

## **BITE WOUND INFECTIONS**

### **Martin Rodriguez**

#### **EPIDEMIOLOGY**

It is estimated that 108 million cats and dogs are kept as pets in the United States. Approximately half of all Americans will suffer a bite wound during their lifetime (1). The CDC estimates that approximately 3.73 million animal bites occur in the United States each year from dogs alone; 20% of the bites require medical attention (1,2). Animal bites are responsible for up to 1% of all emergency health care visits, with dog bites representing up to 90% of all animal bites evaluated in this setting. Cat and human bites represent the second and third most common mammalian bites respectively. Species of animals that cause at least 1 percent of bite injuries are rabbits, skunks, squirrels, horses, hogs, rats, and monkeys. The laboratory animal population is another source of bites; monkeys and rats are the most common offenders (3). The highest incidence of bites occurs in boys 5-9 years of age (4). Approximately half of animal bites occur on the hands and one fourth on the forearm or arm. Facial bites account for 10% of bites, most of them are sustained by children younger than 10 years. Most of the animal bites to children are caused by animals known to the children. Ten to 20 fatal human attacks occur yearly in the United States (5). Human bites occur with regularity among children 2-4 years of age. Human bites are responsible for approximately 1 in 600 emergency department visits. Although most bites occur during fights between children, predominantly with injury to the fingers, forearms, and cheeks, bites during play are not uncommon. As children grow to adolescence and adulthood, bites occur during other activities, such as sports or sexual activity. The annual incidence of snake bites in the United States is about 45000, of which 10% to 18% are attributed to venomous snakes. Between 9 and 15 persons die of snake bites each year in the United States (1).

Bite victims who seek medical attention can be classified into two groups based upon the time of presentation. The first group presents within 8-12 hours of the incident, with fears of contracting rabies or other infections, and/or with concerns of permanent disfigurement of the injured body part; these wounds are often contaminated with bacteria but do not show evidence of infection. The second group seeks help more than 12 hours after the incident, most often presenting with signs and symptoms of developing infections (6,7).

Between 5% and 60% of all bite wounds are complicated by infection. Approximately 20% to 50% of cat bites, 10% to 50% of human bites, and 3% to 20% of dog bites will become infected. With the exception of monkey bites, which have a 25% infection rate, infection developing after mammalian bites is uncommon (5,8). Among children hospitalized for dog bite injuries, two thirds of the wounds are complicated by infection. Anecdotally, bites by wild animals, farm animals, and reptiles have been complicated by infection, but accurate incidence data are not available. Rat bites primarily affect children less than 5 years of age, with the majority of bites inflicted on the face and hands. Infections from laboratory animals bites are rare, but tissue damage is not. Several risk factors for bite wound infection have been reported from multiple studies. Table 1 summarizes these risk factors (6,9).

**Table 1: Risk factors associated with a high rate of infection of bite wounds**

Age < 2 and > 50 years
Comorbidities (liver disease, splenectomy, DM, malignancy, HIV, vascular disease)
Preexisting edema in the area of bite
Chronic alcohol consumption
Use of immunosuppressive drugs (including chronic steroids)
Cat and human bites
Moderate to severe wounds
Puncture wound, large avulsion, crush injury

Presence of foreign material and/or heavily contaminated wound
Hand, wrist or foot wound
Scalp or face in infants and young children
Associated injuries to bone, joint, tendon sheath, or neurovascular structures
Adjacent to prosthetic joint
Delay in care > 24 hours
Improper wound cleansing or débridement

## **PATHOGENESIS**

Injuries from animal bites reflect the anatomy of the teeth and the strength of the jaws of the biting animal. Dog bites tend to cause lacerations, with crush and avulsion injuries as a function of the large, broad, sharp teeth and powerful jaws. Bites by cats, mice, rats and snakes tend to cause puncture wounds because of the characteristic sharp, elongated, needle-like teeth (2). Human bites more closely resemble dog bites than cat bites, with abrasions and laceration being more common than punctures. Most infections from animal bites develop at the site of the bite wound and adjacent tissue. Among the various wound types, puncture wounds have the highest incidence of infection. Puncture wounds tend to seal quickly, producing an environment that facilitates growth of anaerobic organisms. With any bite, but particularly with cat bites, the teeth may penetrate not only skin but also underlying structures such as joints, bones, tendons, or tendon sheaths. Puncture wounds may appear innocuous immediately after the injury, but infection often develops in the deeply inoculated tissues within a few hours and may not be recognized. The severity of bacterial bite-wound infections depends on several factors, including the oral flora of the person or animal inoculating the wound, the anatomic location and structures damaged by the wound, the organisms contaminating the wound from the environment or the skin, and the time elapsed since the injury.

Human bites tend to be more superficial than animal bites, however, the infection rate is high. Human bites can be best separated into occlusional and clenched-fist injuries. Occlusional wounds result from teeth sinking into the skin. The majority of human bites in children are superficial abrasions in which the integrity of the skin is not completely broken. These wounds have a lower rate of infection than puncture or laceration wounds. Also, parents tend to bring children to medical attention rather quickly after a human bite, and infection rates as low as 3% have been reported in this setting (10). Human bites not involving a clenched-fist mechanisms seem to have a low incidence of infection. In a study of 41 sutured facial human bites treated in a plastic surgery clinic, the infection rate was only 2.5% (11). The clenched-fist injury is more common in adults, and occurs after fistfight or after an accidental sports injury, with the opponent's tooth inoculating the deeper tissue planes, and occasionally tendon sheaths, joint and bone of the hand with oral bacteria. When the injured person opens the fist, the extensor tendon of the finger retracts proximally, carrying the inoculated oral flora into the tendon sheath and simultaneously sealing off the wound. This process produces optimal conditions for the development of an anaerobic infection.

## **MICROBIOLOGY**

The mammalian mouth has been reported to support the growth of over 200 species of facultative organisms and obligate anaerobes, with as many as 100 million organisms per mL of saliva. The oral flora of the animal, rather than the skin flora of the victim, is the source of most bacteria isolated from bite wound cultures. Infections are usually polymicrobial and contain mixed aerobic-anaerobic isolates. Routine cultures of bite lesions without signs of infection are not indicated. Some experts recommend routine culture of hand and face bites as a useful precaution because of the potentially devastating consequences of infections in these locations. For wounds that appear infected, microbiologic studies should be performed. Gram stains may be helpful, however, they may not correlate with culture results. Both aerobic and anaerobic cultures should be performed. Ideally the laboratory should be notified that the source of the culture is a bite wound.

*Pasteurella* spp. may be mistaken morphologically for *Neisseria* spp. or *Haemophilus influenzae*. Cultures should be observed for a minimum of 10 days to allow the growth of some anaerobic organisms. Blood cultures should also be considered if the patient is febrile, although the yield is less than 5%. For infections presenting 5 to 7 days after a bite, some authors recommend obtaining fungal cultures in addition to bacterial cultures. Viral cultures and immunofluorescent assays for HSV infection should be obtained if this pathogen is suspected based on the history and appearance of the lesion.

### **Dog and cat bites**

Early studies suggested that the most common organisms causing dog bite infections were streptococci and staphylococci. With improvement in anaerobic culture techniques investigators realized that other organisms were more frequently implicated in infected bite wounds. In a prospective study of 107 infected dog and cat bite wounds, mixed aerobic and anaerobic bacteria were present in 56% of all wounds (12). Thirty-six percent of the wounds demonstrated purely aerobic growth. One dog bite grew only anaerobic organisms, and the remainder 7% of cultures demonstrated no growth. The median number of isolates per culture was 5, approximately 3 were aerobes and 2 were anaerobes. *Pasteurella* spp. was the most common pathogen isolated from both dog and cat bites (50% and 75% respectively). *Pasteurella canis* was the most common isolate of dog bites, and *Pasteurella multocida* subspecies *multocida* and *septica* were the most common isolates of cat bites. The next more frequently encountered aerobic organisms were streptococci, and staphylococci. Streptococci were seen in 46% of the cases, staphylococci were seen in 46% of dog bites and 35% of cat bites, *Staphylococcus aureus* was cultured in 20% of dog bites and 4% of cat bites.

*Staphylococcus intermedius* was found in two cases. Other common organisms were *Neisseria* spp, *Corynebacterium* spp., and *Moraxella* spp. The most common anaerobic organisms isolated were *Fusobacterium* spp., *Bacteroides* spp. (in particular *B. tectum*), *Porphyromonas* spp., *Prevotella* spp., *Propionibacterium acnes*, and *Peptostreptococcus* spp. *Erysipelothrix rhusiopathiae* was isolated from 2 cat bites. Table 2 shows the complete list of isolates. The presence of  $\beta$ -lactamase-producing anaerobic organisms in the mouth flora of these animals is uncommon.

### **Human bites**

Human bites typically transfer a larger number of bacteria to the bite victim than dog or cat bites, primarily because the human mouth carries a higher population of resident bacteria (6). The major difference in the microbiology of human bite wounds and those from either dog or cat bites include the presence of *Eikenella corrodens*, the absence of *P. multocida*, and a higher incidence of  $\beta$ -lactamase-producing organisms. Rare cases of *Streptococcus pyogenes* necrotising fasciitis after human bites have been reported (13).

Earlier studies noted streptococci and staphylococci to be the most common organisms isolated. Most studies used a limited number of patients and did not use adequate anaerobic methodology (14,15). In the 1980s the importance of anaerobic organisms and *E. corrodens* in infected human bites was recognized. *E. corrodens* has been found in over 30% of infected human bites. It is commonly found in combination with streptococci and staphylococci; it is very frequent in infected clenched-fist injuries (16). In a prospective study that was recently published, 50 patients with infected human bite wounds were evaluated and cultured with advanced anaerobic techniques (15). Fifty-six percent of injuries were clenched-fist injuries and 44% were occlusional bites. The median number of isolates per wound culture was 4 (3 aerobes and 1 anaerobe). Aerobes and anaerobes were isolated from 54%, aerobes alone from 44%, and anaerobes alone were isolated from 2%. Streptococci were by far the most common isolates (84%), with *Streptococcus anginosus (milleri)* being the most common (52%). *S. pyogenes* was present in 14% of the cases. Other less frequently found streptococci were *S. oralis*, *S. intermedius*, *S. mitis*, *S. constellatus*, *S. parasanguis*, viridans group *Streptococcus* and *Streptococcus* group C or G. Staphylococci were the next most common isolates (52%), with *S. aureus* being the most common (30%). Other staphylococci were *S. epidermidis* (22%), other coagulase-negative *Staphylococcus* and *S. saprophyticus*. *E. corrodens* was isolated in 30% of the cases, *Haemophilus* spp. in 22%. Less common aerobic organisms were *Corynebacterium* spp. (12%), *Gemella* spp. (12%), *Candida* spp. (8%), *Enterobacter cloacae* (8%), *Neisseria* spp. (8%), *Enterococcus* spp. (6%), *Klebsiella* spp. (6%), and

others. Common anaerobes included *Prevotella* spp. in 36% of the cases (*Prevotella melaninogenica* in 22%), *Fusobacterium* spp. in 34% (*F. nucleatum* in 32%), *Veillonella* spp. (24%), *Peptostreptococcus* spp. (22%), *Campylobacter* spp. (16%), *Eubacterium* spp. (16%), *Actinomyces* spp. (8%), *Lactobacillus* spp. (8%), and others. The importance of the specimen transport and microbiologic methods was shown by the finding that the average number of isolates grown per specimen was double in the reference laboratory compared with the local laboratory. *E. corrodens* and anaerobes were less likely to be isolated in local laboratories.

### **Rodent bites**

The incidence of rat-bite fever after a rodent bite is unknown but the disease has been noted to occur in up to 10% of patients with rodent bites presenting for medical care (2). Rat-bite fever is a clinical syndrome caused by one of two different organisms, *Streptobacillus moniliformis* and *Spirillum minor*, that are part of the normal rodent flora. *Pasteurella* spp., *Leptospira interrogans*, and coagulase-negative *Staphylococcus* have also been reported after rat bites (17).

### **Monkey bites**

A major concern after a monkey bite is transmission of B herpes virus (macaques in particular). The bacteriology of monkey bite wounds is similar to human bites; organisms that have been isolated include streptococci, *Enterococcus* spp., staphylococci, *E. corrodens*, *Neisseria* spp., Enterobacteriaceae, *Bacteroides* spp., *Prevotella* spp., *Fusobacterium* spp. The frequency of  $\beta$ -lactamase-producing anaerobes is similar to the one seen in humans (9,14,18).

### **Reptile bites**

Snake bites appear to have a higher rate of infection by enteric gram-negative bacilli than mammalian bites. This finding is thought to reflect the manner in which a snake swallows its prey headfirst, with the animal's fecal flora subsequently colonizing the snake's mouth. Snake venom is sterile, although extensive necrosis of tissue may occur after a bite, providing an optimal environment for growth of inoculated organisms. Organisms that have been reported are *Pseudomonas aeruginosa*, *Proteus* spp, *Salmonella arizonae*, *Aeromonas* spp., *Vibrio vulnificus*, *Bacteroides fragilis* and *Clostridium* spp. Gram-positive skin flora are also frequently isolated from snake bite wound infections, including staphylococci. *Serratia marcescens* cellulitis has been reported after an iguana bite (19). In persons sustaining alligator bite wounds, infection is caused most commonly by *Aeromonas hydrophila*. Other organisms that have been reported are *Enterobacter agglomerans*, *Citrobacter diversus*, *Proteus* spp., *Pseudomonas* spp., *Serratia* spp., and *Clostridium* spp (20).

### **Pig bites**

Cultures of infected pig bite wounds have grown *Streptococcus agalactiae*, staphylococci, *Streptococcus sanguis*, *S. equisimilis*, *S. suis*, *S. anginosus*, diptheroids, *P. multocida*, *Pasteurella aerogenes*, *Actinobacillus suis*, *Flavobacterium* spp, *H. influenzae*, *Proteus* spp., *E. coli*, *B. fragilis* and *Francisella tularensis* (9,14,21,22).

### **Horse bites**

*S. anginosus* and *S. mutans* infections after horse bites have been reported to cause extensive gas in the tissues. *Actinobacillus lignieresii* has been isolated after a horse bite. Other organisms to consider are *S. aureus*, *Neisseria* spp., *E. coli*, *Pasteurella* spp., *Bacteroides ureolyticus*, *B. fragilis*, *Prevotella melaninogenica*, *P. heparinolytica* (9,21).

Table 2: Aerobic and anaerobic isolates from 50 dog bites and 57 cat bites (12)

BACTERIA	DOG BITE	CAT BITE	BACTERIA	DOG BITE	CAT BITE	BACTERIA	DOG BITE	CAT BITE
	no. of patients (%)			no. of patients (%)			no. of patients (%)	
<b>Aerobes</b>			<b>Aerobes (cont.)</b>			<b>Aerobes (cont.)</b>		
<i>Pasteurella</i>	25 (50)	43 (75)	<i>Moraxella</i>	5 (10)	20 (35)	<i>Actinobacillus</i> †	0	2 (4)
<i>Pst. canis</i>	13 (26)	1 (2)	Other†	5 (10)	18 (32)	<i>Alcaligenes</i>	0	2 (4)
<i>Pst. multocida</i> ssp. <i>multocida</i>	6 (12)	31 (54)	<i>Morax. atarribalis</i>	1 (2)	6 (11)	<i>Alcal. faecalis</i>	0	1 (2)
<i>Pst. stomatis</i>	6 (12)	2 (4)	EF-4b	5 (10)	9 (16)	<i>Alcal. odorans</i>	0	1 (2)
<i>Pst. multocida</i> ssp. <i>septica</i>	5 (10)	16 (28)	<i>Enterococcus</i>	5 (10)	7 (12)	<i>Enterobacter cloacae</i>	0	2 (4)
<i>Pst. dagmatis</i>	2 (4)	4 (7)	<i>Ent. faecalis</i>	3 (6)	2 (4)	<i>Erysipelothrix rhusiopathiae</i>	0	2 (4)
<i>Pst. multocida</i> ssp. <i>gallicida</i>	1 (2)	0	<i>Ent. avium</i>	1 (2)	0	<i>Reimerella anatipesifer</i>	0	2 (4)
Other†	1 (2)	0	<i>Ent. malodominus</i>	1 (2)	0	<i>Rothia dentocariosa</i>	0	2 (4)
<i>Streptococcus</i>	23 (46)	26 (46)	<i>Ent. durans</i>	0	5 (9)	<i>Aeromonas hydrophila</i>	0	1 (2)
<i>Strep. mitis</i>	11 (22)	13 (23)	<i>Bacillus</i>	4 (8)	6 (11)	<i>Pantoea agglomerans</i>	0	1 (2)
<i>Strep. mutans</i>	6 (12)	6 (11)	<i>Bac. firmus</i>	2 (4)	2 (4)	<i>Rhodococcus</i> †	0	1 (2)
<i>Strep. pyogenes</i>	6 (12)	0	<i>Bac. circulans</i>	1 (2)	1 (2)	<i>Streptomyces</i> †	0	1 (2)
<i>Strep. sanguis</i> II	4 (8)	7 (12)	<i>Bac. subtilis</i>	1 (2)	0			
<i>Strep. intermedius</i>	3 (6)	2 (4)	Other†	0	3 (5)	<b>Anaerobes</b>		
<i>Strep. constellatus</i>	2 (4)	2 (4)	<i>Pseudomonas</i>	3 (6)	3 (5)	<i>Fusobacterium</i>	16 (32)	19 (33)
<i>Strep. equinus</i>	1 (2)	3 (5)	<i>Pseud. aeruginosa</i>	1 (2)	0	<i>Fuso. nucleatum</i>	8 (16)	14 (25)
<i>Strep. sanguis</i> I	1 (2)	3 (5)	<i>Pseud. vesicularis</i>	1 (2)	1 (2)	Other†	6 (12)	4 (7)
<i>Strep. agalactiae</i>	1 (2)	1 (2)	<i>Pseud. diminuta</i>	1 (2)	0	<i>Fuso. russii</i>	1 (2)	8 (14)
<i>Strep. sanguis</i>	1 (2)	1 (2)	<i>Pseud. putida</i>	0	1 (2)	<i>Fuso. gonidiaformans</i>	1 (2)	1 (2)
β-Hemolytic, group G	1 (2)	0	<i>Pseud. stutzeri</i>	0	1 (2)	<i>Fuso. alocis</i>	1 (2)	0
<i>Strep. dysgalactiae</i>	1 (2)	0	<i>Actinomyces</i>	3 (6)	2 (4)	<i>Bacteroides</i>	15 (30)	16 (28)
β-Hemolytic, group F	0	1 (2)	<i>Act. viscosus</i>	2 (4)	1 (2)	<i>Ba. tectum</i>	7 (14)	16 (28)
<i>Staphylococcus</i>	23 (46)	20 (35)	<i>Act. neuii</i> ssp. <i>anitratus</i>	1 (2)	0	<i>Ba. forsythus</i>	2 (4)	0
<i>Staph. aureus</i>	10 (20)	2 (4)	Other†	0	1 (2)	<i>Ba. gracilis</i>	2 (4)	0
<i>Staph. epidermidis</i>	9 (18)	10 (18)	<i>Brevibacterium</i> †	3 (6)	2 (4)	<i>Ba. ureolyticus</i>	2 (4)	0
<i>Staph. warneri</i>	3 (6)	6 (11)	<i>Gemella morbillorum</i>	3 (6)	2 (4)	<i>Ba. tectum</i> group E	1 (2)	2 (4)
Other†	3 (6)	0	EF-4a	3 (6)	0	<i>Ba. fragilis</i>	1 (2)	1 (2)
<i>Staph. intermedius</i>	1 (2)	1 (2)	<i>Escherichia coli</i>	3 (6)	0	<i>Ba. ovatus</i>	1 (2)	0
<i>Staph. hominis</i>	1 (2)	1 (2)	<i>Weeksella</i>	2 (4)	4 (7)	<i>Porphyromonas</i>	14 (28)	17 (30)
<i>Staph. auricularis</i>	1 (2)	0	<i>W. virus</i>	0	1 (2)	<i>Porph. nasacae</i>	3 (6)	4 (7)
<i>Staph. colni</i>	1 (2)	0	<i>W. zoobeleum</i>	2 (4)	4 (7)	<i>Porph. cansulci</i>	3 (6)	1 (2)
<i>Staph. xylosum</i>	1 (2)	0	<i>Klebsiella</i>	2 (4)	1 (2)	<i>Porph. gingivalis</i>	2 (4)	6 (11)
<i>Staph. sciuri-lentus</i>	0	2 (4)	<i>K. oxytoca</i>	1 (2)	1 (2)	<i>Porph. canoris</i>	2 (4)	5 (9)
<i>Staph. capitis</i>	0	1 (2)	<i>K. pneumoniae</i>	1 (2)	0	<i>Porph. gingivalis</i>	2 (4)	2 (4)
<i>Staph. haemolyticus</i>	0	1 (2)	<i>Lactobacillus</i>	2 (4)	1 (2)	Other†	2 (4)	0
<i>Staph. lycus</i>	0	1 (2)	<i>L. laetis</i>	1 (2)	0	<i>Porph. circumdentaria</i>	1 (2)	3 (5)
<i>Staph. saprophyticus</i>	0	1 (2)	Other†	1 (2)	1 (2)	<i>Porph. levii-like</i>	1 (2)	0
<i>Staph. simulans</i>	0	1 (2)	<i>Citrobacter</i>	2 (4)	0	<i>Prevotella</i>	14 (28)	11 (19)
<i>Neisseria</i>	8 (16)	11 (19)	<i>Citra. amalonaticus</i>	1 (2)	0	<i>Prev. heparinolytica</i>	7 (14)	5 (9)
<i>N. waerenii</i>	7 (14)	8 (14)	<i>Citra. koseri</i>	1 (2)	0	<i>Prev. intermedia</i>	4 (8)	0
<i>N. subflava</i>	1 (2)	1 (2)	<i>Flavobacterium</i>	2 (4)	0	Other†	1 (2)	4 (7)
Other†	1 (2)	0	Group IIa	1 (2)	0	<i>Prev. zooglyphiformans</i>	2 (4)	1 (2)
<i>N. cinerea-flavescens</i>	0	1 (2)	<i>Flavo. brevis</i>	1 (2)	0	<i>Prev. melaninogenica</i>	1 (2)	1 (2)
<i>N. mucosa</i>	0	1 (2)	<i>Micrococcus</i>	2 (4)	0	<i>Prev. denticola</i>	1 (2)	0
<i>Corynebacterium</i>	6 (12)	16 (28)	<i>Micro. lylae</i>	1 (2)	0	<i>Propionibacterium</i>	10 (20)	10 (18)
Group G	3 (6)	3 (5)	Other†	1 (2)	0	<i>Prop. acnes</i>	7 (14)	9 (16)
<i>Coryne. minutissimum</i>	2 (4)	4 (7)	<i>Proteus mirabilis</i>	2 (4)	0	<i>Prop. acidipropionicus</i>	1 (2)	0
<i>Coryne. aquaticum</i>	1 (2)	8 (14)	<i>Stenotrophomonas maltophilia</i>	2 (4)	0	<i>Prop. freudenreichii</i>	1 (2)	0
<i>Coryne. jeikeium</i>	1 (2)	1 (2)	<i>Capnocytophaga</i>	1 (2)	4 (7)	Other†	1 (2)	0
<i>Coryne. afermentans</i>	1 (2)	0	<i>Cap. ochracea</i>	1 (2)	2 (4)	<i>Prop. avidum</i>	0	1 (2)
Group E	1 (2)	0	Other†	0	3 (5)	<i>Prop. lymphophilum</i>	0	1 (2)
<i>Coryne. pseudodiphtheriticum</i>	1 (2)	0	<i>Eikenella corrodens</i>	1 (2)	1 (2)	<i>Peptostreptococcus</i>	8 (16)	3 (5)
Other†	1 (2)	0	<i>Flavimonas oryzae bitans</i>	1 (2)	1 (2)	<i>Pept. anaerobius</i>	4 (8)	3 (5)
Group B	0	1 (2)	<i>Dermabacter hominis</i>	1 (2)	0	Other†	3 (6)	0
Group F-1	0	1 (2)	<i>Oerskovia</i> †	1 (2)	0	<i>Pept. asaccharolyticus</i>	1 (2)	0
<i>Coryne. kutscheri</i>	0	1 (2)	<i>Pediococcus damnosus</i>	1 (2)	0	<i>Eubacterium</i> †	2 (4)	1 (2)
<i>Coryne. propinquum</i>	0	1 (2)	<i>Stomatococcus mucilaginosus</i>	1 (2)	0	<i>Lactobacillus jensenii</i>	1 (2)	0
<i>Coryne. striatum</i>	0	1 (2)	<i>Acinetobacter</i>	0	4 (7)	<i>Filifactor villosus</i>	0	3 (5)
			<i>Acine. baumannii</i>	0	2 (4)	<i>Clostridium sordellii</i>	0	1 (2)
			<i>Acine. Iwoffii</i>	0	2 (4)	<i>Veillonella</i> †	0	1 (2)

\*Some patients were infected with more than one type or species of bacteria. The abbreviation ssp. denotes subspecies.

†The isolate could not be identified beyond the genus level.

### Marine animal bites

*Vibrio* spp. infection should be suspected in victims of shark bites. *A. hydrophila* and other gram-negative bacilli, as well as *S. aureus* and anaerobes can also infect shark bites. A mycoplasma has been associated with infections after seal bites. Organisms that have been reported after other marine animal bites are *A. hydrophila*, *B. fragilis*, *Chromobacterium violaceum*, *Clostridium perfringes*, *E. rhusiopathiae*, *E. coli*, *Mycobacterium marinum*, *P. aeruginosa*, *Salmonella enteritidis*, *S. aureus*, streptococci, *V. vulnificus*, *Vibrio carchariae*, *Vibrio damsela*, *Vibrio parahaemolyticus*, and *Peptostreptococcus* spp. (9,14,23).

Table 3 shows pathogens that have been associated with specific animal bites (5,20). Table 4 shows systemic infections that can be transmitted by animal bites.

**Table 3: Pathogens associated with specific animal bites**

Cougar: <i>P. multocida</i>
Coyote: <i>P. multocida</i> , <i>F. tularensis</i>
Gerbil: <i>S. moniliformes</i>
Hamster: <i>Acinetobacter anitratus</i>
Lion: <i>P. multocida</i> , <i>S. aureus</i> , <i>E. coli</i>
Panther: <i>P. multocida</i>
Rooster: <i>Streptococcus bovis</i> , <i>Clostridium tertium</i> , <i>Aspergillus niger</i>
Sheep: <i>Actinobacillus</i> spp.
Tiger: <i>P. multocida</i> , <i>Acinetobacter</i> spp., <i>E. coli</i> , streptococci, staphylococci, diptheroids
Wolf: <i>P. multocida</i>

**Table 4: Systemic infections transmitted by animal or human bites**

Viral: Arbovirus (bat), B herpes virus (macaque), CMV (chimpanzee), hantavirus (rodent), HBV, HCV, HIV, rabies, Venezuelan equine encephalitis (bat)
Bacterial: brucellosis (dog), cat-scratch disease (cat, dog, monkey), leptospirosis (dog, mouse, rat), plague (cat), rat-bite fever (dog, gerbil, mouse, rat, squirrel, weasel), syphilis, tetanus (dog), tularemia (cat, dog, other mammals)
Mycobacterial: <i>M. marinum</i> (dolphin), tuberculosis (human)
Fungal: blastomycosis (dog), sporotrichosis (cat)
Parasitic: trypanosomiasis (bat)

### EVALUATION

In any type of bite, it is important to determine the time that has elapsed since injury. Although fulminant infections can occur in less than 12 hours, most infected bites present after that time. Documentation of the mode of injury is essential. Animals inflict a variety of damage from simple scratches and punctures, to massive soft tissue loss. The majority of animal bite victims have no associated injuries. It is essential to evaluate for signs of infection and injuries to deeper structures such as joints, tendons and neurovascular structures. With larger domestic and wild animals, local fractures are encountered. Distal pulses and neurologic function must be checked. For equivocal cases in which exclusion of injuries to deeper structures cannot be made by physical examination alone, imaging by CT or MRI or open exploration by a trained specialist may be indicated (9).

### CLINICAL PRESENTATION

The clinical features of the infection depend on the character and extent of the wound, the anatomic location of the injury, and the organisms responsible for the infection. Localized infection is characterized by swelling, erythema, and tenderness with or without the formation of an abscess. Systemic signs of infection are not usually present. In a study of 59 pediatric and adult patients hospitalized with animal bites, 40 of them were infected at the time of admission (24). Only one third of the patients with infected bite wounds had fever, only 10% had leukocytosis, and only 23% had an elevated erythrocyte sedimentation rate. In another study that reported 39 children with infected human and animal bites, only 7 patients had fever (25). Examination of bones for point tenderness and an assessment of range of motions of joints adjacent to a bite should be performed. Physical findings of soft tissue, bone, or joint inflammation may occur up to 1 or 2 weeks after the injury. Other uncommon complications that can be seen are sepsis, endocarditis, meningitis, and brain abscess.

## **TREATMENT**

### **Local care**

Aggressive wound management of dog-bite wounds is thought to decrease the infection rate (2). Cleansing of the wound with a few hundred mL of high pressure saline is usually effective. Removal of devitalized tissues is also important to prevent a nidus of infection. A study reported a 30-fold reduction rate in infections after adequate débridement, however follow up was not appropriate (26). A more recent study of 769 patients with initially uninfected dog bites found higher rates of infection in wounds that underwent débridement, this may reflect the fact that these wounds had more extensive crush injuries (27). Concentrated forms of povidone-iodine, hydrogen peroxide, or ethyl alcohol should not be used as irrigating solutions because they can cause tissue damage and toxicity (9). Puncture wounds are difficult to débride. It remains to be determined whether an attempt should be made to irrigate bite puncture wounds. Their small cutaneous openings do not permit the solution to drain out adequately, and attempts at irrigation may in fact result in infiltration. No clinical studies have compared excising or incising bite puncture wounds to enhance irrigation. Suturing of puncture wounds is usually not indicated.

Despite the lack of prospective studies, primary wound closure is not typically performed on bite wounds. In general, bite wounds are left open, reevaluated within 2 to 3 days, and managed by secondary intention or delayed primary closure. Some retrospective studies have suggested that primary closure is safe and appropriate in specific settings. A common practice is to avoid primary closure of puncture wounds, crush injuries, wounds that are more than 24 hours old, wounds over the hands, wrists, feet and joints, wounds in immunocompromised individuals, and wounds inflicted by cats or humans (5). Once the wound has been observed for a few days and there are no signs of infection delayed closure can be attempted. Non-infected facial wounds less than 24 hours old can probably be primarily repaired (9). Facial wounds can be closed with high rates of success, probably due to the high vascularity and absence of dependent edema. A retrospective study of 94 patients with animal bites on the face and head showed low rates of infection. Fifty-three patients who had no signs of infection at the time of the initial evaluation received prophylactic antibiotics with either penicillin or clindamycin, of those, four developed an infection; only two of the fifteen patient with no evidence of infection who did not receive prophylaxis developed an infection (28).

### **Antimicrobial therapy**

For wounds that appear infected at the time of initial assessment, antibiotic therapy should be started after a Gram stain and aerobic and anaerobic cultures have been obtained. There is a lack of controlled studies evaluating the use of different antibiotics in infected bite wounds. Empiric treatment should be directed toward the most common infecting organisms. The choice of antibiotics may also be modified based on the most likely pathogens after a specific animal bite. The MICs of commonly isolated organisms from infected human bite wounds are shown in Table 5 (15).

**Table 5: Antimicrobial susceptibility of common bacterial isolates from infected human bites**

Organism	No. of isolates tested	Penicillin		Amoxicillin-clavulanic acid		Doxycycline		Erythromycin <sup>a</sup>		Ciprofloxacin <sup>a</sup>		Moxifloxacin <sup>a</sup>	
		MIC, µg/mL	S, %	MIC, µg/mL	S, %	MIC, µg/mL	S, %	MIC, µg/mL	S, %	MIC, µg/mL	S, %	MIC, µg/mL	S, %
<i>Streptococcus</i> species	37	≤0.015-1	97.3	≤0.015-1	97.3	≤0.06-16	78.4	≤0.125 to >32	70.2	0.5-4	54.1	≤0.06-0.5	100
<i>Staphylococcus aureus</i> <sup>b</sup>	18	0.08 to >8	16.7	0.125-4	100	0.125-32	94.4	≤0.125 to >32	55.8	≤0.06-0.5	100	≤0.06	100
<i>Eikenella corrodens</i> <sup>a</sup>	18	0.5-2	100 <sup>c</sup>	0.25-0.5	100 <sup>d</sup>	0.5-4	72.2 <sup>e</sup>	2-16	16.7 <sup>f</sup>	≤0.06	100	≤0.06	100 <sup>g</sup>
Miscellaneous fastidious GNB <sup>h,i</sup>	13	≤0.015 to >8	92.3 <sup>a</sup>	≤0.015-8	92.3 <sup>g</sup>	0.125-2	100 <sup>f</sup>	≤0.125-16	69.2 <sup>f</sup>	≤0.06-0.25	100 <sup>g</sup>	≤0.06-1	100 <sup>g</sup>
<i>Prevotella</i> species <sup>b</sup>	13	0.08 to >8	46.2	≤0.015-1	100	≤0.06-4	100	≤0.125-0.5	100 <sup>f</sup>	0.5-2	61.5 <sup>a</sup>	0.25-1	100 <sup>g</sup>
<i>Fusobacterium</i> species	10	≤0.015-0.125	100	≤0.015-0.25	100	≤0.06-0.5	100	0.5-16	40 <sup>f</sup>	0.25-2	40 <sup>a</sup>	0.125-0.25	100 <sup>g</sup>
<i>Veillonella</i> species <sup>b</sup>	11	≤0.015-8	72.7	≤0.015-2	100	0.25-2	100	0.5-8	9.1 <sup>f</sup>	≤0.06-0.125	100 <sup>g</sup>	≤0.06-0.25	100 <sup>g</sup>
<i>Peptostreptococcus</i> species	5	≤0.015-0.5	100	≤0.015-2	100	≤0.06-0.25	100	≤0.125-0.25	100 <sup>f</sup>	≤0.06-1	100 <sup>g</sup>	0.125-0.5	100 <sup>g</sup>

Several studies describing in vitro activities of a variety of agents against aerobic and anaerobic bacteria isolated from bite wounds have been published. A study evaluated the activities of cefuroxime, amoxicillin-clavulanate, ciprofloxacin, and other antibiotics against bite wound isolates (29). Cefuroxime showed good in vitro activity against *S. aureus*, *P. multocida*, *Moraxella* spp., and streptococci. Cefuroxime was at least fourfold more active than cephalexin and cefadroxil against these isolates. Cefuroxime had similar activity to penicillin, amoxicillin and amoxicillin-clavulanate against *P. multocida*. Cefuroxime, cephalexin and cefadroxil showed inferior activity against *E. corrodens*, *P. melaninogenicus*, *Bacteroides* spp., *Fusobacterium* spp., and *Peptostreptococcus* spp., when compared to penicillin, amoxicillin-clavulanate and ampicillin. Tetracyclines showed adequate activity against *S. aureus*, *P. multocida*, *Moraxella* spp., and fusobacteria; the activity against streptococci, *E. corrodens* and *Peptostreptococcus* spp. was not adequate. Ciprofloxacin showed adequate activity against *P. multocida*, *E. corrodens*, and *Moraxella* spp., and poor activity against anaerobic isolates.

A similar study evaluated the activities of azithromycin, moxifloxacin, levofloxacin, sparfloxacin, and other antibiotics against bite wound isolates (30). Sparfloxacin, levofloxacin and moxifloxacin were active against all aerobic isolates. Sparfloxacin and levofloxacin were variably active against anaerobic isolates and had poor activity against fusobacteria. Moxifloxacin was active against most anaerobic isolates with the exception of fusobacteria. Azithromycin was more active than erythromycin against many aerobes including *P. multocida* and *E. corrodens*, and it was 2 to 4 dilutions more active against anaerobic isolates. As in the previous study amoxicillin-clavulanate was active against all the isolates. Trimethoprim-sulfamethoxazole was active against the majority of the aerobic organisms including *P. multocida* and *E. corrodens*, but was inactive against most anaerobic isolates. Clarithromycin has been found to be more active in vitro than erythromycin against many bite wound isolates (31), however, it appears to be less active than azithromycin against *P. multocida*, *E. corrodens* and fusobacteria (30,32).

A study compared the activities of moxifloxacin, ciprofloxacin, sparfloxacin, levofloxacin, ofloxacin, and other antibiotics against 309 aerobic and anaerobic bite wound isolates (33). Moxifloxacin was active against all the aerobic isolates. The MICs for streptococci were slightly higher than the ones seen with other aerobic isolates. Moxifloxacin was active against most anaerobic isolates with the exceptions of *F. nucleatum* and other *Fusobacterium* spp., and one strain of *Prevotella loeschii*. In comparison, the other fluoroquinolones tested had similar in vitro activities against the aerobic strains but were less active against the anaerobes,

including *Peptostreptococcus* spp., *Porphyromonas* spp., and *Prevotella* spp. The fusobacteria were resistant to all the antibiotics tested in this study except penicillin and amoxicillin-clavulanate. Gatifloxacin showed similar activity in a study of 308 aerobic and 11 anaerobic isolates from bite wounds (34). Gatifloxacin was active at low MICs against all aerobic bite wound isolates, with the exception of some streptococci (MICs slightly higher but still susceptible). Gatifloxacin was active against *Bacteroides tectum*, *P. heparynolytica*, other *Prevotella* spp, *Peptostreptococcus* spp. and *Porphyromonas* spp. Fusobacteria were sometimes resistant to gatifloxacin as well as the other fluoroquinolones.

The in vitro activity of the carbapenems against bite wound isolates has been reported in a few studies. Ertapenem was tested against 240 aerobic and 180 anaerobic bite wound isolates (35). Ertapenem showed excellent potency against the full range of animal and human bite wounds pathogens, including fusobacteria. Meropenem and imipenem have also shown excellent in vitro activity against similar isolates. The MICs against *Enterococcus* spp. seem to be higher for ertapenem when compared to imipenem and meropenem (36). Linezolid has been tested against 420 aerobes and anaerobes (37). Linezolid was active against all *P. multocida* subspecies *multocida*, and *P. multocida* subspecies *septica* isolates, and most *P. canis*, *Pasteurella dagmatis*, and *Pasteurella stomatis* isolates. Linezolid was also active against fusobacteria, *Porphyromonas* spp., *Prevotella* spp., *Peptostreptococcus* spp., and almost all *B. tectum* isolates. Linezolid did not show adequate activity against *Neisseria* spp., *Moraxella* spp., and *E. corrodens*.

Telithromycin, a ketolide drug, was tested against 268 aerobic and 148 anaerobic bite wound isolates and compared with available macrolides and other antibiotics (32). Telithromycin was found to have adequate activity against almost all aerobic and anaerobic organisms with the exception of fusobacteria. Telithromycin was found to be more active than azithromycin or clarithromycin against bite wound isolates. Tigecycline, a new semisynthetic glycylcycline, was tested against bite wound pathogens (38). It showed excellent activity against aerobic organisms, including tetracycline-resistant streptococci, *Enterococcus* spp, and coagulase-negative *Staphylococcus*, with the exception of *E. corrodens*. Tigecycline was also very active against all anaerobic species, including tetracycline-, doxycycline-, and minocycline-resistant strains of *Prevotella* spp., *Porphyromonas* spp., *B. tectum*, and *Peptostreptococcus* spp. Erythromycin- and moxifloxacin-resistant fusobacteria were also susceptible to tigecycline.

Few antibiotics offer optimal coverage for all the potential pathogens. A  $\beta$ -lactam antibiotic combined with a  $\beta$ -lactamase inhibitor offers excellent coverage for the majority of pathogens. There is ample clinical experience with the use of these agents. The combination of penicillin and an anti-staphylococcal agent or penicillin and clindamycin have also been recommended. Cefoxitin, cefotetan, the carbapenems, or the combination of cefuroxime with either clindamycin or metronidazole also provide adequate coverage, however, there are no clinical data to support their use. Other options to consider in patients with intolerance to  $\beta$ -lactam agents based on the in vitro activity against bite wound isolates include the combination of trimethoprim-sulfamethoxazole or an older fluoroquinolone with clindamycin or metronidazole, azithromycin, one of the newer fluoroquinolones (gatifloxacin, moxifloxacin), telithromycin and tigecycline (not FDA approved yet). Tetracyclines can also be used, however, their activity against streptococci, some anaerobes, and *E. corrodens* are suboptimal. Table 6 summarizes activities of different antibiotics against bacteria frequently isolated from animal and human bites.

## NOTES ON SOME FAMOUS ORGANISMS

Characteristically, *Pasteurella* infections become symptomatic during the first 24 hours after the injury, symptoms include erythema, swelling, tenderness and drainage at the site of the bite. Infections due to *Pasteurella* most often remain localized to the inoculated wound with cellulitis and abscesses, but direct extension to surrounding tissues may occur, leading to lymphangitis and regional lymphadenopathy, and bacteremia may occur with metastatic foci including osteomyelitis, arthritis, tenosynovitis, sepsis, meningitis, brain abscess, pneumonia and endocarditis (17). *Pasteurella* spp. can also produce septic arthritis and

osteomyelitis (17,39). *P. multocida* bacteremia is associated with cirrhosis, diabetes, rheumatoid arthritis, hematologic malignancies, neoplasms and immunosuppression (6,17,40). Wounds that appear infected more than 24 hours after an injury are more likely to be infected with staphylococci and streptococci among other oral flora. Penicillin is the drug of choice for *P. multocida* infections. Ampicillin and amoxicillin-clavulanate also offer adequate activity. Cefuroxime and cefpodoxime have better in vitro activity against *Pasteurella* spp. than other cephalosporins. Nafcillin, dicloxacillin, cefazolin, cefaclor, cefadroxil, and cephalexin have inadequate activity against most strains of *Pasteurella* spp. Clindamycin and the aminoglycosides have poor activity against *Pasteurella* spp. (41).

**Table 6: Antimicrobial susceptibilities of bacteria frequently isolated from bite wounds**

Agent	Staphylococcus aureus	Streptococci	Pasteurella multocida	Fusobacterium spp.	Bacteroides spp.	Prevotella spp.	Porphyromonas spp.	Eikenella corrodens
Penicillin	-	+	+	+/-	+/-	+/-	+	+
Ampicillin	-	+	+	+/-	+/-	+/-	+	+
Amoxicillin/clavulanate	+	+	+	+	+	+	+	+
Dicloxacillin	+	+/-	-	-	-	-	-	-
Cephalexin	+	+	-	-	-	-	-	-
Cefoxitin	+	+	+	+	+	+	+	+
Cefuroxime	+	+	+	-	+/-	+/-	+	+/-
Ceftriaxone	+/-	+	+	+	-	-	+	+
Cefepime	+/-	+	+	+/-	-	-	+	+
Imipenem	+	+	+	+	+	+	+	+
Piperacillin/tazobactam	+	+	+	+	+	+	+	+
Tetracycline	+/-	-	+	+	+/-	+/-	+/-	-
Erythromycin	+/-	+	-	-	+/-	+/-	+	-
Azithromycin	+/-	+	+/-	+/-	+/-	+/-	+	+
Clarithromycin	+/-	+	+/-	+/-	+/-	+/-	+	+
Clindamycin	+	+	-	+/-	+	+	+	-
TMP/SMX	+	-	+	-	-	-	-	+
Ciprofloxacin	+/-	+/-	+	-	+/-	+/-	+/-	+
Levofloxacin	+/-	+	+	-	+/-	+	+	+
Moxifloxacin	+/-	+	+	-	+/-	+	+	+
Gatifloxacin	+/-	+	+	-	+	+	+	+
Metronidazole	-	-	-	+	+	+	+	-
Vancomycin	+	+	-	-	-	-	-	-
Telithromycin	+	+	+	-	+	+	+	+
Tigecycline	+	+	+	+	+	+	+	-
Linezolid	+	+	+/-	+	+/-	+/-	+/-	-

*Capnocytophaga canimorsus* (formerly known as dysgonic fermentor-2 or DF-2) is a fastidious, slow growing, spindle-shaped, gram-negative rod. The cellular morphology is similar to that of *Fusobacterium* spp. Predisposing factors for severe *C. canimorsus* infection are splenectomy, alcoholism, liver disease, corticosteroid therapy, and chronic lung diseases. Severe infections have also been reported in patients with leukemia, lymphoma, and in association with steroid therapy (5,6,42). Complications include septic arthritis, meningitis, endocarditis, renal failure, disseminated intravascular coagulation, and sepsis. Symptoms ensue 1 day to 2 weeks after the bite. *C. canimorsus* has been reported extensively after dog bites, a few case reports of infection after cat bites have also been reported (42). The overall mortality from *C. canimorsus* septicemia exceeds 20%. The organism grows slowly in blood cultures (mean 6 days), a rapid interim diagnosis can be made by Gram staining the buffy-coat preparations (9). Penicillin and amoxicillin are the agents of choice. This organism is also susceptible to clindamycin, cephalosporins, fluoroquinolones, imipenem, macrolides, chloramfenicol, and tetracyclines (33,42). Aminoglycosides, aztreonam, metronidazole, and trimethoprim are not effective (43). Routine administration of antibiotics with activity against *C. canimorsus* is recommended in immunocompromised patients (especially patients with alcoholism or who have undergone splenectomy) after dog bites (43).

*E. corrodens* is a facultative anaerobic gram-negative bacillus that may result in serious, chronic infections, and generally requires more than a week to produce disease. As previously stated it is a common isolate after human bites, particularly in clenched-fist injuries. *E. corrodens* is typically susceptible to penicillin, amoxicillin/clavulanate, and second generation cephalosporins. It is typically resistant to first generation cephalosporins, semi-synthetic penicillins, clindamycin, erythromycin, metronidazole and aminoglycosides.

Rat-bite fever is a syndrome that is caused by two different organisms (5). *S. moniliformis* (Haverhill fever) is a gram-negative bacillus that may cause infection within 12 hours to 10 days after a bite. The bite may be associated with local inflammation, however, healing of the wound often occurs before the onset of the systemic symptoms. Irregularly spiking fevers with chills may occur for up to 3 weeks after the onset of symptoms. An associated morbilliform or petechial rash that can affect palms and soles occurs in 75% to 95% of the patients. Arthralgia and arthritis can also be seen. Endocarditis, myocarditis, pericarditis, and hepatitis have been reported as complications. If the infection is not treated relapsing fever ensues over several months. Rat-bite fever due to *S. minor* (sodoku) has an incubation period of 7 to 21 days, with clinical disease that is usually milder than the one caused by *S. moniliformis*. Some ulceration or eschar in the bite site is usually present when systemic symptoms develop. A maculopapular rash occurs in 75% of the patients, arthritis is uncommon, as are endocarditis and nephritis. An examination of a blood smear for spirochetes may help identifying this organism. This form of rat-bite fever is uncommon in the United States. Rat-bite fever caused by *S. moniliformis* and *S. minor* can occur together. Intravenous penicillin G is the agent of choice for both of the organisms responsible for rat-bite fever. For uncomplicated rat-bite fever, oral penicillin or tetracycline may be effective therapy. Other antibiotics that show in vitro activity against *S. moniliformis* include cephalosporins and vancomycin. Erythromycin has variable activity against *S. moniliformis*.

B herpes virus (Herpesvirus simiae) is enzootic in North African and Asian monkeys and is seen in macaque and rhesus monkeys. The infection can be transmitted in captivity to other monkey species. Infection can be transmitted by bites. The virus may produce a vesicular rash at the site of the bite. However, a high rate of associated complications occurs with ascending paresthesias, and subsequent development of fever and encephalitis. The mortality in humans is 70% and prophylaxis with acyclovir should be considered after discussion with an expert virologist (B virus Resource Center: 404-651-0808) (9).

A syndrome of pain, erythema and massive edema in the hand 4 to 8 days after contact through a skin laceration with the skin of a seal or with a seal tooth or claw has been reported (5). The organism appears to be a mycoplasma. It can also produce lymphangitis, lymphadenopathy and arthritis. Failure to initiate appropriate treatment with tetracycline may result in permanent damage.

## **PROPHYLAXIS**

The use of antibiotic prophylaxis after cat, dog or human bites is controversial. Well-designed studies evaluating the use of antibiotic prophylaxis in bite wounds of similar severity and anatomical location have not been performed. Studies have suggested that wound management and not antibiotic prophylaxis is the most important factor in preventing infection. The majority of studies have been small and have not achieved statistical significance (44). One single prospective, double-blind study, compared the use of amoxicillin-clavulanate and placebo for the prevention of infection after animal bites (45). In wounds less than 9 hours there was no difference in the rates of infection. In older wounds the infection rate was reduced significantly ( $p=0.023$ ). There is no evidence that prophylactic antibiotics prevent infections in human bites elsewhere than the hand (10). A meta-analysis of randomized trials found that prophylactic antibiotics reduced the rate of infection in dog bite wounds, with a RR of 0.56 (46). A Cochrane review found significant heterogeneity between trials. The review found no significant difference in infection rate with antibiotics compared with placebo after dog, cat, or human bites. When the results were analyzed for each wound site, antibiotics significantly reduced infection of the hand only. The review concluded that “there is evidence from one trial

that prophylactic antibiotics reduces the risk of infection after human bites but confirmatory research is needed, there is no evidence that the use of prophylactic antibiotics is effective for dog or cat bites, and there is evidence that the use of prophylactic antibiotics after bites of the hand reduces infection but confirmatory research is needed” (47). A common approach after dog, cat or human bites is to offer prophylactic antibiotics in the circumstances shown in Table 7 (5), however, other authors have been less aggressive, recommending withholding antibiotic prophylaxis when the wound is of minor severity and the patient is not at high risk for infection, or when the patient presents after 24 hours following injury without signs of infection (6). The duration of prophylaxis should be in the order of 3-5 days. The need for prophylactic antibiotics after less common animal bites is also controversial. A similar approach to the one used after human bites is usually followed after monkey bites. A study of 363 snakebite patients suggested that antibiotics should be given only if necrosis is present (48).

**Table 7: Circumstances where the use of prophylactic antibiotics may be indicated**

Dog bites more than 8-12 hours old
Moderate to severe dog bites less than 8-12 hours (edema, crush injuries)
Puncture wounds, particularly if bone or joints were penetrated
Severe facial wounds
All hand bites
Wounds in the genital area
Wounds in immunocompromised or asplenic patients
Moderate to severe cat or human bites

## REFERENCES

1. Goldstein EJC. Bite wounds and infection. *Clin Infect Dis* 1992;14:633-640
2. Bradley JS. Bite-wound infections. In: Jenson JB, Baltimore RS (eds): *Pediatric Infectious Diseases, Principles and Practice*, second edition, pp 602.
3. McDonough JJ, Stern PJ, Alexander JW. Management of animal and human bites and resulting human infections. *Curr Clin Top Infect Dis* 1987;8:11-36
4. Weiss HB, Friedman DI, Coben JH. Incidence of dog bite injuries treated in emergency departments. *JAMA* 1998;279:51-53
5. Edwards MS. Animal bites. In: Feigin RD, Cherry JD, Demmler GJ, Kaplan SL (eds): *Textbook of Pediatric Infectious Diseases*, fifth edition. Saunders, 2004, pp3267
6. Smith PF, Meadowcroft AM, May DB. Treating mammalian bite wounds. *J Clin Pharm Ther* 2000;25:85-99
7. Goldstein EJC, Citron DM, Finegold SM. Dog bite wounds and infection: a prospective clinical study. *Ann Emerg Med* 1980;9:508-512
8. Lindsey D, Christopher M, Hollenbach J, Boyd JH, Lindsey WE. Natural course of the human bite wound: incidence of infection and complications in 434 bites and 803 lacerations in the same group of patients. *J Trauma* 1987;27:45-48
9. Abrahamian FM, Goldstein EJC. Bites. In: Gorbach SL, bartlett JG, Blacklow NR (eds): *Infectious Diseases*, third edition. Lippincott, Williams and Wilkins, 2004, pp1440
10. Schweich P, Fleisher G. Human bites in children. *Pediatr Emerg Care* 1985;1:51-53
11. Earley MJ, Bardsley AF. *Br J Plast Surg* 1984;37:458-462
12. Talan DA, Citron DM, Abrahamian FM, Moran GJ, Goldstein EJC. Bacteriologic analysis of infected dog and cat bites. *N Engl J Med* 1999;340:85-92
13. Wienert P, Heib J, Rinecker H, Sing A. A human bite. *Lancet* 1999;354:572
14. Brook I. Microbiology and management of human and animal bite wound infections. *Prim Care Clin Office Pract* 2003;30:25-39

15. Talan DA, Abrahamian FM, Moran GJ, Citron DM, Tan JO, Goldstein EJC. Clinical presentation and bacteriologic analysis of infected human bites in patients presenting to emergency departments. *Clin Infect Dis* 2003;37:1481-1489
16. Schmidt DR, Heckman JD. *Eikenella corrodens* in human bite infections of the hand. *J Trauma* 1983;23:478-482
17. Weber DJ, Wolfson JS, Swartz MN, Hooper DC. *Pasteurella multocida* infections: report of 34 cases and review of the literature. *Medicine* 1984;63:133-151
18. Goldstein EJC, Ellsworth PP III, Citron DM. Simian bites and bacterial infection. *Clin Infect Dis* 1995;20:1551-1552
19. Hsieh S, Babl FE. *Serratia marcescens* cellulitis following an iguana bite. *Clin Infect Dis* 1999;28:1181-1182
20. Greenberg SB. Serious waterborne and wilderness infections. *Crit Care Clin* 1999;15:387-414
21. Goldstein EJC. Selected nonsurgical anaerobic infections: therapeutic choices and the effective armamentarium. *Clin Infect Dis* 1994;18(Suppl 4):S273-279
22. Van Demark RE, Van Demark RE Jr. Swine bites of the hand. *J Hand Surg* 1991;16:136-138
23. Murphey DK, Septimus EJ, Waagner DC. Catfish-related injury and infection: report of two cases and review of the literature. *Clin Infect Dis* 1992;14:689-693
24. Feder HM, Shanley JD, Barbera JA. Review of 59 patients hospitalized with animal bites. *Pediatr Infect Dis J* 1987;6:24-28
25. Brook I. Microbiology of human and animal bite wounds in children. *Pediatr Infect Dis J* 1987;6:29-32
26. Callaham M. Prophylactic antibiotics in common dog bite wounds: a controlled study. *Ann Emerg Med* 1980;9:410-414
27. Dire DJ, Hogan DE, Riggs MW. A prospective evaluation of risk factors for infections from dog-bite wounds. *Acad Emerg Med* 1994;1:258-266
28. Wolff KD. Management of animal bite injuries of the face: experience with 94 patients. *J Oral Maxillofac Surg* 1998;56:838-843
29. Goldstein EJC, 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;32:1143-1148
30. Goldstein EJC, Nesbit CA, Citron DM. Comparative in vitro activities of azithromycin, bay y 3118, levofloxacin, sparfloxacin, and 11 other oral antimicrobial agents against 194 aerobic and anaerobic bite wound isolates. *Antimicrob Agents Chemother* 1995;39:1097-1100
31. Goldstein EJC, Citron DM. Comparative susceptibilities of 173 aerobic and anaerobic bite wound isolates to sparfloxacin, temafloxacin, clarithromycin and older agents. *Antimicrob Agents Chemother* 1993;37:1150-1153
32. Goldstein EJC, Citron DM, Hunt Gerardo S, Hudspeth M, Merriam CV. Activities of HMR 3004 (RU 64004) and HMR 3647 (RU 66647) compared to those of erythromycin, azithromycin, clarithromycin, roxithromycin, and eight other antimicrobial agents against unusual aerobic and anaerobic human and animal bite pathogens isolated from skin and soft tissue infections in humans. *Antimicrob Agents Chemother* 1998;42:1127-1132
33. Goldstein EJC, Citron DM, Hudspeth M, Hunt Gerardo S, Merriam CV. In vitro activity of Bay 12-8039, a new 8-methoxyquinolone, compared to the activities of 11 other oral antimicrobial agents against 390 aerobic and anaerobic bacteria isolated from human and animal bite wound skin and soft tissue infections in humans. *Antimicrob Agents Chemother* 1997;41:1552-1557
34. Goldstein EJC, 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 wounds infections. *Antimicrob Agents Chemother* 1999;43:1475-1479
35. Goldstein EJC, Citron DM, Merriam CV, Warren YA, Tyrrell K, Fernandez H. Comparative in vitro activity of ertapenem and 11 other antimicrobial agents against aerobic and anaerobic pathogens isolated from skin and soft tissue animal and human bite wound infections. *J Antimicrob Chemother* 2001;48:641-651

36. Goldstein EJC, Citron DM, Merriam CV, Warren YA, Tyrrell KL, Fernandez HT. Comparative in vitro activity of faropenem and 11 other antimicrobial agents against 405 aerobic and anaerobic pathogens isolated from skin and soft tissue infections from animal and human bites. *J Antimicrob Chemother* 2002;50:411-420
37. Goldstein EJC, Citron DM, Merriam CV. Linezolid activity compared to those of selected macrolides and other agents against aerobic and anaerobic pathogens isolated from soft tissue bite infections in humans. *Antimicrob Agents Chemother* 1999;43:1469-1474
38. Goldstein EJC, Citron DM, Merriam CV, Warren Y, Tyrrell K. Comparative in vitro activities of GAR-936 against aerobic and anaerobic animal and human bite wound pathogens. *Antimicrob Agents Chemother* 2000;44:2747-2751
39. Ewing R, Fainstein V, Musher DM, Lidsky M, Clarridge J. Articular and skeletal infections caused by *Pasteurella multocida*. *South Med J* 1980;73:1349-1352
40. Morris MJT, McAllister K. Bacteremia due to *Pasteurella multocida*. *South Med J* 1992;85:442-443
41. Goldstein EJC, Citron DM, Richwald GA. Lack of in vitro efficacy of oral forms of certain cephalosporins, erythromycin, and oxacillin against *Pasteurella multocida*. *Antimicrob Agents Chemother* 1988;32:213-215
42. Valtonen M, Lauhio A, Carlson P, Multanen J, Sivonen A, Vaara M, Lahdevirta J. *Capnocytophaga canimorsus* septicemia: fifth report of a cat-associated infection and five other cases. *Eur J Clin Microbiol Infect Dis* 1995;14:520-523
43. Le Moal G, Landron C, Grollier G, Robert R, Burucoa C. Meningitis due to *Capnocytophaga canimorsus* after receipt of a dog bite: case report and review of the literature. *Clin Infect Dis* 2003;36:e42-e46
44. Callahan M. Controversies in antibiotic choices for bite wounds. *Ann Emerg Med* 1988;17:1321-1330
45. Brakenbury PH, Muwanga C. A comparative double blind study of amoxicillin/clavulanate vs placebo in the prevention of infection after animal bites. *Arch Emerg Med* 1989;6:251-256
46. Cummings P. Antibiotics to prevent infection in patients with dog-bite wounds: a meta-analysis of randomized trials. *Ann Emerg Med* 1994;23:535-540
47. Medeiros I, Saconato H. Antibiotic prophylaxis for mammalian bites. *Cochrane Database Syst Rev* 2001;2:CD001738
48. Blaylock RS. Antibiotic use and infection in snakebite victims. *S Afr Med J* 1999;89:874-876