Bite Wounds in Dogs and Cats

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Introduction

Accidental wounds, including bite wounds in dogs and cats, account for a substantial part of the small animal veterinarian’s caseload. The true incidence of dog and cat bites is unknown but dog bite wounds were responsible for 10% of canine admissions to an emergency service in one study. In spite of the frequency of bite wounds, there remains little objective information to guide veterinarians’ management of these injuries. This article reviews the local mechanisms of trauma and systemic mediators involved in severe bite wound injuries, and provides a plan for stabilization, injury assessment, and definitive bite wound management.

Local Tissue Injury

In bite wounds, shearing, tensile, and compressive forces often combine to damage tissues. Shearing forces, such as those that divide tissues when a sharp incision is made with a scalpel blade, are responsible for linear lacerations seen in some bite wounds. Little energy is transmitted to the wounded tissues in a simple shearing injury, and tissue devitalization is often minimal. Wounds of this kind are much more resistant to infection than those caused by compression forces. Compressive and tensile forces are involved in the majority of dog and cat bite wounds. Tensile force on the skin is created when the teeth strike at an angle of less than 90°, and results in avulsion of the skin from underlying tissue. Avulsion of muscles can also occur in the deeper tissue layers, resulting in hernias and muscle devitalization. Compression of the skin by the teeth results in either puncture wounds, crushing injury, or both, depending on the shape of the teeth. Compression by the narrow, sharp canine teeth of cats frequently causes puncture wounds. Compression exerted by the broader, flatter premolar and molar teeth of dogs causes crushing injuries of varying severity. Fractures can occur when enough compressive force is applied to the head, limbs, or the chest wall. Crushing causes swelling, ischemia, and necrosis. In experimental crush injury, tissue blood flow and resistance to infection decreased in proportion to the greater amount of energy absorbed by the wound.

The initial appearance of many bite wounds is deceptive, because the majority of tissue damage occurs below the skin. On limbs, the teeth of the attacking animal can fracture bones. Bites located over the thorax and abdomen frequently penetrate these body cavities; in addition, the teeth crush, lacerate, and avulse muscle and subcutaneous fat and create large areas of dead space, while leaving only small, seemingly innocuous puncture wounds in the skin. This subcutaneous area is inoculated with bacteria from the skin and the attacking animal’s mouth. All open bite wounds and the vast majority of closed bite wounds should therefore be considered contaminated, meaning that they contain bacteria, devitalized tissue and sometimes foreign material. Untreated, bacteria will rapidly proliferate and invade the tissues, resulting in an infected wound.

Systemic Effects of Bite Wounds

Small, single bite wounds can have obvious systemic effects if they involve an organ or body system vital for life. For example, bite wounds penetrating the cranial vault can cause
death or coma. Bite wounds affecting the cervical spinal cord can cause respiratory muscle paralysis. Small perforations of the thoracic wall can cause a life-threatening open pneumothorax. Lacerations of major vessels can cause severe hemorrhage. Detailed management of neurologic and respiratory emergencies is covered in other chapters of this book.

Systemic effects are most frequently seen in animals with multiple, severe wounds. It is clear that severe tissue trauma, with or without the presence of infection, can initiate the systemic inflammatory response (SIRS), in which multiple inflammatory, immunological, coagulation, and fibrinolytic cascades are activated and interact. Inflammation is normally a protective physiological response that occurs only at the site of injury and is tightly controlled. Excessive activation or loss of local control of inflammation leads to a generalized inflammatory response identified as A[SIRS@. A[Sepsis@ has recently been defined as SIRS with a documented infection, and A[severe sepsis@ is defined as SIRS, a documented infection, and hemodynamic compromise. All of these clinical syndromes are commonly seen in animals with severe bite wound injuries.

Local inflammation has several important roles in normal wound healing. Initially, vasoconstriction, activation of platelets and the intrinsic and extrinsic clotting cascades facilitates blood clot formation and minimizes blood loss in the wounded area. The clot also provides a supply of cytokines (proteins acting as intercellular signals) and growth factors generated by activated platelets, the coagulation and complement pathways, and injured cells. Subsequently, vasoconstriction of all vessels in the wounded area occurs. Endothelial permeability increases in small venules, and fluid containing enzymes, antibodies, and complement proteins leaks into the wound. The clot cytokines attract circulating neutrophils and monocytes to the wound. Neutrophils and monocytes adhere the endothelium in local vessels and migrate into the wound via diapedesis.

This initial phase of inflammation provides all the elements necessary for the subsequent debridement or cleaning up of the wound. This is mediated through first neutrophils, then macrophages. Neutrophils release enzyme containing granules that degrade extracellular materials, proteins that attack bacteria, and produce cytokines that stimulate the inflammatory response and activate local fibroblasts and keratinocytes. Macrophages phagocytose bacteria and extracellular debris and release tumor necrosis factor (TNF), interleukin 1 (IL-1), transforming growth factor (TGF), and insulin like growth factor 1. These factors are vital for fibroplasia, granulation tissue formation and epithelialization during the repair phase of wound healing. However both TNF and IL-1 are also important mediators of SIRS. During the A[Repair@ phase of wound healing macrophages, fibroblasts, and blood vessels migrate into the wound. It is extremely important to note that large hematomas or remaining necrotic tissue and bacteria will prevent this migration of fibroblasts and capillaries into the wound. Not only will infected, devitalized tissue delay wound healing, but it serves as stimulus for ongoing SIRS.

A complete discussion of SIRS and sepsis is beyond the scope of this lecture, and the reader is referred to several excellent reviews for further information. However, it is important for the clinician to understand that the same cells and cytokines responsible for the normal protective inflammatory response to wounding cause the systemic changes seen in severe sepsis. This often occurs when the inciting insult is overwhelming or local regulatory control of inflammation is lost. Cytokines released from activated platelets, neutrophils, monocytes, and endothelial cells initiate generalized activation of complement, kinins, the coagulation system, and secondary messenger systems including prostaglandins, leucotrienes, nitric oxide, and
oxygen free radicals. This results in peripheral vasodilation, increased microvascular permeability and fluid loss, thrombosis of small vessels, and release of destructive enzymes from neutrophils and macrophages. These changes rapidly lead to a lack of adequate blood flow (perfusion) to tissues. Anaerobic tissue metabolism and cell death ensues. If severe enough, the profound perfusion deficits to tissues cause damage to multiple body organs. This has been termed Multiple Organ Dysfunction Syndrome (MODS) in humans, and is often fatal.

From this discussion of inflammation, wound healing, and SIRS, two basic points stand out. Firstly, wound healing will not progress past the inflammatory phase until dead or infected tissue is removed; and secondly, a wound containing devitalized or infected tissue can serve as an ongoing stimulus for SIRS/sepsis. Treatment of SIRS/sepsis is unlikely to be successful until the inciting cause of the systemic inflammation is removed. Hence, our improved understanding of the cellular and molecular mechanisms of wound healing and inflammation support the age old surgical principles of wound management: debridement, that is carefully cutting away devitalized or infected tissue; and lavage, using a balanced electrolyte solution under pressure to cleanse the wound.

**Initial Management of the Bite Wound Patient**

Initial management of an animal with bite wounds should proceed in a planned, orderly manner. The initial examination should focus on potentially life threatening problems, including injuries to the central nervous system, injuries causing inadequate ventilation, severe bleeding, and inadequate circulation. Intravenous access is mandatory in all animals. Catheters should be placed with minimal restraint if possible, to minimize struggling and further respiratory distress.

Animals with injury to the central nervous system often present stuporous or comatose. Such animals should be intubated and ventilated if either their respiration appears to be inadequate, or if the gag reflex protecting the airway from aspiration is depressed or absent. The most common injuries that severely compromise the respiratory system in an animal with bite wounds are crushing injuries to the larynx or trachea, tracheal lacerations, chest wall injury, and open or closed pneumothorax. Animals with crushing injuries to the upper airway present with severe bruising over the affected area and severe inspiratory dyspnea accompanied by stridor. Oxygen supplementation and corticosteroids can be helpful in some animals, but if the injury is severe, the veterinarian must provide an airway. Intubation should always be tried initially, as it is a procedure far more familiar to most veterinarians than tracheostomy. A small amount of a short acting general anesthetic such as propofol should be administered intravenously and the larynx clearly visualized for intubation using a laryngoscope. Severe dyspnea with no clinical signs of upper airway obstruction should raise the veterinarian’s index of suspicion for a pneumothorax. The chest should be tapped as both a therapeutic and a diagnostic procedure. Thoracic radiographs are initially contraindicated in severely dyspneic animals. Bite wounds on the thorax should be carefully examined for evidence of penetration into the pleural space. Small penetrations can be sealed with water soluble gel (K-Y jelly) and covered with a bandage whilst air is evacuated from the pleural space by butterfly needle thoracocentesis. Animals with large penetrating chest wounds generally are dyspneic enough to require immediate intubation. A chest tube is then placed through the bite wound, the area sealed with water soluble gel and a bandage, and the pleural space emptied of air. Once the animal is stabilized the thoracic wound is thoroughly surgically explored.

Bite wounds will occasionally lacerate a single large blood vessel and the animal will
present with ongoing external hemorrhage. On an extremity, this can be controlled on an emergency basis by placing a blood pressure cuff on the limb proximal to the hemorrhaging vessel and inflating it to just greater than systemic blood pressure. In most animals with bite wounds, circulatory insufficiency is caused by internal blood loss or shock associated with SIRS/sepsis. Two large gauge catheters should be placed intravenously. Blood should be obtained from the catheters for an extended data base. A warm, balanced electrolyte solution should be administered intravenously. The rate of administration is based on the veterinarian’s assessment of the degree of hemodynamic compromise. In animals with severe shock and no evidence of pulmonary contusions, the initial rate of fluid administration should be 60-90 ml/kg/hr for dogs, and 40-60 ml/kg/hr for cats. Although controlled clinical trials on animals with pulmonary contusions have not been performed, many emergency veterinary specialists feel that a more limited initial volume resuscitation (10-20ml/kg/hr) is indicated in such cases and minimizes subsequent respiratory compromise. Transfusion with fresh whole blood or stored packed red cells may be necessary if fluid resuscitation or ongoing hemorrhage lowers the packed cell volume below 25.

After eliminating immediate threats to life and beginning resuscitation, a more thorough physical examination focusing on all body systems is performed. The response to the initial resuscitation is monitored. All wounds should be covered at this time if possible to prevent further wound colonization with hospital bacteria that have substantial antibiotic resistance. Further evaluation of the wound(s) should ideally be performed under aseptic conditions.

Once the bite wound patient is stabilized, the veterinarian must focus on: Assessing the extent of the injuries; minimizing the bacterial population in the wound through aggressive wound debridement and lavage; repairing fractures and defects in the thoracic and abdominal walls; and deciding between immediate and delayed wound closure.

**Injury Assessment**

In an unpublished survey of 263 animals with bite wounds presented to The Emergency Service at the Veterinary Hospital, University of Pennsylvania in a one year period, the most common wound sites were: 1. The limbs; 2. The head and neck; 3. Regions over the thoracic and abdominal cavities; and 4. The perineal region. There are specific evaluation and management concerns with wounds in each of these locations.

**Limb wounds:** Viability of an affected limb can be clinically assessed by the temperature and color of the leg, and by cutting a toenail if systemic blood pressure is adequate. Measurement of toe web temperature and selective angiography may be necessary in some cases. The possibility of normal limb function should be determined after careful orthopedic and neurologic examination. Neurologic examination should be delayed until after resuscitation, as animals in shock are often poorly responsive to noxious stimuli.

**Head and neck wounds:** Animals with head and neck wounds should have careful examinations focusing on possible damage to the central nervous systems and the upper airway. Fractures of the skull, mandible, and cervical vertebrae can also occur. Bites to the neck can crush or perforate the larynx and trachea. This can result in severe dyspnea and subcutaneous emphysema. The esophagus is rarely perforated. Occasionally, bites to the neck can damage the caudal laryngeal nerves, causing laryngeal paralysis, dyspnea, and stridorous respiration.

**Thoracic and abdominal wounds:** The possibility of penetration into the thoracic and abdominal cavities should be assessed by surgical exploration of these wounds. Peritoneal lavage (See
chapter by Swann and Hughes) may be useful for determining abdominal penetration in wounds that are more than several hours old. In the authors' experience both radiography and blunt probing of thoracic or abdominal wounds to determine body cavity penetration are unreliable diagnostic tests, and are often misleading. The authors have seen many cases of bite wounds penetrating either the thoracic or abdominal cavity in which there was no pneumothorax/pleural effusion or pneumoperitoneum/peritoneal effusion visible radiographically.

Perineal wounds: Perineal wounds should be carefully evaluated for rectal or urethral penetration. They are often severely contaminated even if the rectum is not perforated, as bacteria contaminating the perineal skin can colonize the entire wound rapidly. They should be treated as surgical emergencies and explored as soon as the animal is stabilized.

Stabilization of the patient before anesthesia is imperative; many of these animals present suffering from poor tissue perfusion due to SIRS/sepsis. In the majority of cases, this can be corrected by aggressive therapy with intravenous crystalloid solutions, with colloid solutions such as blood, plasma or synthetic colloids given if necessary (See chapter by Drobatz). However, as discussed previously, complete stabilization may not be possible because of infection and necrotic tissue in the bite wounds. In these animals, delaying surgery in the hope of clinical improvement is usually of little benefit. The authors prefer an aggressive period of stabilization (usually 2 - 4 hours) followed by anesthesia and surgery.

**Wound Exploration, Debridement and Lavage**

Once the animal is anesthetized, a sterile, water soluble gel is placed in the wounds to prevent any further contamination during preparation. An extremely large area is clipped around the wounds and prepared for aseptic surgery. The area around the wound is scrubbed with either chlorhexadine or iodine in a detergent. The use of surgical scrub solutions in the wound itself is controversial. It is clear that bacteriocidal concentrations of both chlorhexadine diacetate and povidone-iodine are lethal to fibroblasts in vitro. Hence, the benefits of removing dirt and debris and killing bacteria inoculated into the wound must be weighed against the risk of damaging healthy tissue and impairing wound healing. Scrubbing of the wound itself should probably be limited to wounds with gross contamination. All of the scrub solution should be flushed immediately after scrubbing with balanced electrolyte solution.

Debridement of all necrotic and infected tissue is vital for successful wound management. Any remaining necrotic fat or muscle enhances bacterial growth. The lowered oxygen tension within devitalized tissue impairs bacterial killing by leucocytes. Debridement begins superficially and progresses deeper into the wound. Tissue at each layer of the wound is carefully assessed for viability before resection. All fat of questionable viability is removed. Muscle viability can be judged by bleeding at surgery. Viable muscle also will twitch when gently pinched. Debridement should not be conservative because of concerns about postoperative loss of function, as this is rarely a problem even after extensive muscle removal. Debridement is best performed with sharp dissection. The use of electrocautery should be kept to a minimum. Vital structures such as nerves and tendons should be preserved wherever possible. In some cases, the surgeon must recognize that preservation of a limb is not possible and be prepared to perform an amputation. A thorough exploration of the thoracic or abdominal cavity is required during debridement of penetrating thoracic or abdominal bite wounds. The lungs, liver, spleen, digestive, and urogenital tracts should be carefully examined for perforating or crushing injuries.
During exploration, wounds are copiously lavaged. The effectiveness of lavage is proportional to the volume of solution used. However, volume lavage alone is ineffective; the lavage fluid should be delivered under pressure. When subjected to lavage, particles with small surface areas such as bacteria experience less force than particles with larger surface areas, such as blood clots. Consequently, it takes more lavage pressure to remove bacteria from wounds than to remove larger clots, tissue debris, and foreign material. High pressure lavage, using a 35-ml syringe and a 19 gauge needle, successfully cleanses a wound of particulate matter, soil, and bacteria. Higher lavage pressures may be deleterious by forcing bacteria deeper into a wound and opening deeper tissue planes. Experimentally, Lactated Ringer’s solution or phosphate buffered saline were less damaging to embryonic fibroblasts in vitro and are preferred for lavage over either tap water or normal saline.

**Fracture, Body Wall Repair**

Fractures should be explored and small avascular fragments removed. Larger fragments may be useful for reconstruction. Opinion is divided over immediate repair. Depending on the fracture and the extent of surrounding soft tissue injuries, some surgeons may choose to repair the fracture at the initial surgery. Others may immobilize the limb in a cast, splint, or padded bandage for 24 to 48 hours with the objective of having healthy soft tissues surrounding the fracture at the time of definitive repair. Rigid immobilization is vital for bone healing in potentially infected fractures. Multidimensional external fixateurs or bone plates provide the necessary rigid fixation for definitive repair. A large autogenous cancellous bone graft should be placed in and around the fracture after repair and lavage.

Defects created in the thoracic and abdominal wall by debridement of necrotic tissue require reconstruction. Healthy tissue with an adequate blood supply is used for reconstruction if it is available. Some defects in the thoracic wall can be managed by advancing the diaphragm. Muscle flaps available for thoracic wall reconstruction include the latisimus dorsi, external abdominal oblique, and the rectus abdominis muscle. In some wounds, there is insufficient muscle for reconstruction of the chest wall. These can be managed by stabilizing the ribs in the affected area by passing heavy gauge sutures around pairs of adjacent ribs. A chest tube is placed and skin is closed over the defect. This approach was successful in treating 9 of 11 dogs with severe chest wall bite injuries. Alternatively, damaged ribs can be resected and the thoracic wall defect repaired by placing polypropylene mesh into the defect and suturing it intrapleurally to the remaining muscles surrounding the defect. The mesh is then covered by an omental pedicle flap, mobilized through a separate flank incision, and then covered with skin. The cranial sartorius muscle flap can be used to reconstruct caudal abdominal wall defects. Although not reported in the literature, a polypropylene mesh/omentum technique similar to that used for thoracic defects can be used for large abdominal wall defect repair.

**Wound Repair**

The aim of debridement and lavage is to convert a contaminated wound containing devitalized tissue into one clean enough to be sutured closed. The previously held concept of a golden period, that is a time from wounding to surgery within which surgical treatment allows all wounds to be closed (6-12 hours), has largely been abandoned. Although this 6 to 12 hour period after wounding is considered to be the time necessary for bacterial multiplication and invasion of the wound, the situation in many wounds, especially bite wounds, cannot be
simplified to a judgement based on time alone. The surgeon must judge a wound not only on the
time from wounding, but the amount of local tissue trauma that might compromise the animal’s
defenses, the degree of contamination, the type and number of bacteria inoculated into the
wound, and the adequacy of the wound’s blood supply before deciding on open or closed wound
management. Closure should be delayed if any doubt exists concerning the viability of tissue or
amount of infection remaining in the wound.

Primary closure in bite wounds requires strict adherence to basic surgical principles. Only healthy tissue should be present after debridement and lavage. Hemostasis must be excellent, as blood clots provide a superior medium for bacterial growth. Tissue layers should be gently apposed to minimize dead space, using as few fine, absorbable sutures as possible, as suture material decreases the number of bacteria required for wound infection. Ideally, the wound should be tension free. One or more drains may be required for dead space management. They may either be gravity dependent, or closed suction drains. They should exit through separate stab incisions in the sterile field, and be covered by a sterile dressing postoperatively.

In many bite wounds, delayed primary closure is the best alternative for wound management. The wound is left open after the initial debridement, and covered with a sterile, permeable dressing, such as Vaseline gauze. This is surrounded by a thick absorbent layer of padding, a conforming bandage, and an elastic, adhesive bandage layer. Delayed primary closure allows for ongoing assessment of wound healing. The wound is examined during the daily bandage change, and when considered free of contamination, a second surgical procedure is performed to close it, usually 3 to 5 days after the initial surgery. In some areas such as the distal limbs, extensive skin loss necessitates the use of a skin graft or flap for definitive repair. A healthy bed of granulation tissue should be allowed to develop before a free skin graft is performed.

**Antibiotics in Animals With Bite Wounds**

The use of antibiotics in dog and cat bite wounds is poorly understood. There are few objective studies documenting the bacteria contaminating bite wounds in dogs and cats, and no studies documenting either risk factors for bite wound infection, indications for antibiotic treatment in bite wounds, or the bacteria causing true wound infections.

In human bite wounds, the use of antibiotics to prevent infection is controversial. Some authors favor antibiotic use in all cases, while others recommend antibiotics for wounds known to be at high risk of infection. There is one meta-analysis of randomized trials of antibiotics to prevent infection in humans with dog bite wounds. This study found the risk of infection in the control (non-antibiotic treated population) was 16%; the relative risk for infection in the patients given oral antibiotics was 0.56 (95% confidence interval, 0.38 to 0.82). In essence, this data implies that if 100 human patients with bite wounds were given oral antibiotics, 84 would not become infected regardless of treatment, 9 will become infected in spite of the treatment, and in 7 the antibiotics will prevent infection. The author of the study suggests that in humans, the majority of bite wounds are minor and may not require antibiotic treatment, and that prophylactic antibiotics should be limited to patients with wounds that are at high risk for infection. Risk factors for infection in humans include full thickness puncture, hand or lower extremity wounds, wounds requiring surgical debridement or involving joints, tendons, ligaments, or bones, patients with compromised immune function, and patients with prosthetic implants. By contrast, no such risk factor analysis for infection exists for bite wounds in dogs.
and cats. Previously published information and the authors' clinical experience indicates that there is a higher incidence of severe or multiple bite wounds in dogs and cats compared to humans.

In a study of 37 dog bite wounds at VHUP, S. intermedius was the most common bacteria cultured. Enterococcus was cultured more frequently than E. coli (11 vs 10 isolates in 37 cases), and a wide range of other aerobic and anaerobic bacteria were also cultured. Antibiotic sensitivities were variable, and neither the bacterial isolates nor the likely antibiotic sensitivity could be predicted with any accuracy prior to the results of culture and sensitivity testing. There was no one antibiotic that could be relied upon to kill all bacteria in all wounds. In a large study of infected human dog and cat bites a complex mix of microbes were cultured. Significantly more organisms were isolated from specimens sent to a reference laboratory than those sent to local microbiological laboratories. This implies that more fastidious culture and isolation techniques could also yield more bacteria from veterinary patients. Mixed aerobic and anaerobic populations were present in 56% of wounds; aerobes alone were isolated from 36% and anaerobes alone were isolated from 1%. Pasteurella species were the most common bacteria isolated from both dog and cat bites.

From the previous discussion, it is apparent that insufficient experimental and clinical evidence exists to allow scientific recommendations on the antibiotic treatment of bite wounds in dog and cats. Antibiotic treatment is justified in clinically infected wounds and probably warranted in fresh wounds that have full thickness punctures or lacerations with surrounding crush injury, cellulitis or avulsion, and in animals with decreased immune resistance due to diabetes mellitus, hyperadrenocorticism or sepsis. Antibiotics should not be used as a substitute for appropriate wound management. In most instances, antibiotics are not truly prophylactic, but administered to treat a possible developing or subclinical infection. Given the uncertain flora and antimicrobial sensitivity in individual animals, cultures at the time of surgical debridement may be useful. However, intraoperative cultures do not always predict bacteria likely to cause infections in other clinical situations, and so wounds with suspected postoperative infections should be re-cultured. In humans, the majority of dog and cat bite wound isolates were sensitive to a beta lactam antibiotic and a beta lactamase inhibitor. Over 50% of bacteria cultured from bite wounds in dogs had similar sensitivities. In animals requiring parenteral medication, either a penicillin combined with an aminoglycocide or a second generation cephalosporin combined with a fluoroquinolone will provide broad spectrum coverage. However, the latter combinations will not kill the majority of Enterococcus species, which are often sensitive only to Ampicillin, Clavamox, Timentin, or Vancomycin.

**Postoperative Management**

Postoperatively, the bite wound patient is monitored carefully. Respiratory function should be assessed using clinical parameters (respiratory rate and effort) and blood gas measurement or pulse oximetry if the chest was damaged by the bite. Cardiovascular status and perfusion are evaluated frequently. These animals are at risk for sepsis in spite of early, aggressive medical management and wound debridement. Tissues which appeared healthy on initial inspection can progress to necrosis in spite of appropriate debridement and antibiotic therapy. Any deterioration in the animal's condition should raise suspicion of ongoing tissue necrosis in the wound. The wound(s) are inspected, and if there is any doubt about tissue viability, re-explored. The wound is again aggressively lavaged and debrided to healthy,
bleeding tissue.

**Infectious Diseases**
Bite wounds can transmit several viral diseases including rabies in both dogs and cats, and feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) in cats. The American Veterinary Medical Association approved guidelines for rabies control recommend that unvaccinated animals bitten by a mammal or bat that is not available for testing be euthanatized immediately. If the owner is not prepared to euthanatize the animal, it should be isolated for 6 months and vaccinated 1 month prior to release. Animals that are vaccinated should be revaccinated immediately, kept under the owner=s control, and observed carefully for 45 days. The chance of infection with either FeLV or FIV from a bite from an infected cat is difficult to predict. Serologic testing of bitten cats should be performed six months after the bite wound was sustained.

**Selected Reading**
17. Parent C. Unpublished bite wound survey, University of Pennsylvania School of Veterinary Medicine, 1993.
25. Singleton AO, Julian J. An experimental evaluation of methods used to prevent infection in wounds which have been contaminated with feces. Ann Surg 151:912, 1960