very high-voltage sources may induce an electric arc without physical contact with an electrical source. The arc may contact an animal, resulting in electrocution, or pass near an animal. Air surrounding an electrical arc may reach temperatures up to 20,000 °C and cause singeing of hair or burned skin.

Exposure to high-voltage electrical conductors often results in severe electrical burns because of the heat energy caused by contact with the electricity source. The epidermal effects of low-voltage exposure may be subtle or absent, depending on the duration of exposure. Brief exposure to a low electrical potential difference may cause no visible soft tissue damage. Longer exposure—as when an animal is trapped against the electrical source—may result in mild to severe burns.

Muscles contract in response to the physiologic electrical currents passing through the nerves. Exposure to rapid, exogenous current induces tetanic or sustained contraction until the source of electricity is removed. All muscles within the path of the current are affected, but the effect of contraction of larger muscles will be stronger in relation to smaller muscle bundles. Muscular contraction may be strong and fast enough to cause fractures or joint dislocations. In heavily muscled animals exposed to high-voltage shocks even of very short duration, violent contraction may result in compartment syndrome. This may also occur with prolonged exposure to low-voltage current.

The nervous system provides the least resistance to the flow of exogenous electricity. Thus, the brain, spinal cord, and peripheral nerves are acutely susceptible to electrothermal injury. Neurons may undergo conformational change that leads to increased cellular permeability, or electroporation, which eventually results in cell death [29]. Electrocution may cause a direct effect on the eyesight through damage to the optic nerve or occipital lobe of the brain. The current also affects the intima and adventitia of blood vessels through heat damage. This results in increased vascular permeability and thrombosis. Microscopic changes that may be evident in survivors within hours of electrocution include edema and local ischemic change. Eosinophilic neuron cell bodies with pyknotic nuclei, indicating neuronal necrosis, may follow.

Circulatory damage due to electrocution may also manifest in other areas of the body. Vascular stasis, rhabdomyolysis, and ischemia may lead to transient or severe disseminated intravascular coagulation. These effects may be acute or delayed.

Following the immediate effects of the electrocution event, secondary effects of temporary paralysis or autonomic disturbances may begin within a few days. Late effects of electrocution start to manifest 5 days after the event and include brain stem dysfunction and movement disorders. These delayed effects may be due to free radicals released from damaged neurons, causing progressive demyelination [30].

In humans, the motor neuronal system is more affected by electrocution injury than the sensory system. This may be due to the relative physiologic emphasis placed on the motor system in humans. Animals often have one or more highly developed senses, depending on their evolutionary ecology. Thus, evaluation of a suspected or confirmed electrocution victim should include assessment of the potential effect of electrical current and heat on the sensory neurons.

Skin burns may occur at the site of contact in electrocutions with AC power or at entry and exit points in DC or AC electrocution. Because electrical energy is translated into heat energy at points of contact with an animal, burns due to electrocution can be described the same way as burns due to direct thermal heat injury. The effects of electrocution may be subtle, resulting in little to no discernible burns to the skin. Minor or focal damage to the hair or feathers of an electrocution victim can be highlighted by viewing the affected skin through a red filter while illuminating the area with an alternate light source (ALS) tuned to wavelengths between 530 and 560 nm [31]. Keratin that has been electrothermally damaged has photoluminescent properties that make it glow bright orange-red when viewed through a red filter under these wavelengths of visible light. Glabrous skin that has been damaged by heat or electricity may appear grossly to be merely abraded, ashy, or scratched (Fig. 2.4). These lesions, however, can be differentiated by

Fig. 2.4 Left foot of a golden eagle (*Aquila chrysaetos*). Ashy discoloration of the skin on the medial aspect of the third digit is a result of electrothermal injury

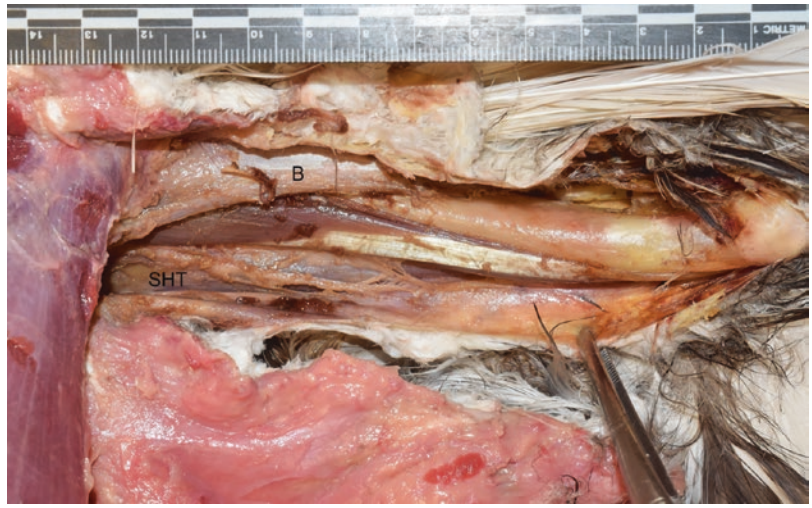

Fig. 2.5 Inner aspect of the left humerus of a bald eagle (*Haliaeetus leucocephalus*) that had singed feathers over both wings. The biceps muscle (B) and scapular head of the triceps (SHT) muscle are discolored brown due to the electrical current that passed through the soft tissues of the wings

Fig. 2.6 Right leg and tail of a common raven. The foot has been amputated and cauterized by an electrical current emanating from a power line

the use of an ALS as traumatically damaged skin does not photoluminesce.

Electrocution from contact with power within a home infrequently results in visible burns of the skin. Gross and histologic changes may be seen in prolonged contact with in-home electrical contacts. More commonly, effects of this low-voltage electrocution result in cellular disturbances that may be witnessed behaviorally, grossly, and histologically if an animal survives the initial insult. Assessment of the scene and a careful history are often needed to diagnose electrocution of an animal with little to no visibly discernible singeing or burning.

Metal particles from electrical wires may be transferred to the skin with which it has direct contact [32]. Arcing of electricity may also result in metal deposition. The metal is not generally detectable grossly or radiographically and must be explored via histochemical analysis or scanning electron microscopy of skin at the point of contact. Iron, copper, or aluminum can be detected on electrocuted skin via these methods.

Wild birds may be exposed to lower-voltage distribution power lines or higher-voltage transmission lines [4]. High-voltage electrocution is more likely to result in singeing of the plumage and burns to the skin. Rapid vaporization of water at the point of electrical contact can result in separation of the skin from the underlying muscle or bone. In these cases, the affected subcutis and musculature appear coagulated, dry, and discolored brown (Fig. 2.5). Muscle may take on the appearance of cooked meat with tan discoloration and separation of myofibers. With very high-voltage events, thermal amputation and cauterization of digits or limbs may occur (Fig. 2.6). In low-voltage electrocution of wild birds, subtle singeing of feathers and skin may occur on the inner aspects of the wings distal to the humeroulnar joints and on the feet and legs distal to the stifles.

The special sense organs, the respiratory system, and the GI tract may also suffer direct damage due to an electrocution event [1]. Because of the high water content in the eye, an electrical

current may cause the retina to detach or may cause direct damage to the cornea. Vascular damage within the eye itself may result in intraocular hemorrhage or thrombosis. Cataracts may develop weeks to years after an animal receives an electrical injury to the head or neck. Rupture of the eardrums is a common sequel to electrocution in humans but is not well documented in mammals. Hearing loss due to nerve and soft tissue damage in the ears may manifest immediately after the electrocution event or may have delayed development.

Electrical arcing may result in the production of ozone, which can act as an irritant to mucus membranes and the lungs [1]. Inhalation of ozone may result in pulmonary edema and hemorrhage. High-voltage electrical injury may cause rapid vaporization of fluids due to heat energy production. The expansion of gasses may result in tears in the intestinal wall or intra-abdominal hemorrhage.

Blunt force trauma may be secondary to electrocution injury. Electrical arcs from power lines may produce a blast force that propels an animal into objects or to the ground. Also, birds that are electrocuted on power poles may fall to the ground. Mild bruising may be evident. Hemopericardium is a common sequel to power pole electrocution and may result from direct electrical damage to the vasculature or deceleration injury upon hitting the ground. In deceleration injury, the heart base vessels are torn, resulting in hemorrhage into the pericardial sac.

Animals that are partially or fully submerged in water that becomes electrically excited may be electrocuted. In these instances, burning of the skin does not occur, but a line of demarcation between the electrified water and the air may be noted on the skin. Similar to scalds and depending on the duration of exposure to the electrified water, damage to the skin may be superficial or deep.

Histologically, there may be separation of the skin layers [3]. Intraepidermal separation of the skin is much more common in electrocution injury than in skin that has been exposed to a flame or hot objects. The presence of concurrent intraepidermal and subepidermal separation is also more common in electrocuted skin than in heat-damaged skin. Similar to heat-related burns, there may also be elongation of basal cell nuclei and smudging or coagulation necrosis of the dermal collagen.

2.9.2 Lightning Strike

Animals remaining outdoors during a thunderstorm may be hit by a lightning flash. Flashes that travel between the atmosphere and the ground are termed “lightning strikes” [33]. The duration of a strike is incredibly brief, lasting only around 300 ms. However, a flash of lightning may transmit energy up to 10^{10} J at an electromagnetic frequency of up to 10 MHz. This amount of energy is capable of boiling water instantaneously. The trunks of trees that are struck by lightning may crack or explode due to the rapid boiling of the internal sap and expansion of steam within a rigid framework. Humans and other animals, being soft bodied and more malleable, can better absorb the effects of gas expansion. However, tears in the soft tissues may result if the expanding gas overwhelms the elastic capacity of the affected tissue.

Direct lightning strike occurs when a shaft of electrically charged air passes through an animal and contacts the ground. Animals may also be struck by a sideflash, current that emanates from a nearby object that was directly struck by the flash. In contrast, a contact strike happens when an animal is in direct contact with something, such as a metal wall or fencepost that is directly hit by lightning. Additionally, lightning may travel to the ground through a conductor (e.g., tree, building) and travel through the ground to an animal standing close by (ground strike).

Lightning may travel over the surface of the body instead of using the body as a direct conduit in its path to the ground [1]. This event is termed a “flashover” and does not generally result in soft tissue burns or cardiac arrhythmias. Transformation of water on the surface of

the body into steam may result in torn clothing. Internal and external pressure differences may also result in rupture of the tympanic membranes or contusion of internal organs.

Clinically, lightning strike may result in immediate cardiac asystole and respiratory arrest. These conditions may result from depolarization of the myocardium and transient paralysis of the medullary respiratory center.

Physical evidence of the lightning strike may be subtle on both the animal and the surrounding ground. Rarely, arborizing or fernlike patterns may be seen on the ground due to the movement or charring of grass or substrate by the rapidly traveling bolt of current. These patterns are referred to as keraunographic markings when seen on the earth. Similar markings may be seen on glabrous human skin following lightning strike and are referred to as Lichtenberg figures [34]. Histologic alterations have not correlated well with this visible manifestation and they are not true burns. These patterns on the skin may disappear within 12–24 h of the initial injury. Similar findings have not been reported in animals, likely because fur or feathers obscure the change.

Cases of individual or group electrocution due to lightning strike have been documented in livestock and, rarely, in wildlife [31, 35, 36]. Herds of cattle, swine, or caribou may be exposed to inclement weather. Moisture on the pelage due to precipitation decreases the resistance of the skin to the electrical current. Additionally, the tendency of animals to congregate under trees during a storm also increases the chances of ground strike, as current may initially strike the tree and travel through the wet ground and up the legs of the surrounding animals.

Occasionally, large flocks of birds, such as snow geese, may fly through an electrostatic field in the atmosphere, triggering a lightning flash, similar to the effect of an aircraft [31]. The flash may originate at an extremity of the flock (e.g., the lead bird) and pass through all the birds within the group. Because of the immediate, debilitating muscular and cardiopulmonary effects of the lightning flash, the entire flock may fall to the ground. Gross changes of electrothermal injury in these events may be subtle and include slight discoloration and curling of feathers on the lateral aspects of the neck and leading edges of the patagia (Fig. 2.7). The eardrums may be ruptured. More common are signs of blunt force impact with the ground and deceleration injury, such as subcutaneous bruising, liver laceration, and vascular tears.

External evidence of lightning strike in mammals may include linear or patchy areas of singed hair. These areas may be highlighted by wetting the hair or using an alternate light source to look for photoluminescent, singed hairs as previously described in Section 2.9.1 (Fig. 2.8). Charring or burning of the skin due to lightning strike is uncommon. Bloat may be accelerated in lightning victims, appearing even in freshly killed animals. The bloat may be robust and explosive—care should be taken on initial incision of the carcass to avoid rapid release of gasses trapped in body cavities and luminous organs.

Lightning strike may cause muscular contraction similar to that seen in anthropogenic electrocution. Food animals are bred to have robust muscle mass and may be more susceptible to fractures or dislocations as a result of lightning strike. In one case report [36], lightning affected a small herd of pigs; one died at the scene and three presented with hind limb paralysis. In all three survivors, strong and rapid contraction of the hind limb musculature resulted in fractures of the spine at the lumbosacral junction and transection of the spinal cord. Sagittal transection of the spine in this case revealed the lesions, as external signs of blunt force trauma or burns were not present.

Histologic changes in lightning strike victims are present only when there is gross evidence of tissue damage and are similar to those in anthropogenic electrocution events. Animals that survive the initial strike and die hours or days later may have microscopically evident inflammation or necrosis of the skin, skeletal muscle, or heart.

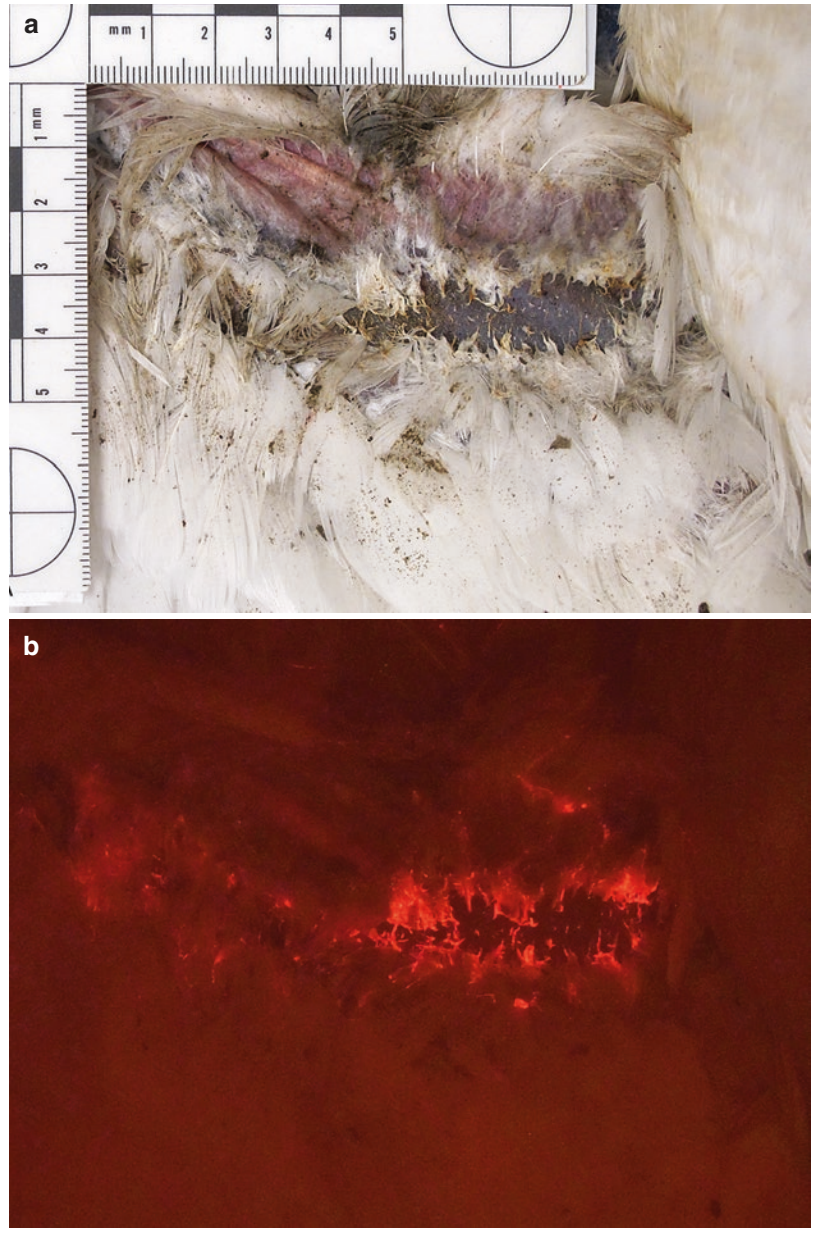

Fig. 2.7 Inner aspect of the right wing of a snow goose (*Chen caerulescens*). This was one of many geese that were found dead in a muddy field after a thunder storm and determined to have been struck by lightning. (**a**) At room light, a patch of apteria is lined by feathers that are discolored brown. (**b**) When illuminated by 560 nm wavelength light and viewed through a red filter, the feathers surrounding the area of alopecia photoluminesce bright orange

2.10 Chemical Burns

The skin and external covering (hair in mammals) provide protection against exogenous chemicals with which an animal may come into contact. The hair coat may act as a barrier to exogenous chemicals, preventing some amount of the compound from reaching the skin. However, once the chemical penetrates the pelage and contacts the skin, the hair coat may also prolong the contact between the chemical and the skin as flushing of the affected area to remove the offending agent may be less efficient.

The most harmful chemical classes are strong acids and bases. Pets may be exposed to battery or hydrochloric acid in abuse situations. These acids cause coagulation necrosis, and the depth of skin damage is dictated by the pH of the com-

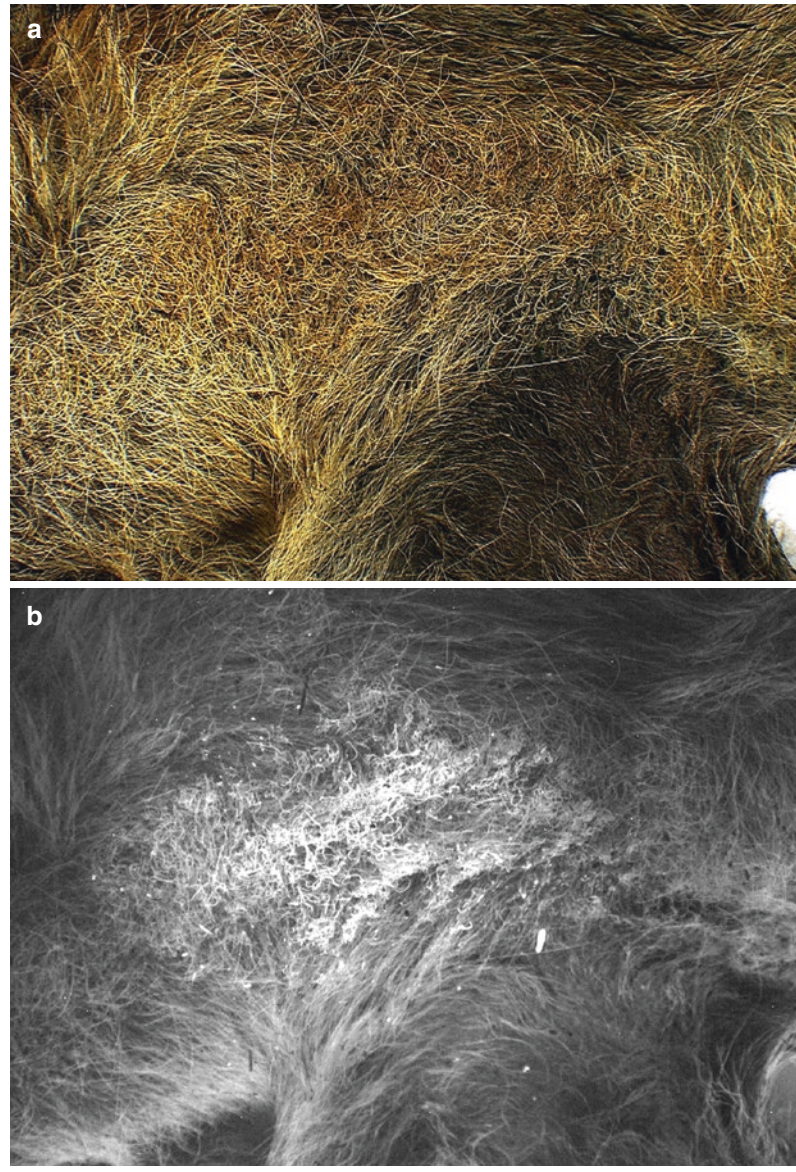

Fig. 2.8 Hair on the flank of a gray wolf (*Canis lupus*). This wolf was found dead after a thunderstorm and was determined to have been struck by lightning. (**a**) The patch of hair is curled but of normal coloration. (**b**) The same patch of hair is examined using X-ray florescence spectroscopy. The singed hair is brightly florescent

pound and the duration of direct exposure. For example, contact with sulfuric acid—with a pH of 0.3–2.1—for as little as 1 min may cause full-thickness necrosis of the skin [1]. The burn created may be blanched or brown and leathery. Due to nerve damage, the center of the lesion may be non-painful.

Acids may also volatilize, affecting the respiratory tract and mucus membranes in gas suspension. Some acidic compounds may produce a systemic effect after absorption through the skin. For example, chromic acid in contact with as little as 1% of the body surface area may cause renal and hepatic damage, coagulopathy, and CNS disturbances [37]. Specific changes include acute renal tubular and hepatocyte necrosis due to the effect of the absorbed compound's effect on epithelial structures. In severe cases, there may be intravascular hemolysis, which contributes to renal and hepatic failure.

Because of the ability of alkaline chemicals to solubilize lipids, burns from basic chemicals

tend to penetrate deeper into the skin. The result is a liquefactive necrosis—in contrast to the coagulative necrosis of acid burns—and the gross appearance is soft and gelatinous. The visible effects of alkali burns may take several hours to days to fully develop. Alkaline chemicals that animals may come into contact with include bleach, lye, and calcium oxide (lime). Lye is directly corrosive and is found in many household cleaning agents. Deep penetration of the skin by lye may allow the passage of hydroxyl ions and result in systemic effects. Calcium oxide is a commonly used gardening compound and combines with water to form calcium hydroxide, which has a pH of 11.

Differentiation between chemical burns and natural dermatopathology (e.g., atopy, lupus) rests largely in the distribution of the lesions. Many systemic hypersensitivity or autoimmune disorders manifest as symmetrical hair loss or inflammation around the eyes and nose, often sparing the mucus membranes in these areas. A chemical burn to the face would affect the skin, corneas, and mucus membranes to an equal degree. Also, the act of pouring or splashing a chemical on an animal will usually produce a pattern of injury that is asymmetrical and of varying severity throughout the wound area.

Legal Note The findings and conclusions in this chapter are those of the author and do not necessarily represent the views of the US Fish and Wildlife Service.

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