Local Tissue Injury

In bite wounds, shearing, tensile, and compressive forces often combine to damage tissues. Little energy is transmitted to the wounded tissues in a simple shearing injury, and tissue devitalization is often minimal. Tensile forces result 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 exerted by the broader, flatter premolar and molar teeth of dogs causes crushing injuries of varying severity. 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. 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. 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

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 “SIRS”. “Sepsis” has recently been defined as SIRS with a documented infection, and “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.

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. If severe enough, the profound perfusion deficits in SIRS/Sepsis causes damage to multiple organs. This has been termed “Multiple Organ Dysfunction Syndrome” (MODS) in humans, and is often fatal. It is important to realize that: i. Wound healing will not progress past the “Inflammatory” phase until dead or infected tissue is removed; and ii. 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.

The initial examination of the bite wound patient 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. Crushing injuries to the larynx or trachea, tracheal lacerations, chest wall injury, and open or closed pneumothorax can all severely compromise respiration. 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. 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.

The most common bite wound sites in dogs and cats are: 1. The limbs; 2. The head and neck; 3. Regions over the thoracic and abdominal cavities; and 4. The perineal region.

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. 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. 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 may be useful for determining abdominal penetration in wounds that are more than several hours old. 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.

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. 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. 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.

The aim of debridement and lavage is to convert a contaminated wound containing devitalized tissue into one clean enough to be sutured closed. 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.

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
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.

In a study of 37 dog bite wounds, 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 of humans.

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 avulsion, and in animals with decreased immune resistance due to diabetes mellitus, hyperadrenocorticism or sepsis. 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 b lactam antibiotic and a b 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 fluroquinolone 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 Vancomyacin.

References
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