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Microbiology and management of human and animal bite wound infections

Itzhak Brook, MD, MSc

Department of Pediatrics, Georgetown University School of Medicine, 4431 Albemarle Street, NW, Washington, DC, USA

Human and animal bites and other orally contaminated wounds are relatively common. According to the US Public Health Service, more than 1 million animal bites that require medical attention occur in the United States each year, and these bites account for approximately 1% of emergency room visits [1]. The microbiology of bite wounds generally is polymicrobial, reflecting the aerobic and anaerobic microbiology of the oral flora of the biter and the skin of the victim, as well as the environment. Clinical microbiology laboratories often do not detect these organisms, however. Bite wounds include scratches, punctures, lacerations, and evulsions. Although these wounds may look innocuous initially, they frequently lead to serious infection with a potential for serious complications [2,3].

Dog bites are an extremely common problem in the United States. Dog bites account for 80% to 90% of all animal bites requiring medical care [4] and almost 1% of emergency department visits. Although half of all bites are trivial, at least 10% require suturing and follow-up visits, and 1% to 2% of all bite wounds require hospitalization [5]. Most dog bites are in the predominant extremity. Children are especially prone to animal bites, especially of the face. Of 12,777 mammalian bites reported from 1990 through 1992 [6], 25% occurred in children less than 6 years of age, and 34% were in children 6 to 17 years old. Women are more often bitten by cats, and young men are commonly bitten by dogs.

Monkey and simian bites are becoming more common, occur more frequently in men, and often affect the upper extremity, especially the hand. These bites are often the most severe type of animal bite wounds. More than 130 such bite cases are reported [7], and their complications include cellulitis, osteomyelitis, tenosynovitis, and flexion contractures. Other types of animal bites include those of horses, pigs, and aquatic animals.

E-mail address: ib6@georgetown.edu

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Microbiology

A variety of organisms that generally result from the aerobic and anaerobic microbial flora of the oral cavity of the biting animal, rather than the victim's own skin flora, can be recovered from bite wounds. Most infections are polymicrobial. The role of anaerobes in bite wound infections has been increasingly appreciated [8–12]. Anaerobes have been isolated from more than two thirds of human and animal bite wound infections, especially those associated with abscess formation [8–12]. *Streptococcus pyogenes* is generally found in human bites; *Pasteurella multocida* in animal bites [13,14]; *Eikenella corrodens* in both animal and human bites (mostly with the latter); *Capnocytophaga canimorsus* (formerly called Centers for Disease Control (CDC) group DF-2) [15], *Capnocytophaga cynodegmi*, *Neisseria weaveri* (formerly M-5) [16,17], *Weeksella zoohelcum* (formerly IIj) [18], *Neisseria canis* [19], *Staphylococcus intermedius* [20], NO-1 [21], and EO-2 [22] in dog bites; a *Flavobacterium* group (IIb-like organism) in infected pig bites [23]; and *Actinobacillus* species in horse and sheep bite wounds [24]. *Vibrio* species, *Plesiomonas shigeloides*, *Aeromonas hydrophila*, and *Pseudomonas* species have caused infections in bites occurring in marine settings [25–27]. Tularemia can be transmitted from cats [27], herpes B virus from monkeys, rat bite fever and sodoku from rats, hepatitis B virus from humans, and leptospirosis from dogs and rodents.

Human bites

Earlier studies noted alpha-hemolytic streptococci and *Staphylococcus aureus* to be the most common organisms isolated [28,29]. The presence of anaerobic spirochetes and fusiform bacilli was noted to correlate with a less-favorable prognosis. Most studies that did not use anaerobic methodology have reported *S. aureus* to be the most frequent organism isolated, recovered from 62% to 80% of wounds and the one most often correlated with severity of and complications from human bite infection [30]. Penicillin-resistant gram-negative rods alone or in mixed culture have been reported in 24% to 43% of bite wounds cultured [30,31].

Two studies that used anaerobic methodologies were first to report the recovery of anaerobic bacteria in human bites in adults [32] and children [33]. Goldstein et al [32] recovered anaerobic bacteria in 18 of 34 human bite wounds and clenched-fist injuries. A total of 42 strains of anaerobic bacteria was isolated. *Bacteroides* species were the most frequent isolates (21 isolates), none of which were *Bacteroides fragilis*. The predominant anaerobic gram-negative bacilli recovered were pigmented *Prevotella* and *Porphynomonas* spp (11 isolates). There were four strains of *Fusobacterium nucleatum* and 10 anaerobic gram-positive cocci. The predominant aerobes recovered were *S. aureus* (10 strains), group A beta-hemolytic streptococci (9 strains), and *Eikenella corrodens* (4 strains).

Brook [33] evaluated 18 children with human bites (Table 1). Aerobic bacteria only were isolated in two specimens (11%), anaerobic bacteria only in one (6%), and mixed aerobic and anaerobic flora in 15 (83%). A total of 97 isolates (range, 1–8 per specimen) was recovered from human

Table 1
Aerobic facultative and anaerobic bacteria isolated from 21 children with animal bite wounds and 18 children with human bite wounds

	Animal bite	Human bite
Aerobic and facultative isolates		
Streptococcus sp		
Alpha-hemolytic	2	5
Group A beta-hemolytic		3
Non–group A beta-hemolytic	3	2
Gamma-hemolytic	2	6
Enterococcus sp		2
Staphylococcus aureus	7	9
Staphylococcus epidermidis	3	2
Neisseria sp	2	2
Corynebacterium sp	3	5
Pasteurella multocida	6	
Eikenella corrodens	1	3
Klebsiella pneumoniae		1
Pseudomonas fluorescens group	3	
Acinetobacter calcoaceticus		1
M-5	3	
Haemophilus influenzae		1
Haemophilus parainfluenzae		2
Haemophilus aphrophilus	2	
Total	37	44
Anaerobic isolates		
Peptostreptococcus sp	7	11
Peptostreptococcus magnus	2	3
Peptostreptococcus asaccharolyticus		4
Veillonella parvula	3	2
Veillonella sp	1	1
Bifidobacterium sp		2
Eubacterium sp	1	2
Fusobacterium sp	1	3
Fusobacterium nucleatum	3	6
Fusobacterium necrophorum		1
Bacteroides sp	1	6
Prevotella melaninogenicus	1	4
Prevotella intermedius	2	2
Prevotella oralis		3
Bacteroides ovatus		1
Bacteroides ureolyticus		2
Subtotal	22	53
Total	**59**	**97**

Data from Brook I. Microbiology of human and animal bite wounds in children. J Pediatr Infect Dis 1987;6:29.

bites (Table 1) (5.4 per specimen): 44 aerobes (2.4 per specimen) and 53 anaerobes (3.0 per specimen). Beta-lactamase activity was noted in 13 isolates that were recovered in 11 patients. These isolates were all nine isolates of *S. aureus*, two of the six pigmented *Prevotella* and *Porphynomonas* sp, one of three *Prevotella oralis*, and the single isolate of *Bacteroides ovatus*. The results of these studies show normal oral flora, rather than skin flora, to be the source of most bacteria isolated from human bite wound cultures.

Animal bites

Many studies of animal bite wounds and infections have focused on the isolation of *Pasteurella multocida* [34,35] and disregarded the role of anaerobes. Studies of the gingival canine flora, with an effort to correlate it with bite wound bacteriology [36–39], also have been reported. These studies illustrated that the spectrum of organisms found in these wounds is much greater and includes polymicrobial aerobic and anaerobic flora (Box 1). Almost any oral flora isolate is a potential pathogen and therefore the bacteriology of these bite wounds varies and needs further study.

Holst et al [14] investigated the distribution of 159 *P. multocida* isolates from bite wounds. *P. multocida* accounted for 60% of the isolates and was

Box 1. Common bacterial isolates from dog and cat bite wounds

Pasteurella multocida subsp. multocida
Pasteurella multocida subsp. septica
Pasteurella dagmatis
Pasteurella canis
Pasteurella stomatis
Pasteurella haemolytica
Capnocytophaga canimorsus
Capnocytophaga cynodegmi
Alpha-hemolytic streptococci
Beta-hemolytic streptococci
Enterococcus sp
Staphylococcus aureus
Staphylococcus intermedius
Staphylococcus epidermidis
Haemophilus felis
Haemophilus aphrophilus
Corynebacterium sp
Micrococcus luteus
Moraxella sp
Neisseria canis
Neisseria weaveri
Acinetobacter calcoaceticus

Actinobacillus actinomycetemcomitans
Eikenella corrodens
Weeksella zoohelcum
EB-4
Bacteroides tectum
Peptostreptococcus
Fusobacterium nucleatum
Fusobacterium russii
Prevotella melaninogenica
Prevotella heparinolytica
Prevotella intermedia
Prevotella brevia
Prevotella buccae
Prevotella denticola
Prevotella zoogleoformans
Porphyromonas cangingivalis
Porphyromonas canoris
Porphyromonas circumdentaria
Porphyromonas salivosa
Porphyromonas sulci
Porphyromonas asaccharolytica
Veillonella parvula
Leptotrichia buccalis

recovered in all cases of bacteremia. *Pasteurella septica* accounted for 13% of isolates, was more commonly isolated in cat than in dog bites, and caused more CNS complications. *P. canis* (biotype 1) was recovered from 18% of wounds, in all cases in dog bites. Isolates less frequently recovered include *Pasteurella stomatis*, *Pasteurella dagmatis*, *Pasteurella gallinarum*, *Pasteurella haemolytica*, and *Pasteurella pneumotropica*, and they also have been associated with endocarditis and bacteremia.

Anaerobic bacteria play a prominent role in bite wound infections [8–12,40]. They can be isolated from approximately 75% of dog and cat bite wound infections, mostly from those with abscess formation [8–12,40] (Table 1). Anaerobic gram-negative bacilli are the predominant anaerobic isolates (they were called *Bacteroides* in earlier studies). The most frequently isolated strains include *Porphyromonas salivosa*, *Porphyromonas gingivalis*, and *Porphyromonas canoris*. Other frequently encountered isolates include *Porphyromonas cangingivalis*, *Porphyromonas cansulci*, *Porphyromonas circumdentaria*, *Porphyromonas levii*–like strains, and some unidentified species [11]. Saccharolytic *Bacteroides* and *Prevotella* species also are often recovered from dog and cat bite wounds. These species include *Bacteroides tectum*, *Prevotella heparinolytica*, *Prevotella zoogleoformans*, *Prevotella buccae*, and *Prevotella oris* [10].

Using optimal aerobic and anaerobic cultural methods, Goldstein et al [38] studied 27 dog bite wounds and recovered 109 organisms, of which 87 were aerobes and 22 were anaerobes. All positive culture specimens yielded multiple organisms, most of which were potential pathogens. *P. multocida* was isolated from 7 of 27 wounds (30%), and the most common aerobic isolates were the alpha-hemolytic streptococci (12 strains) and *S. aureus* (five strains). Anaerobic pathogens were present in 41% of wounds. These pathogens included gram-negative bacilli (five strains, all but one belonging to the pigmented *Prevotella* and *Porphynomonas* sp) and *Fusobacterium* spp (five strains). These authors and others [17] studied other animal bites (cats, squirrels, other rodents, and rattlesnakes) and had similar data.

Brook [33] studied 21 children who sustained animal bites, 17 from dogs and four from cats. Aerobic bacteria only were recovered in five children (24%), anaerobic bacteria only in two children (10%), and mixed aerobic and anaerobic isolates in 14 children (66%). A total of 59 bacterial isolates (range, 1–6 per specimen) was recovered (2.8 per specimen) (Table 1): 37 aerobes (1.8 per specimen) and 22 anaerobes (1.0 per specimen). Beta-lactamase activity was noted in five isolates. These isolates were five of the seven isolates of *S. aureus*.

Talan et al [39] studied infected wounds of 50 patients with dog bites and 57 patients with cat bites. They recovered a median of five bacterial isolates per culture (range, 0–16). Aerobes and anaerobes were isolated from 56% of the wounds, aerobes alone from 36%, and anaerobes alone from 1%. *Pasteurella* sp were the most frequent isolates from both dog bites (50%) and cat bites (75%). *Pasteurella canis* was the most common isolate of dog bites, and *P. multocida* subspecies *multocida* and *septica* were the most common isolates of cat bites. Other common aerobes included streptococci, staphylococci, moraxella, and neisseria. Common anaerobes included fusobacterium, bacteroides, porphyromonas, and prevotella. Isolates not previously identified as human pathogens included *Reimerella anatipestifer* from two cat bites and *Bacteroides tectum*, *Prevotella heparinolytica*, and several porphyromonas species from dog and cat bites. *Erysipelothrix rhusiopathiae* was isolated from two cat bites.

The bacteriology of monkey and simian bite wounds is similar to human bites, in which aerobic and anaerobic pathogens are the infecting agents. These pathogens include *Streptococcus*, *Enterococcus*, and *Staphylococcus* spp, *E. corrodens*, *Neisseria* spp, *Enterobacteriaceae*, and anaerobes such as gram-negative bacilli and *Fusobacterium* spp.

A potential pathogen with some monkey bites is B virus (also known as *Herpesvirus simiae* or Cercopithecine herpes virus 1) [41,42]. Such infection may cause fatal encephalomyelitis in bitten individuals. It is enzootic in North African and Asian monkeys and includes the macaque and rhesus monkeys. The infection can be transmitted in captivity to other monkey species.

The organisms recovered from horse bite wounds include *S. aureus, Streptococcus* spp, *Neisseria* spp, *Escherichia coli*, *Actinobacillus lignieresii*, *Pasteurella* species, *Bacteroides ureolyticus*, *B. fragilis*, other anaerobic gram-negative bacilli, *Prevotella melaninogenica*, and *Prevotella heparinolytica* [24,43,44]. Sheep bites are associated with *Actinobacillus* species [24].

The organisms recovered from pig bite infections are *Staphylococcus* spp, *Streptococcus* spp (including *Streptococcus sanguis*, *Streptococcus suis*, and *Streptococcus milleri*), diphtheroids, *P. multocida*, other *Pasteurella* species, *Haemophilus influenzae*, *Actinobacillus suis*, *Flavobacterium* IIb–like organisms, *Bacteroides fragilis*, and other anaerobic gram-negative bacilli [23, 45–48].

Aquatic animal bite infections are caused by organisms of their marine environment [26,49–54]. These infections often involve *Vibrio* and *Aeromonas* spp, which also can be recovered from the oral cavity of the shark [44,49–51]. *Vibrio carchariae* is found in shark bite infections [50]. *Aeromonas hydrophila* has been recovered from cellulitis that occurred after a piranha bite [53] and alligator bites [54]. Isolates from catfish bites and injuries [26] have been *Vibrio* spp (as *Vibrio vulni ficus*), *Vibrio damsela*, *Pseudomonas* spp, *Enterobacteriaceae*, *A. hydrophilas*, and *Peptostreptococcus* spp.

Bird pecking and bites can cause serious infections. A brain abscess that was caused by *Streptococcus bovis*, *Clostridium tertium*, and *Aspergillus niger* developed in an infant after a rooster pecked his skull [55]. An owl attack caused cellulitis due to non-fragilis *Bacteroides* species [56], and a swan bite induced cellulitis due to *Pseudomonas aeruginosa* [57].

Ferrets caused severe facial injuries in three infants, and *S. aureus* was recovered in one patient's cellulitis [58].

Pathogenesis

The potential for infection of human or animal bites is great. For example, a dog's teeth are not sharp but can exert a pressure of 200 to 450 psi [59]. This pressure is strong enough to perforate sheet metal and result in a crush injury with much devitalized tissue, rather than a laceration. The average dog mouth harbors more than 64 species of bacteria, including *S. aureus*, *P. multocida*, anaerobic bacteria (especially of the pigmented *Prevotella* and *Porphynomonas* sp) and CDC types IIj (*Bergeyella zoohelcum* and EF-4 [*Pasteurella*-like])—all known human pathogens [32,38]. Because anaerobes predominate in the normal oral flora of humans and various animals, it follows that they have an important role in oral contamination of bite wounds.

It is also evident that these wounds contain polymicrobial aerobic and anaerobic flora, which are known to have a synergistic relationship, thus making the infection harder to eradicate [60]. This situation is especially the case in human bite wounds, in which mixed aerobic and anaerobic flora were recovered in 83% of cases [33].

Differences were noted in the microbiology of human bite wounds and animal bite wounds [33]. The most striking difference was the higher number of isolates per wound in human bites compared with animal bites (5.4 versus 2.8 isolates per specimen). This difference mainly is related to the higher isolation rate of anaerobic bacteria (mostly gram-negative bacilli) in human bite wounds compared with animal bite wounds (3.0 versus 1.0). Differences in other flora in these wounds also were noted. *P. multocida*, *Pseudomonas fluorescens* group, and *N. weaveri* (formerly M-5), which are part of the oral flora of dogs, were recovered only in animal bite wounds.

A number of risk factors determining the likelihood of wound infection have been identified and define the patient likely to develop this complication [61]. An important risk factor is delay of more than 24 hours in seeking treatment. Puncture wounds are much more likely than other types to become infected. Facial wounds show an infection rate of only 4% regardless of treatment, whereas hand wounds have an infection rate of 28% [61]. Septicemia mainly occurs in compromised hosts, asplenic patients who are prone to fulminant septicemia and shock with *Capnocytophaga canimorsus* [62–64] or *Eubacterium plautii* [65] after dog bites.

Diagnosis

The symptoms occurring after a bite depend on the animal species inflicting the injury. Immediate local or systemic symptoms can be severe after venomous animals (snake, lizard, spider, and so forth). Human or dog bites generally do not cause immediate symptoms different from those of a laceration injury. Because of the direct introduction of oral and skin flora into the wound, however, if an infection occurs, it develops quite rapidly. The signs of infection can include redness, swelling, and clear or pussy discharge. The adjacent lymph nodes may be enlarged, and reduction in range of movement of an extremity can be present. In severe cases, there may be a peripheral leukocytosis of 15,000 to 30,000 cells per cubic millimeter. Presence of eschariform lesions in sick-appearing individuals may suggest the presence of *C. canimorsus* infection [66].

Human bites generally are more severe than animal bites, particularly in clenched-fist injuries when the skin over the knuckles is penetrated after striking the teeth of another person. The teeth may cause a deep laceration that implants oral and skin organisms into the joint capsules or dorsal tendons, causing septic arthritis or osteomyelitis. Radiographs of hands injured by teeth are recommended [67]. Measurement of sedimentation rate or C-reactive protein can be helpful in cases of osteomyelitis and pyogenic arthritis to determine the duration of antimicrobial therapy.

Not all bites cause infection. Approximately 2% to 5% of all typical dog bite wounds seen in emergency departments become infected [5,61]. This figure includes, however, many trivial surface abrasions. Wounds that fully penetrated the skin have an infection rate of 6% to 13%, depending on location [61]. In comparison, the infection rate of clean lacerations of all types repaired in the emergency department is approximately 5% [68].

Culture specimens for both aerobic and anaerobic bacteria should be obtained from human and animal bite wounds. In wounds that are contaminated by soil or vegetative debris, culture for mycobacteria and fungi should be done. The use of Gram's stain as an indicator of the presence of pathogens in the wound can be of assistance.

Management

Wound management includes the administration of proper local care and the utilization, when needed, of proper antimicrobial agents. Evaluation and wound care for bites include recording medical history (animal involved, provoked or unprovoked attack, current medications, splenectomy, mastectomy, allergies, chronic disease, and immunosuppression); examining the wound and related structures (odor of exudates, depth, type, and location of wound, range of motion, joint involvement, edema or crush injury, nerve and tendon damage, presence of infection); obtaining wound cultures; irrigating the wound with saline and debridement; taking radiographs (when bone penetration is suspected); performing wound approximation; administering antimicrobials and tetanus and rabies immunizations (when indicated); evaluating for herpes B virus (in monkey bites); and re-examining the patient at 24 and 48 hours. The incident should be reported to the local health department when indicated.

The rules governing the management of any laceration also apply to animal bites: cleanse, explore, irrigate, debride, drain, and possibly suture. Bite wounds should be washed vigorously with soap or a quaternary ammonium compound and water. This step is of primary importance in reducing the high inoculum of the oral flora of the biting human or animal. The physician should explore for damage to tissues caused by crushing or tearing and search for damaged tendons, blood vessels, joints, and bones. Radiographic examination for fractures and foreign bodies should be done when feasible. The wound should be irrigated through a 19-gauge needle with 150 mL or more of sterile normal saline or lactated Ringer's solution. Devitalized tissues should be debrided. Drainage of the wound, when necessary, can be done in customary fashion or with gentle suction using a 19-gauge scalp vein tubing connected to a vacuum blood-collecting tube [69]. Whether bite wounds that are clinically uninfected and are seen within 24 hours should be surgically closed remains controversial [38,61,70]. Margins of puncture wounds should be excised and left open after irrigation. Margins of other wounds should be excised carefully and primary closure should be carried out, with or without drainage [69]. The utility of suturing fresh bite wounds less than 6 hours after the injury is undetermined, except for face wounds. Delayed primary closure or edge approximation should be the practice in wounds associated with crush injury, pre-existing edema, and hand and feet injuries.

In case of bite by a monkey that may be a B virus carrier, the wound should be thoroughly scrubbed with soap or detergent and irrigated for at least 15 minutes to reduce the viral inoculum [42]. Viral culture tests should be performed after cleansing. Serum for acute viral B virus-specific serology should be stored at $-20^{\circ}C$ and compared with a second sample obtained 3 weeks later. Antiviral therapy with acyclovir, valcyclovir, or famcyclovir should be administered to patients with moderate-risk or high-risk wounds [42].

Bites of the hand are at high risk for deep damage and severe infection because sharp teeth may penetrate tendon sheaths or the midpalmar space. Nardi and Zuidema [71] recommend that human bites be treated by widely opening the wound, debriding, and irrigating thoroughly. Primary closure and tendon nerve repair should be delayed. After debridement and irrigation, dog bites can be considered clean, and primary closure can be carried out. Hospitalization for several days is recommended, with immobilization by splinting or bulky dressings and elevation.

Bites to the face, especially of children, require meticulous management. Nearly all facial bite patients do well with careful debridement, ample irrigation and cleansing, and loose closure by suture. Close follow-up for 5 days or longer is required. Subsequent plastic reconstruction may be considered, and consultation with a plastic surgeon at the time of initial repair may be helpful.

Early treatment of all human bites, especially those to the hand, must be thorough and aggressive. Unfortunately, the injury sometimes is seen only after severe infection has occurred. Clenched-fist injuries require more intensive care, preferably by a hand surgeon, to evaluate the degree of injury to the tendon, sheath, joint capsule, joint, and bone.

Rabies prevention should be instituted after dog bites that indicate such measures [72]. This preventive measure includes hyperimmune serum and active immunization. A tetanus toxoid booster should be administered if the patient has been adequately immunized previously and has received the last dose within the past 10 years. Tetanus immune globulin (human) is required if tetanus immunization has not taken place or is inadequate.

The infectious complications of dog bites make the concept of prophylactic antibiotics attractive. Using antibiotics may be helpful, particularly in high-risk wounds such as those of the hand. The choice of a particular antibiotic for prophylaxis or treatment must be based on bacteriology. Unfortunately, no one antibiotic can be expected to effectively treat infections caused by all the organisms that can be present in an infected bite.

The role of prophylactic antimicrobial therapy in bite wounds presented early is uncertain [61,67,69]; however, because these wounds usually are contaminated with potential pathogens, prophylactically treating all patients having deep bite wounds with antibiotics is advisable. These wounds include puncture wounds, facial bites, and any wound over a tendon or bone. Antimicrobial therapy should be administered for all bite wounds, with the exception of those patients who present 24 hours or more after injury and have no clinical signs of infection. Antimicrobial therapy of bite wounds is not usually a prophylactic, but rather a therapeutic, intervention.

Because no single antimicrobial eradicates all of the major pathogens responsible for bite wound infections, establishing a specific etiologic diagnosis by obtaining cultures is useful to guide therapy [73]. Penicillin or ampicillin are the most active agents against *P. multocida* and the other oral flora; however, *S. aureus* and almost half of the anaerobic gram-negative bacilli present in human bite wounds are resistant to these drugs [74]. The recovery of beta-lactamase–producing organisms from more than 40% of bite wounds raises the question of whether penicillins are adequate therapy for bite infections [33]. Although oxacillin is effective against *S. aureus*, it has poor activity against many bite isolates; 18% of *P. multocida*, 24% of *Bacteroides* sp, and more than 50% of other aerobic gram-negative strains were found to be resistant to this antimicrobial [74]. Tetracyclines are good alternatives but should not be used in young children. When *S. aureus* is suspected (based on the Gram stain of aspirate, which is specific but not sensitive), penicillin and a penicillinase-resistant penicillin should be used. The combination of amoxicillin and clavulanic acid has been shown to be effective in therapy for human bites and dog bites [73,75]. This effectiveness is related to the wide spectrum of activity of the combination against most pathogens isolated from these wounds. First-generation cephalosporins are not as effective as the amoxicillin and clavulanic acid combination because of resistance of some anaerobic bacteria and *Eikenella corrodens*. Clindamycin and the penicillinase-resistant penicillins should not be administered without penicillin because of their poorer activity against *P. multocida*. Erythromycin is generally ineffective against *P. multocida, Moraxella* species, fusobacteria, and peptostreptococci. Azithromycin is generally more active than clarithromycin against all *Pasteurella* species. Azithromycin and clarithromycin are only modestly effective against *E. corrodens* and *Peptostreptococcus* spp. Cefoxitin or the combination of penicillin or a first-generation cephalosporin plus a beta-lactamase–resistant penicillin or the combination of ticarcillin and clavulanic acid will provide adequate parenteral therapy for animal and human bites. The newer quinolones (moxifloxacin and gatifloxacin) also possess good activity against all major bite wound pathogens, including anaerobic bacteria [76]; however, their use in pediatric cases is restricted.

E. corrodens, a capnophilic gram-negative rod that is part of the normal oral flora [28], can be isolated from 25% of human bite wounds [32]. This finding is of note because of the unusual antibiotic sensitivity pattern of *E. corrodens*. It is susceptible to penicillin, ampicillin, and the quinolones but resistant to oxacillin, methicillin, nafcillin, and clindamycin, and some strains are resistant to cephalosporins [73,74,76]. When isolated, *E. corrodens* should have susceptibility testing if cephalosporin therapy is to be considered.

When antibiotics are used in this manner and are combined with good wound dressing, most bite wounds may be sutured with good results and an acceptable infection rate. Duration and route of antibiotic therapy should be individualized based on the site involved, the culture results, and the response to treatment. A 7- to 14-day course is usually adequate for infections limited to soft tissue, and a minimum of 3 weeks' therapy is required for those infections involving joints or bones.

Complications

Hand wounds present a special problem, because 30% or more become infected [59,61]. Because of the presence of avascular tendon and sheath spaces, the propensity for spread of infection, and disastrous results of such infection on function, the threat of complications after bite wounds must be addressed. In addition to local wound infection, other complications include lymphangitis, local abscess, septic arthritis, tenosynovitis, and osteomyelitis [33,67]. Rare complications include endocarditis, meningitis [77], brain abscess [78], and sepsis with disseminated intravascular coagulation [79], especially in immunocompromised individuals. Patients who are especially prone to complications include those receiving systemic corticosteroids, those with lupus erythematosus, and those with acute leukemia. Rabies also must be considered; its prophylaxis entails considerable expense and morbidity [72].

References

[1] McCaig LF. National Hospital Ambulatory Medical Care Survey: 1998 emergency department summary. Adv Data 2000;10:1–23.
[2] Farmer CB, Mann RJ. Human bite infections of the hand. South Med J 1966;59:515–8.
[3] Mann RJ, Hoffeld TA, Farmer CB. Human bites of the hand: twenty years of experience. J Hand Surg 1977;2:97–104.
[4] Thomson HG, Svitek V. Small animal bites: the role of primary closure. J Trauma 1973; 13:20–3.
[5] Kizer KW. Epidemiologic and clinical aspects of animal bite injuries. JACEP 1979;8:134–41.
[6] Litovitz TL, Klein-Schwartz W, White S, et al. 2000 annual report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. Am J Emerg Med 2001;19:337–95.
[7] Goldstein EJC, Pryor EP, Citron DM. Monkey bites and infection. Clin Infect Dis 1995; 20:1551–2.
[8] Goldstein EJC. New horizons in the bacteriology, antimicrobial susceptibility and therapy of animal bite wounds. J Med Microbiol 1998;47:95–7.
[9] Goldstein EJC, Citron DM, Finegold SM. Role of anaerobic bacteria in bite wound infections. Rev Infect Dis 1984;6(Suppl 1):177–83.

[10] Alexander CJ, Citron DM, Gerardo SH, Clams MC, Talan D, Goldstein EJC. Characterization of saccharolytic *Bacteroides* and *Prevotella* isolates from infected dog and cat bite wounds in humans. J Clin Microbiol 1997;35:406–11.
[11] Citron DM, Clams MC, Gerardo SH, Abrahamian F, Talan D, Goldstein EJC. Frequency of isolation of *Porphyronionas* species from infected dog and cat bite wounds in humans and their characterization by biochemical tests and AP-PCR fingerprinting. Clin Infect Dis 1996;23(Suppl 1):78–82.
[12] Goldstein EJC, Citron DM, Nesbit C, et al. Emergency Medicine Animal Bite Infection Study Group. Prevalence and characterization of anaerobic bacteria from 50 patients with infected cat and dog bite wounds. In: Bennett EA, editor. Anaerobic pathogens. Sheffield (UK): Sheffield Academic Press; 1997. p. 177–85.
[13] Weber DJ, Wolfson JS, Swartz MN, Hooper DC. *Pasteurella multocida* infections: report of 34 cases and review of the literature. Medicine (Baltimore) 1984;63:133–54.
[14] Holst E, Rollof J, Larsson L, Nielsen JP. Characterization and distribution of *Pasteurella* species recovered from infected humans. J Clin Microbiol 1992;30:2984–7.
[15] Brenner DJ, Hollis DG, Fanning GR, Weaver RE. *Capnocytophaga canimorsus* sp. nov. (formerly CDC group DF-2), a cause of septicemia following dog bite, and *C. cynodegmi* sp. nov., a cause of localized wound infection following dog bite. J Clin Microbiol 1989; 27:231–5.
[16] Graham DR, Band JD, Thornsberry C, et al. Infections caused by *Moraxella, Moraxella urethralis, Moraxella*-like groups M-5 and M-6, and *Kingella kingae* in the United States, 1953–1980. Rev Infect Dis 1990;12:423–31.
[17] Andersen BM, Steigerwalt AG, O'Connor SP, et al. *Neisseria weaveri* sp. nov., formerly CDC group M-5, a gram-negative bacterium associated with dog bite wounds. J Clin Microbiol 1993;31:2456–66.
[18] Reina J, Borrell N. Leg abscess caused by *Weeksella zoohelcum* following a dog bite. Clin Infect Dis 1992;14:1162–3.
[19] Guidourdenche M, Lambert T, Riou JY. Isolation of *Neisseria canis* in mixed culture from a patient after a cat bite. J Clin Microbiol 1989;27:1673–4.
[20] Talan DA, Goldstein EJC, Staatz D, Overturf GD. *Staphylococcus intermedius*: clinical presentation of a new human dog bite pathogen. Ann Emerg Med 1989;18:410–3.
[21] Hollis DG, Moss CW, Daneshvar MI, et al. Characterization of Centers for Disease Control group NO-1, a fastidious, nonoxidative, gram-negative organism associated with dog and cat bites. J Clin Microbiol 1993;31:746–8.
[22] Moss CW, Wallace PL, Hollis DG, Weaver RE. Cultural and chemical characterization of CDC groups EO-2, M-5 and M-6, *Moraxella* (*Moraxella*) species, *Oligella urethralis*, *Acinetobacter* species, and *Psychrobacter immobilis*. J clin Microbiol 1988;26:484–92.
[23] Goldstein EJC, Citron DM, Merkin TE, Pickett MJ. Recovery of an unusual *Flavobacterium* group IIb-like isolate from a hand infection following pig bite. J Clin Microbiol 1990;28:1079–81.
[24] Peel MM, Hornidge KA, Luppino M, et al. *Actinobacillus* spp. and related bacteria in infected wounds of humans bitten by horses and sheep. J Clin Microbiol 1991;29:2535–8.
[25] Erickson T, Vanden Hoek TL, Kuritza A, Leiken JB. The emergency management of moray eel bites. Ann Emerg Med 1992;21:212–6.
[26] Murphey DK, Septimus EJ, Waagner DC. Catfish-related injury and infection: report of two cases and review of the literature. Clin Infect Dis 1992;14:689–93.
[27] Capellan J, Fong IW. Tularemia from a cat bite: case report and review of feline-associated tularemia. Clin Infect Dis 1993;16:472–5.
[28] Manson ML, Koch SL. Human bite infections of the hand. Surg Gynecol Obstet 1930; 51:591–5.
[29] Welch CE. Human bite infection of the hand. N Engl J Med 1936;215:901–5.
[30] Guba AM, Mulliken JB, Hoopes JE. The selection of antibiotics for human bites of the hand. Plast Reconstr Surg 1975;56:538–41.

[31] Shields C, Patzakis MJ, Meyers MH, Harvey JP Jr. Hand infections secondary to human bites. J Trauma 1975;15:235–6.
[32] Goldstein JC, Citron DM, Wield B, et al. Bacteriology of human and animal bite wounds. J Clin Microbiol 1978;8:667–72.
[33] Brook I. Microbiology of human and animal bite wounds in children. J Pediatr Infect Dis 1987;6:29–32.
[34] Francis DP, Holmes MA, Brandon G. *Pasteurella multocida* infections after domestic animal bites and scratches. JAMA 1975;233:42–5.
[35] Hawkins LG. Local *Pasteurella multocida* infections. J Bone Joint Surg 1969;51:362–6.
[36] Saphir DA, Carter GR. Gingival flora of the dog with special reference to bacteria associated with bites. J Clin Microbiol 1976;3:344–9.
[37] Bailie WE, Stowe EC, Schmitt AM. Aerobic bacterial flora of oral and nasal fluids of canines with reference to bacteria associated with bites. J Clin Microbiol 1978;7:223–31.
[38] Goldstein EJC, Citron DM, Finegold SM. Dog bite wounds and infection: a prospective clinical study. Ann Emerg Med 1980;9:508–12.
[39] Talan DA, Citron DM, Abrahamian FM, Moran GJ, Goldstein EJ. Bacteriological analysis of infected dog and cat bites. Emergency Medicine Animal Bite Infection Study Group. N Engl J Med 1999;340:85–92.
[40] Goldstein EJC, Citron DM, Finegold SM. Dog bite wounds and infection: a prospective clinical study. Ann Emerg Med 1980;9:508–12.
[41] Weigler BJ. Biology of B virus in macaque and human hosts: a review. Clin Infect Dis 1992;14:555–67.
[42] Holmes GP, Chapman LE, Stewart J, Straus SE, Hilliard JK, Davenport DS. B Virus Working Group. Guidelines for the prevention and treatment of B virus infections in exposed persons. Clin Infect Dis 1995;20:421–39.
[43] Bailey GD, Moore LVH, Love DN, Johnson JL. *Bacteroides heparinolyticus*: deoxyribonucleic acid relatedness of strains from the oral cavity and oral-associated disease conditions of horses, cats, and humans. Int J Syst Bacteriol 1988;38:42–4.
[44] Benaoudia F, Escande F, Simonet M. Infection due to *Actinobacillus lignieresii* after a horse bite. J Clin Microbiol Infect Dis 1994;13:439–40.
[45] Barnham M. Pig bite injuries and infection: report of seven human cases. Epidemiol Infect 1988;101:641–5.
[46] Escande F, Bailly A, Bone S, Lemozy J. Actinobacillus suis infection after a pig bite. Lancet 1996;348:888.
[47] Rolle U. *Haemophilus influenzae* cellulitis after bite injuries in children. J Pediatr Surg 2000;35:1408–9.
[48] Morgan MS. Treatment of pig bites. Lancet 1996;348:1246.
[49] Auerbach PS, Yajko DM, Nassos PS, et al. Bacteriology of the marine environment: implications for clinical therapy. Ann Emerg Med 1987;16:643–9.
[50] Pavia AT, Bryan JA, Maher KL, Hester R Jr, Framer JJ III. *Vibrio carchariae* infections after a shark bite. Ann Intern Med 1989;111:85–6.
[51] Buck JD, Spotte S, Gadbaw JJ Jr. Bacteriology of the teeth from a great white shark: potential medical implications for shark bite victims. J Clin Microbiol 1984;20:849–51.
[52] Rosenthal SG, Bernhardt HE, Phillips JA III. *Aeromonas hydrophila* wound infections. Plast Reconstr Surg 1974;53:77–9.
[53] Revord ME, Goldfarb J, Shurin SB. *Aeromonas hydrophila* wound infection in a patient with cyclic neutropenia following a piranha bite. Pediatr Infect Dis J 1988;7:70–1.
[54] Flandry F, Lisecki EJ, Domingue GJ, Nichols RL, Greer DL, Haddad RJ Jr. Initial antibiotic therapy for alligator bites. South Med J 1989;82:262–6.
[55] Berkowitz FE, Jacobs DWC. Fatal case of brain abscess caused by rooster pecking. Pediatr Infect Dis J 1987;6:941–2.
[56] Davis B, Wenzel RP. Stringes scalp: *Bacteroides* infection after an owl attack. J Infect Dis 1992;165:975–6.

[57] Eberly RJ, Hayek LJ. Antibiotic prophylaxis after a swan bite. Lancet 1997;350:340.
[58] Paisley JW, Lauer BA. Severe facial injuries to infants due to unprovoked attacks by pet ferrets. JAMA 1988;259:2005–6.
[59] Chambers G, Payne J. Treatment of dog bite wounds. Minn Med 1969;52:427–30.
[60] Brook I, Hunter V, Walker RI. Synergistic effect of *Bacteroides*, *Clostridium*, *Fusobacterium*, anaerobic cocci and aerobic bacteria on mortality and induction of subcutaneous abscesses in mice. J Infect Dis 1984;149:924–9.
[61] Callaham M. Treatment of common dog bites: infection risk factors. JACEP 1978;7:83–7.
[62] Hicklin H, Verghese A, Alvarez S. Dysgonic fermenter 2 septicemia. Rev Infect Dis 1987;9:884.
[63] Carpenter PD, Heppner BT, Gnann JW. DF-2 bacteremia following cat bites: report of two cases. Am J Med 1987;82:621–3.
[64] Kullberg B-J, Westendorp RGJ, van't Wout JW, Meinders AE. Purpura fulminans and symmetrical peripheral gangrene caused by *Capnocytophaga canimorsus* (formerly DF-2) septicemia—a complication of dog bite. Medicine (Baltimore) 1991;70:287–92.
[65] Garre M, le Henaff C, Tande, et al. Fulminant *Eubacterium plautii* infection following dog bite in asplenic man. Lancet 1991;338:384–5.
[66] Kalb R, Kaplan MH, Tenenbaum MJ, et al. Cutaneous infection at dog bite wounds associated with fulminant DF-2 septicemia. Am J Med 1985;78:687–90.
[67] Szalay GC, Sommerstein A. Inoculation osteomyelitis secondary to animal bites. Clin Pediatr 1972;11:687–9.
[68] Galvin JR, DeSimone D. Infection rate of simple suturing. JACEP 1976;5:332–3.
[69] Graham WP III, Calabretta AM, Miller SH. Dog bites. Am Fam Physician 1977;15:132–7.
[70] Maroy SM. Infections due to dog and cat bites. Pediatr Infect Dis 1982;1:351–4.
[71] Nardi GL, Zuidema GD. Surgery: a concise guide to clinical practice. 3rd edition. Boston: Little, Brown; 1972.
[72] Update on emerging infections from the Centers for Disease Control and Prevention. Update: rabies postexposure prophylaxis guidelines. Ann Emerg Med 1999;33:590–7.
[73] Goldstein EJ. Selected nonsurgical anaerobic infections: therapeutic choices and the effective armamentarium. Clin Infect Dis 1994;18(Suppl 4):S273–9.
[74] Goldstein EJC, Citron DM, Vagvolgyi AE, Finegold SM. Susceptibility of bite wound bacteria to seven oral antimicrobial agents, including RU-285, a new erythromycin: consideration for choosing empiric therapy. Antimicrob Agents Chemother 1986;29:556–9.
[75] Goldstein EJ, Citron DM. Comparative activities of cefuroxime, amoxicillin-clavulanic acid, ciprofloxacin, enoxacin, and ofloxacin against aerobic and anaerobic bacteria isolated from bite wounds. Antimicrob Agents Chemother 1988;32:1143–8.
[76] Goldstein EJ, Citron DM, Merriam CV, Tyrrell K, Warren Y. Activity of gatifloxacin compared to those of five other quinolones versus aerobic and anaerobic isolates from skin and soft tissue samples of human and animal bite wound infections. Antimicrob Agents Chemother 1999;43:1475–9.
[77] Bracis R, Seibers K, Jullien RM. Meningitis caused by IIj following a dog bite. West J Med 1979;131:438–40.
[78] Klein DM, Cohen ME. *Pasteurella multocida* brain abscess following perforating cranial dog bite. J Pediatr 1978;92:588–9.
[79] Check W. An odd link between dog bites, splenectomy. JAMA 1979;241:225–6.