Journal of Hospital Infection (2005) **61**, 1–10

Available online at www.sciencedirect.com

SCIENCE DIRECT®

www.elsevierhealth.com/journals/jhin

REVIEW

Hospital management of animal and human bites

M. Morgan*

Department of Medical Microbiology, Royal Devon and Exeter Foundation Trust, Church Lane Heavitree, Exeter EX2 5AD, UK

Available online 5 July 2005

KEYWORDS
Animal bites;
Infections

Summary The management of bites severe enough to warrant hospital admission is complex. This review includes the epidemiology, clinical management, investigations, microbiology and role of antimicrobials for all types of animal and human bites likely to be encountered in UK hospitals.

Epidemiology

Few of the 200 000 patients with animal bites attending accident and emergency departments in the UK annually[1] need admission. Those needing inpatient treatment usually follow severe trauma, or have high-risk wounds needing debridement or infected wounds due to incorrect management in primary care.

Dogs are responsible for 80–90% of animal bites,[2,3] especially in children. The majority of attacks are unprovoked and one-third involve the family dog.[4] Of the 17% of children[5] bitten badly enough to warrant medical attention, up to 80%[6] present with facial and cervical injuries. Adults tend to be bitten on the extremities, particularly hands. Overall, due to the comparatively superficial nature of the injuries, which are easily cleaned, only 20% of dog bites become infected. However, the infection rate increases to 36% when the hand is involved.[7]

Comparatively more cat bite victims seek medical attention. The higher rate of infection, up to 80% of hand bites by cats,[6] is largely due to the characteristically deep puncture wounds. These are difficult to clean and prone to infection with anaerobes and pasteurella,[7] the latter up to 10 times more common with cat bites than dog bites.[8]

Human bites are the third most common bites seen, with an overall infection rate of 18%. Although small children often bite their playmates,[9,10] human bites in childhood are less prone to bacterial infection than adult bites.[11]

Bites by more unusual animal pets are increasingly common in UK accident and emergency departments. In Exeter, during 2003, we treated pig, rabbit, snake and rat bites, together with single cases of alpaca, piranha and iguana bites.

Management of bites presenting within 24 h

Indications for hospital admission include systemic manifestations of infection, involvement of joints or tendons, immunocompromised patient, significant hand bites or bites requiring reconstructive surgery,

* Tel.: +44 1392 402970; fax: +44 1392 412835.
E-mail address: marina.morgan@rdehc-tr.swest.nhs.uk

0195-6701/$ - see front matter
doi:10.1016/j.jhin.2005.02.007

severe cellulitis and infection refractory to oral therapy.[2] All human bites with puncture wounds[12] and all bites potentially involving a joint should be admitted for surgical exploration debridement and joint irrigation.

The management of animal bites can be categorized into several stages.

Clinical management

Resuscitation.
Full history including whether the attack was provoked, animal species, time since injury, time since bite, antibiotic allergies.
Animal bites: rabies and tetanus prophylaxis if indicated.
Human bites: assess need for antiviral prophylaxis (see below).

Assessment of the need for antibiotics

Previous history—underlying immunocompromise, e.g. splenectomy.
Physical examination with assessment of infection risk such as depth of wound, degree of crush injury and devitalized tissue, lymphadenopathy, nerve damage, damage to tendons, bones and joints, and examination of the wound, under anaesthetic if necessary.

Other assessments

Forensic investigation of human and animal bite victims demands careful photography of wounds.
DNA profiling and matching of dental impressions to bite marks can be done for prosecution of human aggressors and the owners of animals that bite victims.
Systemic and local evaluation, temperature, C-reactive protein, erythrocyte sedimentation rate.
X-rays to exclude fractures and embedded teeth.

Wound management

Thorough irrigation and debridement of the wound.
Facial bites can be closed primarily whatever the species involved, since bleeding is profuse and wounds are easily cleaned.
Delayed closure where possible if limb bite.
Elevation and immobilization of limb.

Established wound infection

Infected, sutured wounds should be opened and drained.
Fluctuant wounds should be incised and drained.
Swabs should be taken from deep within the wound and cultured as soon as possible.
Persistent adenitis following a cat scratch or bite should not be drained; instead, serology for cat scratch disease (*Bartonella henselae*) should be performed.[13]

Microbiological

Wounds should be cultured if clinically infected.
Early bite wound cultures are rarely useful.
Antibiotics when indicated, depending on risk factors for infection, including:
- patient factors, alcoholism or cirrhosis, immunocompromise such as asplenic; and
- wound factors, e.g. over 6-h-old, devitalized, sutured wounds, full-thickness wounds involving tendons, ligaments and joints, wounds to limbs especially hands.

Resuscitation

Exsanguination following carotid trauma is the major cause of death in children less than 10 years of age following dog attacks. Fatalities are particularly associated with attacks by pit bull terriers, Rottweilers and German Shepherds,[4] although even Jack Russells cause significant injuries in small children.[14,15]

Not surprisingly, given their jaw strength and tenacity, pit bull terriers inflict the most serious bites. In addition to a biting force of up to 450 pounds per square inch (enough to crush sheet steel),[16] pit bulls grind their molars into soft tissue, causing particularly large wounds and significant devitalization. The canine tooth puncture wound anchors the victim, while the other teeth bite, shear and tear the tissues, causing stretch lacerations. Such injuries, together with claw marks, are diagnostic of a dog attack.

General wound management

Removal of foreign bodies (teeth) should be followed by proper cleansing using normal saline, 1% povidone iodine or tap water at body temperature and a cloth gauze to remove excess dirt. Even the simplest wounds need copious irrigation and wound toilet. Infection following animal bites is usually apparent within 12–48 h, with redness, swelling and serosanguinous or purulent drainage. *Pasteurella multocida* infections are particularly

aggressive, usually presenting within 12 h[17] unless partially treated.

Laboratory tests

Blood cultures should be taken if there is systemic toxicity or immunosuppression. Septic shock following animal bites in asplenic or cirrhotic patients, with a clinical picture reminiscent of meningococcal septicaemia with purpura fulminans, malar purpura, is a classical presentation of *Capnocytophaga canimorsus* (Dysgonic Fermenter type 2/DF2) infection.[18,19] Mortality is 40%.[20] The diagnosis is confirmed easily by examining a peripheral blood film (preferably a buffy coat) for organisms. Slender, intracellular Gram-negative tapering rods typical of *C. canimorsus* were found in peripheral blood films in 12 of 13 cases.[18] Therapy with penicillin, cephalosporins or ciprofloxacin has been recommended, although one case of penicillin-resistant DF2 has been reported.[21] *C. canimorsus* is fastidious and always resistant to gentamicin.

Tetanus prophylaxis

Tetanus following human bites is extraordinarily rare,[22,23] and prophylaxis is largely unnecessary with human and UK domestic animal bites. However, it is recommended that immunoglobulin and tetanus toxoid should be administered to patients with a history of two or fewer immunizations.

Antiviral prophylaxis

Thorough wound toilet, irrigation and debridement, where necessary, is mandatory for all bite wounds, and irrigation with virucidal povidone iodine is advisable where possible.

Travellers bitten abroad by almost any animal need assessment for rabies prophylaxis.[24] Even bites by British bats warrant rabies prophylaxis.[25–27] Despite reports of transmission through sexual bites,[28] there are no current recommendations for specific antiviral prophylaxis for herpes 1 and 2 virus infection. Hepatitis B immunoglobulin is indicated when the assailant is known to be hepatitis B sAg or eAg positive. Rapid hepatitis B vaccination is a sensible precaution for all victims when the assailant's status is unknown.

Hepatitis C has been transmitted via bites.[29] During one incident, hepatitis C but not human immunodeficiency virus (HIV) was transmitted by a dually infected patient.[30] Despite the very low transmission rate, HIV postexposure prophylaxis may be considered when the attacker is thought to be at high risk of infection, and the victim presents early enough. Human bites should be managed as inoculation injuries, and victim and donor should be counselled accordingly.

Although two seroconversions occurred among the five victims of a biting toddler,[31] biting is an inefficient way of transmitting HIV.[32] None of eight healthcare workers bitten by an HIV-positive patient seroconverted.[33] In the rare instances where transmission has occurred, significantly bloody saliva was involved.[34–36]

When counselling victims of human bites, it can be useful to roughly compare the relative risk of transmission of blood-borne viruses to reassure the patient. Hence, with the highest risk of transmission during true inoculation injury of hepatitis C being 3% and HIV 0.3%, the risk of bite wounds transmitting HIV infection is at least 20 times less.[37,38]

Radiological examination of bite wounds

Accurate assessment is difficult when the superficial wound appears trivial, although broken teeth may have penetrated a joint or produced deep tissue trauma and devitalization. X-rays must be obtained for all clenched fist injuries and for penetrating scalp wounds in children. Early radiological examination is necessary to exclude the presence of teeth or dental fragments and bony damage. With human bites, lateral X-rays of the hand are important to delineate swelling or lacerations of the soft tissues, obscured on standard frontal views by bone.

Bites to the head and neck

Seventy-six percent of bites to the face in children affect lips, nose or cheeks.[6] Assessment of facial bites begins with a complete physical examination and intra-oral examination to exclude cheek lacerations with an intra-oral communication.

Children with facial or cranial bites need cervical immobilization until cervical lesions are excluded. Careful inspection and appropriate imaging is necessary. A small scalp puncture wound may indicate anchoring of teeth to the cranium during shaking, overlying intracranial injury and facial fractures.

Penetrating wounds of the neck and thoracic

inlet are especially dangerous, and early angiography and exploration may be necessary. Only 10% of dog bites to adults involve the head and neck, and infections in this region are rare, reported in only 1.4% of cases.[39] Primary closure of head and neck wounds with antibiotic prophylaxis is associated with a very low risk of infection (1%).[40]

Bites to the extremities and hand

Avulsed ears, noses or other body parts should be kept cool pending plastic surgery for re-attachment. Infections are extremely rare, since bleeding is usually profuse.

Established bite-related infection in the hand often results in permanent impairment of function, hence management should be extremely aggressive and documented thoroughly. Failure to take into account the high risk of polymicrobial infection and inadequate initial surgical debridement are the major factors associated with poor outcome.

Hands are especially prone to infection because of the numerous small compartments and absence of significant soft tissues separating the skin from bone and joints.[17] With some 42 species of bacteria present in human saliva,[41] joint space infections resulting from human bites, particularly 'fight bites', are rapidly destructive. Up to 25% of human bites to the hand can be infected with *Eikenella* spp.[42] The lowest infection rates are associated with the most aggressive management regimens.[43,44] Meticulous cleansing and debridement of 215 lacerations and perforations due to dog bites, together with antibiotic prophylaxis, resulted in only one infection.[44]

Bites to the hand exemplify the need for a strict protocol of vigorous debridement and irrigation. With infected bite wounds, loculated areas need to be opened to allow blood flow and antibiotics to penetrate, and specimens to be taken for full culture. All wounds should be left open. Immobilization of the limb until marked clinical improvement is seen is particularly necessary for clenched fist injuries. Where adequate debridement of deep wounds, especially cat bites, is not possible, irrigation with 250 mL of saline, using a 19- or 20-gauge needle or plastic intravenous catheter on a 35-mL syringe, is essential.[43,44]

To suture or not to suture?

The major risk of early wound closure is infection. Since clean, bleeding facial wounds rarely become infected, primary repair after surgical debridement and wound toilet with antibiotic prophylaxis is the favoured approach. In one series, where primary closure, reconstruction with a local flap, mucosal advancement, split skin or full-thickness grafts were necessary, minor infection occurred in only one patient.[45]

Bite wounds to the hand should be allowed to heal by secondary intention where possible. The most common pitfall is to underestimate the seriousness of 'fight bite' wounds. The clenched fist wound is often inflicted with considerable force, frequently resulting in metacarpophalyngeal joint capsule perforation, tendon rupture and sometimes fracture.[2] An apparently innocuous 3–5-mm laceration over a dorsal metacarpophalyngeal joint may overlie a deep bacterial inoculum, sucked deep into the wound with extension. Open access to the joint spaces and tendon sheath allows rapid spread of infection to the wrist and dorsum of the hand. Only when felt clean and free of infection, should the wound edges be re-approximated. Significant bites require a 'second look' operative procedure 24–48 h after initial radical debridement to ensure that there is no collection of pus, dead tissue or focus of persisting infection.[17]

Management of snake bites and envenomation

The only indigenous venomous snake in the UK is the adder, usually biting victims during the summer months. Approximately 70% of adder bites result in trivial symptoms developing within minutes of the bite.[46]

Feelings of faintness, nausea and drowsiness are common. Hypotension is the most important sign of systemic envenoming, usually developing within 2 h and may resolve or progress. Abdominal colic, incontinence of urine or faeces, sweating, vasoconstriction, tachycardia and angio-oedema may be delayed for several hours. Rarely, mild coagulopathy and bleeding occur, with seizures (due to hypotension or cerebral oedema) renal failure or cardiac arrest.[46]

Envenomation occurs in 50% of UK adder bites, causing dermonecrosis and haematological abnormalities. There is usually oedema, sometimes massive and usually present within 2 h. Symptomatic snakebite victims should be monitored in hospital for 24 h, with blood pressure and limb girth in the area of the bite assessed regularly.

A white cell count, creatine phosphokinase, bicarbonate and coagulation screen should be

done as a baseline, with 12-hourly electrocardiograms (ECG) if hypotensive. General resuscitation measures should be applied if symptoms develop. Indications for Zagreb antivenom include severe envenomation, swelling of the limb within 2 h of the bite, a white cell count $>20\,000/mm^3$, haemorrhage, hypotension or ECG abnormalities.[12] Since the advent of Zagreb antivenom, deaths are rare (1.22 per thousand bites).[12] The last UK death occurred in 1975.[46]

When dealing with envenomation from imported venomous snakes, specialist advice should be sought regarding the identification and appropriate antivenom preparation. Despite anecdotal reports of inactivation of snake venom by electric shock, this is not recommended[47] and the use of tourniquets are similarly discouraged as the limb should only be immobilized to prevent the spread of venom.[46]

The oral flora of tropical snakes shows a predominance of Gram-negative organisms, including *Hafnia* spp. and pseudomonads.[48,49] Infection following snake bites is relatively unusual, with reports of Gram-negative organisms such as vibrios and aeromonads.[50,51] Snake venom is a particularly potent antibacterial, but has little or no activity against anaerobes. Hence, thorough cleaning and debridement of the wound is vital. Where felt justified, any prophylactic antibiotics should be active against anaerobes.[48] Prophylactic antibiotics are particularly recommended after rattlesnake bites.[51]

Microbiology of human and animal bites

Many sexually related human bites present late due to the embarrassment factor. Serious infections including streptococcal toxic shock syndrome,[52] Fournier's gangrene,[53] genital ulcer[54,55] and syphilis[56] have been reported after penile bites.

Studies of human bite infections have found an average of five different micro-organisms per wound, 60% being anaerobes.[57] Unlike animal bite wounds, up to 45% of the anaerobes isolated from clenched fist injuries are beta-lactamase producers. *Eikenella corrodens* is present in animal and human mouth flora,[58] and present in 25% of human bites to the hand.[59,60]

In the routine laboratory, lack of familiarity with the more unusual pathogens in animal saliva can make the culture and identification of organisms from animal bites difficult. Using modern identification kits, it is easy to misidentify organisms, particularly the unusual Gram negatives and pasteurellae.[61,62] Almost 30% of 'penicillin-sensitive *Staphylococcus aureus*' isolated from dog bites are in fact *Staphylococcus intermedius*.[63] Differentiation is by the modified Voges Proskauer reaction, *S. intermedius* being positive.

The historical inadequacy of culture methods in the older studies, which often excluded anaerobic culture, explains the paucity of potential pathogens, particularly anaerobes, reported. Plates should be incubated for five to seven days for optimal isolation of unusual pathogens.[64]

Most UK laboratories still perform a comparatively rudimentary anaerobic culture, confining their report to 'mixed anaerobes, sensitive to metronidazole'. In contrast and using very comprehensive anaerobic culture methods, one-third of animal bites yielded *Fusobacterium* spp. and 30% yielded *Bacteroides* spp.[65] In 56%, the anaerobes were mixtures of fusobacteria, *Porphyromonas* and *Prevotella* spp. One cat bite yielded *Clostridium sordellii*.[65]

With prolonged cultures and more sophisticated enrichment media, the number of pathogens isolated increases dramatically. Using supplemented brucella agar, for example, increases the isolation of *Porphyromonas* spp. dramatically.[64]

P. multocida is usually isolated from those bite wounds presenting within 12 h of the bite. This organism deserves special consideration because of the propensity for metastatic infection and severe sequelae, the mortality of pasteurella septicaemia exceeding 30%.[66]

Although pasteurella infection produces an early intense inflammatory response with considerable tissue involvement, paradoxically, regional lymphadenopathy, lymphangitis or fever occurs in less than 20% of patients. Historically believed to be less common in dog bites, *P. multocida* was in fact isolated from more than half the dog bites cultured.[63] Deep-seated infection and tenosynovitis due to *P. multocida* can be difficult to eradicate, even with days of high-dose intravenous antibiotics to which the organism is sensitive.[67,68]

In addition to staphylococci, streptococci, *P. multocida* and anaerobes present in the oral flora of virtually all animals, many other pathogens have been isolated from infected bites. The increasingly diverse pet population means that bites from exotic animals are now seen. Table I illustrates some of the more unusual organisms to consider when culturing wounds.

Antibiotic prophylaxis for human bites

The role of antibiotic prophylaxis in uncomplicated

Table I Unusual organisms reported from animal bites

Species of animal	Organisms reported
Rats	*Streptobacillus moniliformis*,[69] *Leptospira* spp.,[70] cowpox virus[71]
Horses, donkeys	*Actinobacillus lignieresii*,[72,73] anaerobes,[74] *Pasteurella caballii*,[62,73] *Staphylococcus hyicus*[75]
Hamsters, guinea pigs	Acinetobacter,[76] *Pasteurella* spp.[77]
Gerbils	*Streptobacillus moniliformis*[78]
Sheep	*Actinobacillus* spp.[73]
Pigs	*Pasteurella* spp., *Escherichia coli*, *Proteus* spp., *Streptococcus milleri*, anaerobes,[79] *Flavobacterium type* 2[80,81]
Birds	*Pseudomonas* spp.,[82] *Bacteroides* spp.,[83] *C. tetani*[84]
Ferrets	*Mycobacterium bovis*[85]
Iguanas	*Serratia marcescens*,[86,87] Group B streptococcus
Marine animals	*Aeromonas* spp., *Vibrio* spp., *Pseudomonas* spp., atypical mycobacteria[88]

bite wounds is controversial. The human mouth can contain more than 40 different species of bacteria,[41] including *E. corrodens* (2.6%)[59] and *Veillonella* spp. An average of five different organisms is found in infected human bite wounds,[57] but unlike animal bites, the anaerobes in human bites are usually beta-lactamase positive.

In general, when the patient is seen within 3 h, the wound is clean and does not involve the hand. If no signs of infection are present, antibiotic prophylaxis can be avoided.[7,89]

Antibiotic prophylaxis for animal bites

Antibiotic prophylaxis in animal bites is even more controversial. Certainly not all animal bites warrant antimicrobial prophylaxis and each case should be decided on its merits. No large, double-blinded studies comparing the most recommended prophylactic antibiotic (co-amoxiclav) with other antimicrobials have been performed.

A plethora of old, badly conducted studies comparing a variety of differing antimicrobials, with inadequate microbiological analysis of wounds[7] have led to the proliferation of differing recommendations with little valid evidence. Recommendations vary from empirical prophylaxis for all animal bites[17] to restricting prophylaxis to injuries or patients deemed to be at high risk of infection,[7] and a variety of antimicrobials are suggested. In one trial, patients given prophylactic penicillin had a 10% rate of infection compared with 25% in those given no prophylaxis,[90] but another trial, involving 55 children with 'simple' dog bites, concluded that prophylactic penicillin was unnecessary.[91]

In two double-blind, placebo-controlled studies, the value of antimicrobial prophylaxis in dog and cat bite wounds presenting within 24 h was compared. The authors concluded that prophylaxis was warranted in cat bites but not in dog bites.[92,93] Sometimes the 'prophylaxis' is actually early therapy. Such a double-blind study of 'prophylactic' co-amoxiclav administered to 185 bite victims appeared to show a significant benefit, leading the authors to conclude that prophylaxis was best 9–24 h after the injury.[94]

Another double-blind trial of prophylactic co-trimoxazole resulted in a reduction in wound infection rates from 13.8% (placebo group) to 5.5% (treatment group) but did not reach statistical significance.[95] Of note is that both these studies excluded high-risk hand bites, which were far more likely to have been infected and therefore probably benefited from prophylaxis.

Meta-analyses have been done to attempt to resolve these issues. At first glance, one involving eight randomized trials where antibiotics reduced the infection rate by 42% seems persuasive evidence for prophylaxis.[96] However, these figures were considerably skewed by the inclusion of one trial with an abnormally high infection rate of 60%. A more recent Cochrane review of eight studies[97] concluded that there was no evidence that prophylactic antibiotics were effective for cat and dog bites. However, there was evidence of a reduction in infection following human bite prophylaxis, especially bites to the hand (2% vs 28% in the control group).[97] Again, the paucity of studies and the inclusion of only one series of cat bites make the results difficult to extrapolate. Several authors concluded that prophylaxis was not beneficial in simple facial dog bites, but recommended prophylaxis in puncture wounds,[91,98,99] primary closures,[45] high-risk patients or patients with oral-cutaneous (through and through) human bites.[100]

A new area of controversy regarding bite prophylaxis involves patients with prosthetic joints. Numerous reports of prosthetic joint infections

with *P. multocida* following cat bites have prompted several authors to advocate prophylactic antibiotics.[101–103]

Overall, the consensus is that prophylaxis should be considered for all bite wounds after primary closure,[104] puncture wounds, cat bites to hand and wrist, clenched fist injuries and crush wounds with devitalized tissue.[2,7] Prophylaxis has also been recommended for patients with medical conditions predisposing to infection after animal bites such as mastectomy, prosthetic joints, diabetes mellitus, immunosuppression and splenectomy.[105] Asplenics and cirrhotic patients are especially susceptible to *C. canimorsus* infection. Hence, such patients should be advised to seek medical attention and prophylactic antibiotics after even trivial animal bites.[20]

Cat and dog bite infections are polymicrobial, with an average of 2.8–3.6 organisms isolated per wound cultured.[106,107] Pasteurellae and *Bacteroides* spp. predominate, compared with human bites, and the latter is more likely to contain *E. corrodens* and *Veillonella* spp.[58] *E. corrodens* has been reported in 20% of clenched fist injury infections, with only 82% susceptible to penicillin.[59] Hence, prophylaxis should cover staphylococci, streptococci, anaerobes, pasteurellae and *Eikenella* spp. Therefore, the prophylactic antibiotic of choice for companion animal and domestic animal bites is co-amoxiclav, effective against all of 173 aerobic and anaerobic isolates isolated from domestic animal bites.[108]

Antimicrobial therapy of infected bites

Infected bites presenting less than 12 h after the injury are likely to be infected with *Pasteurella* spp., whereas those presenting more than 24 h after the event are predominantly infected with staphylococci or anaerobes, often beta-lactamase producers. Whilst beta-lactamase-producing *P. multocida* have been reported rarely,[109] all remain susceptible to co-amoxiclav. Alternatives include second-generation cephalosporins, doxycycline, co-trimoxazole and fluoroquinolones.

Nearly 70% of *P. multocida* are resistant to erythromycin[110] and all are resistant to flucloxacillin and clindamycin.

For pig bites, where *Flavobacterium 2B* is inherently resistant to co-amoxiclav,[81] and marine animal bites, where *Vibrio*, *Aeromonas* and *Pseudomonad* spp. are likely pathogens,[88] the addition of ciprofloxacin may be justified.

Alternative prophylaxis for patients allergic to penicillin

Erythromycin must not be used alone for animal bites. Not only does erythromycin not cover *Fusobacterium* spp., *Moraxella* spp. and peptostreptococci, but less than 30% of pasteurellae are sensitive. Similarly, erythromycin is not an option as the sole agent for human bites in the penicillin-allergic individual, since only 63% of *Eikenella* spp. are sensitive.[59] Clinical failures of erythromycin have resulted in breakthrough pasteurella infections, including fulminant septicaemia and meningitis.[111,112]

For truly penicillin-allergic patients, effective alternatives to co-amoxiclav include tetracyclines, a second-generation cephalosporin with anti-anaerobic activity such as cefoxitin, or combination therapy with clindamycin and a fluoroquinolone.

Pregnant women with a history of skin rash following penicillin should be offered cefoxitin or ceftriaxone. Any other situations deserve the consideration of a medical microbiologist, since much depends on patient factors, the nature of the injury and the animal species involved.

Among the newer antibacterials, linezolid[113] proved extremely effective against all *P. multocida*, staphylococci, streptococci, fusobacteria, porphyromonas and peptostreptococci and almost all *Bacteroides tectum* isolates. However, like erythromycin, linezolid will not cover *Moraxella catarrhalis* or *E. corrodens*.

Inpatient therapy for infected bites

Hospital admission is indicated for patients with rapidly spreading cellulitis, signs of sepsis or any involvement of bone or joint.[12]

Patients presenting 'early', i.e. less than 12 h after the incident, usually do so because of spreading cellulitis or septic arthritis. Such patients have an unusually high incidence of pasteurella infection, particularly if bitten by a cat. The pathogens in companion animal bites are predictable, especially following inadequate debridement or incorrect antibiotic prophylaxis, which contributes significantly to the excessive morbidity due to pasteurella infection. Seventy percent of patients admitted with pasteurella-infected bites have received inadequate or incorrect antibiotics, usually flucloxacillin or erythromycin, both indefensible choices.[114]

Co-amoxiclav, even parenterally, may well not suffice as initial therapy for severe infections. Bites

likely to be infected with pasteurellae or 'exotic animal' bites may need initial intravenous ciprofloxacin treatment in addition to the anaerobic cover provided by a beta-lactam or metronidazole.

High-dose, intravenous benzyl penicillin combined with metronidazole and ciprofloxacin provide good initial cover, although for very severe infections, we use imipenem-cilastatin (500 mg qds IV) and clindamycin (900 mg qds IV) until Gram stains or cultures are available. For severely penicillin-allergic patients, ciprofloxacin 400 mg bd IV plus metronidazole 500 mg tds would replace the imipenem.

Length of treatment

When cellulitis is already present, a therapeutic course of 10–14 days may be necessary, extended to three weeks for tenosynovitis, four weeks for septic arthritis and six weeks for osteomyelitis. In practice, intravenous therapy until the C-reactive protein falls to less than 50 mg/L is a pragmatic and objective indication for changing to oral antibiotics. If C-reactive protein does not fall rapidly or remains static, clinical re-appraisal and a second debridement is advisable, particularly with joint space infections.

References

1. Baxter DN. The deleterious effects of dogs on human health: dog-associated injuries. *Community Med* 1984;**6**: 29.
2. Griego RD, Rosen T, Orego IF, Woolf JE. Dog, cat and horse bites: a review. *J Am Acad Dermatol* 1995;**33**:1919—1929.
3. Harris D, Imperato P, Oken B. Dog bites; an unrecognised epidemic. *Bull N Y Acad Med* 1974;**50**:981—984.
4. Sacks JJ, Lockwood R, Hornreich J, Sattin RW. Fatal dog attacks 1981–94. *Pediatrics* 1996;**9**:891—895.
5. Barnham M. Once bitten twice shy; the microbiology of animal bites. *Rev Med Microbiol* 1991;**2**:231—236.
6. Wright JC. Reported cat bites in Dallas. Characteristics of the cats, the victims and the attack events. *Public Health Rep* 1990;**105**:420—424.
7. Callaham ML. Prophylactic antibiotics in common dog bite wounds; a controlled study. *Ann Emerg Med* 1980;**9**: 410—414.
8. Francis DP, Holmes MA, Brandon G. *Pasteurella multocida*: infections after domestic animal bites and scratches. *JAMA* 1975;**233**:42—46.
9. Lindsey D, Christopher M, Hollenbach J, Boyd JH. Natural course of the human bite wound: incidence of infection and complications in 434 bites and 807 lacerations in the same group of patients. *J Trauma* 1987;**1**:45—48.
10. Garrard J, Leland N, Smith DK. Epidemiology of human bites to children in a day care centre. *Am J Dis Child* 1988;**142**: 643—650.
11. Infectious Diseases and Immunisation Committee. A bite in the playroom: managing human bites in day care settings. *Can J Infect Dis* 1998;**9**:267—269.
12. Higgins MAG, Evans RC, Evans RJ. Managing animal bite wounds. *J Wound Care* 1997;**6**:377—380.
13. Bass JW, Vincent JM, Peron MD. The expanding spectrum of *Bartonella* infection: cat-scratch disease. *Pediatr Infect Dis* 1997;**16**:163—179.
14. Cole RP. Dog bites to children. *BMJ* 1991;**303**:465.
15. Levene S. Dog bites to children. *BMJ* 1991;**303**:466—468.
16. Chambers GH, Payne JF. Treatment of dog bite wounds. *Minn Med* 1969;**52**:427—430.
17. Wiggins ME, Akelman ME, Weiss AP. The management of dog bites and dog bite infections of the hand. *Ann Emerg Med* 1980;**9**:410—414.
18. Kullberg B-J, Westendorp RG, Van't Wout JW, Meinders AE. Purpura fulminans and symmetrical gangrene caused by *Capnocytophaga canimorsus* (formerly DF-2) septicaemia—a complication of dog bites. *Medicine* 1991;**70**:287—291.
19. Job L, Horman JT, Grigor JK, Israel E. Dysgonic-fermenter-2: a clinico-epidemiologic review. *J Emerg Med* 1989;**7**: 185—192.
20. Morgan MS. *Capnocytophaga canimorsus* in peripheral blood smears. *J Clin Pathol* 1994;**47**:681—682.
21. Pirez RE. DF2 infection. *West Med J* 1988;**148**:90—92.
22. Muguti GI, Dixon MS. Tetanus following human bite. *Br J Plast Surg* 1992;**45**:614—615.
23. Agrawal K, Ramachandrudu T, Hamide A, Dutta TK. Tetanus caused by human bite of the finger. *Ann Plast Surg* 1995;**34**: 201—212.
24. Vodopja I. Current issues in human rabies immunisation. *Rev Infect Dis* 1988;**10**(Suppl. 4):S758—S763.
25. Whitby JE, Heaton PR, Black EM, Wooldridge M, McElhinney LM, Johnstone P. First isolation of a rabies-related virus from a Daubenton's bat in the United Kingdom. *Vet Rec* 2000;**147**:385—388.
26. CDSC. Bat brings rabies to Britain. *Commun Dis Rep CDR Wkly* 1996;**6**:205.
27. Jackson AC, Brock Fenton M. Human rabies and bat bites. *Lancet* 2001;**357**:1714.
28. Rosario NC, Blair A, Rickman LA. Herpetic hickey. *N Engl J Med* 1987;**31**:754—755.
29. Dusheiko GM, Smith M, Scheuer PJ. Hepatitis C virus transmitted by human bite. *Lancet* 1990;**33**:653—654.
30. Figueiredo JFC, Borges AS, Martinez R, Martinelli A, Villanova MG. Transmission of hepatitis C virus but not human immunodeficiency virus type 1 by a human bite. *Clin Infect Dis* 1994;**19**:546—547.
31. Shirley LR. Risk of transmission of human immunodeficiency virus by the bite of an infected toddler. *J Pediatr* 1989;**114**: 425—427.
32. Tereskerz PM, Bentley N, Jagger J. Risk of human immunodeficiency virus infection after a human bite. *Lancet* 1996;**365**:1512.
33. Tsoukas C, Hadgis T, Schuster J, Thuberge L, Feorino P, O'Shaughnessy M. Lack of transmission of HIV through human bites and scratches. *J Acquir Immune Defic Syndr* 1998;**1**:505—507.
34. Vidmar L, Polijak M, Tomazik J, Aema K, Laus I. Transmission of HIV-1 by human bite. *Lancet* 1996;**354**: 1762—1763.
35. Anonymous. Transmission of HIV by human bite. *Lancet* 1987;**2**:522.
36. Khajotia RR, Lee E. Transmission of human immunodeficiency virus through saliva after a lip bite. *Arch Int Med* 1997;**157**:1901.

37. Pretty IA, Anderson GS, Sweet DJ. Human bites and the risk of human immunodeficiency virus transmission. *Am J Forensic Med Pathol* 1999;**20**:232–239.
38. Richman KM, Rickman LS. The potential for transmission of human immunodeficiency virus through human bites. *J Acquir Immune Defic Syndr* 1993;**6**:402–406.
39. Guy RJ, Rosen T, Orengo IF, Wolf JE. Successful treatment of acute head and neck dog bite wounds without antibiotics. *Ann Plast Surg* 1986;**1**:45–48.
40. Mcheik JN, Vergniss P, Bondonny JM. Treatment of facial dog bite injuries in children: a retrospective study. *J Pediatr Surg* 2000;**35**:580–583.
41. Mann RT, Hoffield TA, Farmer CB. Human bite of the hand: twenty years of experience. *J Hand Surg (Am)* 1977;**2**: 97–104.
42. Dellinger EP, Wertz M, Miller SD, Coyle MB. Hand infections. *Arch Surg* 1988;**123**:745–751.
43. Spiegl JD, Szabo RM. A protocol for the treatment of severe infections of the hand. *J Hand Surg* 1988;**13A**:254–259.
44. Zook EG, Miller M, Van Beek AL. Successful treatment protocol for canine fang injuries. *J Trauma* 1980;**20**: 243–246.
45. Javaid M, Feldberg L, Gipson M. Primary repair of dog bites to the face: 40 cases. *J R Soc Med* 1998;**3**:414–416.
46. Reading CJ. Incidence, pathology and treatment of adder (*Viperus berus* L.) bites in man. *J Accid Emerg Med* 1996; **13**:346–351.
47. Dart RC, Gustafson RA. Failure of electric shock treatment for rattlesnake envenomation. *Ann Emerg Med* 1991;**20**: 659–661.
48. Thisyakorn U, Thisyakorn C. *Aeromonas* infection following a snake bite; a case report. *Southeast Asian J Trop Med Public Health* 1988;**19**:307–308.
49. Wu CH, Yang DY, Hung DZ, Peng YC. Snakebite complicated with *Vibrio vulnificus* infection. *Vet Hum Toxicol* 2001;**43**: 283–285.
50. Blaylock RS. Antibiotic use and infection in snakebite victims. *S Afr Med J* 1999;**89**:874–876.
51. Goldstein EJ, Citron DM, Gonzalez H, Russell FE, Finegold SM. Bacteriology of rattlesnake venom and implications for therapy. *J Infect Dis* 1979;**140**:818–821.
52. Behar DM, Edelshtein S, Ben-Ami H, Mansano R, Edoute Y. Human bite on the penile shaft from oral sex as a portal of entry for streptococcal toxic shock syndrome. *Isr Med Assoc J* 2000;**2**:945–947.
53. Bernstein AM, Celanpo T, Sibulkin A. Fournier's gangrene of the penis. *South Med J* 1976;**69**:1242–1245.
54. Weinert P, Heiss J, Roinecker H, Sing A. A human bite. *Lancet* 1999;**354**:572.
55. Rosen T, Conrad N. Genital ulcer caused by human bite to the penis. *Sex Transm Dis* 1999;**26**:527–530.
56. Fiumara NJ, Exner JH. Primary syphilis following a human bite. *Sex Transm Dis* 1981;**3**:21–22.
57. Goldstein EJC. Management of human and animal bite wounds. *J Am Acad Dermatol* 1989;**21**:1275–1279.
58. Goldstein EJC, Barones MF, Miller TA. *Eikenella corrodens* in hand infections. *J Hand Surg (Am)* 1983;**8**:563–567.
59. Rayan GM, Doewnard D, Cahill S, Flournoy DJ. A comparison of human and animal mouth flora. *J Okla State Med Assoc* 1991;**84**:510–515.
60. Rayan GM, Downard D, Cahill SL, Flournoy DJ. *Eikenella corrodens* in human mouth flora. *J Hand Surg (Am)* 1988;**6**: 953–956.
61. Frederiksen W, Tanning B. Possible misidentification of *Haemophilus aprophilus* as *Pasteurella gallinarum*. *Clin Infect Dis* 2001;**32**:987–988.
62. Schlater LK, Brenner DJ, Steigerwalt AG, Moss CW, Lambert MA, Packer RA. *Pasteurella caballi*, a new species from equine clinical specimens. *J Clin Microbiol* 1989;**27**: 1769–1774.
63. Talan DA, Staatz A, Goldstein EJC, Singer K, Overturf GD. *Staphylococcus intermedius* in canine gingiva and canine inflicted wound infections: a laboratory characteristic of newly recognised zoonotic pathogen. *J Clin Microbiol* 1989; **27**:78–81.
64. Goldstein EJC. New horizons in the bacteriology, antimicrobial susceptibility and therapy for animal bite wounds. *J Med Microbiol* 1998;**47**:95–97.
65. Talan DA, Citron DM, Abrahamian FM, Moran GJ, Goldstein EJC. Bacteriologic analysis of infected cat and dog bites. *N Engl J Med* 1999;**340**:285–292.
66. Raffi F, Baron D, Drudgeon HB, Nicolas F, Courtieu AL. *Pasteurella multocida* bacteraemia: report of thirteen cases over twelve years and review of the literature. *Scand J Infect Dis* 1987;**19**:385–393.
67. Fahmy FS, Morgan MS, Saxby PJ. *Pasteurella* tenosynovitis following a dog bite. *Injury* 1994;**25**:262–263.
68. Spagnuolo PJ, Friedman RI. Penicillin sensitivity of invasive and non-invasive *Pasteurella multocida*. *J Antimicrob Chemother* 1979;**5**:324–325.
69. Hagelskjaer L, Sorensen I, Randers E. *Streptobacillus moniliformis* infection: 2 cases and a literature review. *Scand J Infect Dis* 1998;**30**:309–311.
70. Gollop J, Katz A, Ruddy R, Sasaki D. Rat-bite leptospirosis. *West J Med* 1993;**159**:76–77.
71. Postma BH, Diepersloot RJA, Niessen GJCM, Droog RP. Cowpox-virus-like infection associated with a rat bite. *Lancet* 1991;**337**:733–734.
72. Benaoudia F, Escande F, Simonet M. Infection due to *Actinobacillus lignieresii* after a horse bite. *Eur J Clin Microbiol Infect Dis* 1994;**13**:439–440.
73. Peel MM, Hornidge KA, Stacpoole AM, Weaver RE. *Actinobacillus* and related bacteria in infected wounds of humans bitten by horses and sheep. *J Clin Microbiol* 1991;**29**: 2535–2538.
74. Escande F, Valee E, Aubert F. *P. caballii* infection following a horse bite. *Zentralbl Bakteriol* 1997;**285**:440–444.
75. Osterlund A, Nordlund E. Wound infection caused by *Staphylococcus hyicus* subspecies *hyicus* after a donkey bite. *Scand J Infect Dis* 1997;**29**:95.
76. Goldstein EJC, Citron DM. Anaerobic osteomyelitis following a horse bite. *Infect Dis Clin Pract* 1998;**2**:357–358.
77. Martin RW, Martin DL, Levy CS. *Acinetobacter* osteomyelitis from a hamster bite. *Pediatr Infect Dis J* 1988;**67**:364–365.
78. Lion C, Conroy MC, Dupu ML, Escande F. *Pasteurella* sp. group infection after a guinea pig bite. *Lancet* 1995;**346**: 901–902.
79. Wickens EG, Miller JG, Cockroft PM, Okubadjeo OA. Rat bite fever in a gerbil feeder. *J Infect* 1988;**16**:177–180.
80. Barnham M. Pig bite injuries and infection: report of seven human cases. *Epidemiol Infect* 1988;**101**:641–645.
81. Goldstein EJ, Citron DM, Merkin TRE, Pickett MJ. Recovery of an unusual *Flavobacterium group 2B* like isolate from a hand infection following a pig bite. *J Clin Microbiol* 1990; **28**:1079–1081.
82. Ebrey RJ, Hayek LJE. Antibiotic prophylaxis after a swan bite. *Lancet* 1997;**350**:340.
83. Davis B, Wenzel RP. Striges scalp *Bacteroides* infection after an owl attack. *J Infect Dis* 1992;**165**:975–976.
84. Kara CO, Cetin CB. Cephalic tetanus as a result of rooster pecking: an unusual case. *Scand J Infect Dis* 2002;**34**: 64–66.

85. Jones JW, Pether JV, Rainey HA, Swinburn CR. Recurrent *Mycobacterium bovis* infection following a ferret bite. *J Infect* 1993;**26**:225–226.
86. Tsieh S, Babl FE. *Serratia marcescens* cellulitis following an iguana bite. *Clin Infect Dis* 1999;**28**:225–226.
87. Teitel AD. B(n)ite of the iguana. *Am J Emerg Med* 1990;**8**: 567–568.
88. Auerbach PS, Yajko ADM, Nassos PS, Kizer KW, McCosker J. Bacteriology of the marine environment: implications for clinical therapy. *Ann Emerg Med* 1987;**2**:643–649.
89. Dreyfuss UY, Orthop M, Seiger M. Human bites of the hand: a study of one hundred and six patients. *J Hand Surg* 1985;**10A**:884–889.
90. Callaham M. Controversies in antibiotic choices for bite wounds. *Ann Emerg Med* 1988;**17**:1321–1329.
91. Boenning DA, Fleischer GR, Campos JM. Dog bites in children: epidemiology, microbiology and penicillin prophylactic therapy. *Am J Emerg Med* 1983;**1**:17–21.
92. Elenbaas R, McNabey WK, Robinson WA. Prophylactic oxacillin in dog bite wounds. *Ann Emerg Med* 1982;**11**: 248–251.
93. Elenbaas R, McNabey WK, Robinson WA. Evaluation of prophylactic oxacillin in cat bite wounds. *Ann Emerg Med* 1984;**13**:155–157.
94. Brakenbury PH, Muwanga C. A comparative double blind study of amoxycillin/clavulanate vs. placebo in the prevention of infection after animal bites. *Arch Emerg Med* 1989;**6**:251–256.
95. Jones DA, Stanbridge TN. A clinical trial using co-trimoxazole in an attempt to reduce wound infection rates in dog bite wounds. *Postgrad Med J* 1985;**61**:593–594.
96. Cummings P. Antibiotics to prevent infection in patients with dog-bite wounds; a meta analysis of randomised trials. *Ann Emerg Med* 1993;**23**:535–540.
97. Medeiros I, Saconato H. *Antibiotic prophylaxis for mammalian bites (Cochrane review)* The Cochrane Library 2003 [Issue 4].
98. Dire DJ. Emergency management of dog and cat bite wounds. *Emerg Med Clin North Am* 1992;**10**:719–736.
99. Dire DJ. Cat bite wounds: risk factors for infection. *Ann Emerg Med* 1991;**9**:973–979.
100. Morgan JP, Haug RH, Murphy MT. Management of facial dog bite injuries. *J Oral Maxillofac Surg* 1995;**53**:435–441.
101. Maradona JA, Carton JA, Rodriguez Guarardado A, Lizon Castellano J. Prosthetic joint infection by *Pasteurella*. *Eur J Clin Microbiol Infect Dis* 1997;**16**:623–625.
102. Gomez-Reino JJ, Sha M, Gorevic P, Lusskin R. *Pasteurella multocida* arthritis. *J Bone Joint Surg* 1980;**62A**: 1212–1213.
103. Takwale VJ, Wright ED, Bates J, Edge AJ. *Pasteurella multocida* infection of a total hip arthroplasty following cat scratch. *J Infect* 1997;**34**:263–264.
104. Moore F. I've just been bitten by a dog. *BMJ* 1997;**314**: 88–89.
105. Presutti RJ. Prevention and treatment of dog bites. *Am Fam Phys* 2001;**63**:1567–1572.
106. Weber DJ, Hansen AR. Infections resulting from animal bites. *Infect Dis Clin North Am* 1991;**5**:663–680.
107. Brook I. Microbiology of human and animal bite wounds. *Am Fam Phys* 1987;**141**:703–705.
108. Goldstein EJ, 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.
109. Lion C, Lozniewski A, Rosner V, Weber M. Lung abscess due to beta-lactamase producing *Pasteurella multocida*. *Clin Infect Dis* 1999;**29**:1345–1346.
110. Weber D, Wolfson JS, Swartz MN, Hooper DC. *Pasteurella multocida* infections report of 34 cases and review of the literature. *Medicine* 1984;**63**:133–154.
111. Levin JM, Talan DA. Erythromycin failure with subsequent *Pasteurella multocida* meningitis and septic arthritis in a cat-bite victim. *Ann Emerg Med* 1990;**19**:1458–1461.
112. Kumar A, Devlin HR, Vellend H. *Pasteurella multocida* meningitis in an adult: case report and review. *Rev Infect Dis* 1990;**12**:440–448.
113. Goldstein EJ, Citron D, Merriam CV. Linezolid activity compared to those of selected macrolides and other agents against aerobic and anaerobic pathogens isolated from soft tissue bites infections in humans. *Antimicrob Agents Chemother* 1999;**43**:1469–1474.
114. Holm M, Tarnvik H. Hospitalisation due to *Pasteurella multocida*–infected animal bite wounds: correlation with inadequate primary antibiotic medication. *Scand J Infect Dis* 2000;**32**:181–183.