Human and animal bites
Managing and preventing infection

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Animal bites are an increasing public health problem, with half the Australian population expected to experience a significant animal bite at some time in their life. Infections from bites can range from the self-limiting to the life-threatening, with characteristics often dependent on the specific animal inflicting the bite.

Human and animal bites may lead to serious injury, including transmission of infection to the recipient. The nature of the bite, the species of the animal and the conditions of the host are all factors to consider when managing a patient with a bite injury. The organisms involved in infection often originate from the oral cavity of the offending biter, as well as the environment where the injury has occurred.[1] Although some overlap exists, the profile of infection transmitted differs depending on the animal inflicting the bite.

In one Australian study, dog bites represented the vast majority of bite injuries (79.6%), followed by human bites (8.7%), cat bites (7.2%), horse bites (1%) and rat bites (0.8%).[2] Many Australian households report pet ownership, and exotic animals are becoming increasingly popular as pets. Patients who are immunocompromised are at a higher risk of infection,[3] as are people with more comorbidities. Urbanisation has brought humans in close proximity to our native species, and increasing travel abroad brings people into proximity with less-familiar species as well as emerging zoonoses.

Animal bites are a growing public health risk, and half of all Australians will experience a significant animal bite at some time in their life.[4] The management of animal bites should involve an integrated approach that includes careful consideration of the human–animal–ecosystem interaction.

This article reviews the characteristics of some of the infections from bites by a range of domestic, occupational and exotic animal

KEY POINTS

- The nature of the bite, the species of the biting animal and the conditions of the host must all be considered when managing patients with bite injuries.
- Although some overlap exists, the profile of the infection transmitted differs depending on the animal inflicting the bite.
- Human bites result in greater infection and complication rates than other animal bites and are typically polymicrobial.
- Dogs are responsible for most mammalian bites, with almost 20% of these bites becoming infected, followed by cats, with up to 80% of cat bites becoming infected.
- Immediate management of bites includes assessment, surgery, antibiotic therapy and tetanus toxoid vaccination.
- GPs have an important role in preventing bite wounds, and follow up of patients treated for bite wounds represents an important opportunity to educate, and offer vaccinations to, those at risk of further bites.

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species as well as humans; describes the immediate management of bites; and discusses strategies to prevent bites and minimise infective complications. Arthropod bites are not discussed.

Human bites

Human bite injuries comprise clenched-fist injuries, sustained when a closed fist strikes the teeth of another person, and occlusive bites, resulting from direct closure of teeth on tissue.[5] Clenched-fist injuries are more common than occlusive bites, particularly in men,[6] with most human bites occurring on the hands.[7] Human bites result in a greater infection and complication rate than animal bites. Cultures of human bites are typically polymicrobial.[7] Mixed aerobic and anaerobic organisms are common, with the most common isolates including *Streptococcus* spp. and *Eikenella corrodens*, which occurs in up to one-third of isolates.[7] *E. corrodens* is a fastidious Gram-negative organism that has been associated with septic arthritis and culture-negative infective endocarditis (as part of the HACEK group [*Haemophilus* spp., *Aggregatibacter* ssp., *Cardiobacterium hominis, E. corrodens* and *Kingella* spp.]).[8]

Some authors suggest that all patients with human bites should be commenced on antibiotic prophylaxis, given the high risk of infection.[6] The choice of antibiotic therapy should cover *E. corrodens*, which is resistant to first-generation cephalosporins (such as cefalexin), flucloxacillin and clindamycin, antibiotics that are often used for skin and soft tissue infections. Table 1 provides the Australian Therapeutic Guidelines' antibiotic recommendations for empirical therapy of bite infections,[9] and Table 2 lists additional antibiotic recommendations according to specific animal and pathogens.[5-8,10-45]

Blood-borne viruses, particularly hepatitis B virus (HBV), may also be transmitted by human bites, although the risk is low and evidence is limited to case reports.[6] Baseline serology, including human immunodeficiency virus (HIV) serology; HBV surface Ab (HBsAb), core Ab (HBcAb) and surface Ag (HBsAg); and hepatitis C virus (HCV) serology, should be performed in human bite recipients. HIV post-exposure prophylaxis is generally not recommended, but may be considered in very high-risk situations (i.e. in patients with bites from known HIV-positive sources where complicated wounds occur).[46] If the recipient has no immunity to hepatitis B, and chronic HBV infection has been excluded, vaccination should be initiated. If the recipient is unvaccinated, and the source is known to be HBsAg positive, or if the HBV status of the source cannot be ascertained, the exposed person should receive a dose of hepatitis B immunoglobulin (HBIG) within 72 hours of a percutaneous exposure.[47] HCV serological testing is performed three months after exposure.[46] When relevant and possible, testing of both parties for blood-borne viruses is also recommended, as these can also be transmitted from the patient to the biter (reverse exposure).[48]

Dog bites

Most mammalian bites (about 80%) are dog bites,[2] usually occurring from a dog that is known to the bite recipient.[49]

Infection status	Antibiotic recommendation
TABLE 1. THERAPEUTIC GUIDELINE RECOMMENDATIONS FOR EMPIRICAL ANTIBIOTIC THERAPY OF ANIMAL AND HUMAN BITES[9]	
Infection not established (therapy duration: 5 days)	• Amoxicillin/clavulanic acid 875/125 mg (child: 22.5/3.2 mg/kg up to 875/125 mg) orally, 12-hourly • If commencement of oral therapy is likely to be delayed give procaine penicillin 1.5 g (child: 50 mg/kg up to 1.5 g) intramuscularly as a single dose, followed by amoxicillin/clavulanic acid • For patients who are hypersensitive to penicillins, see below
Established mild infection (therapy duration: 5 days)	• Amoxicillin/clavulanic acid 875/125 mg (child: 22.5/3.2 mg/kg up to 875/125 mg) orally, 12-hourly • For patients who are hypersensitive to penicillins, give ciprofloxacin 500 mg (child: 12.5 mg/kg up to 500 mg) orally, 12-hourly Plus Clindamycin 450 mg (child: 10 mg/kg up to 450 mg) orally, 8-hourly, Or Metronidazole 400 mg (child: 10 mg/kg up to 400 mg) orally, 12-hourly Plus either Doxycycline 200 mg (child 8 years or older: 4 mg/kg up to 200 mg) orally, for the first dose, then 100 mg (child 8 years or older: 2 mg/kg up to 100 mg) orally, daily Or Trimethoprim/sulfamethoxazole 160/800 mg (child 1 month or older: 4/20 mg/kg up to 160/800 mg) orally, 12-hourly
Established moderate to severe infection (therapy duration: 14 days)*	• Piperacillin/tazobactam 4/0.5 g (child: 100/12.5 mg/kg up to 4/0.5 g) intravenously, 8-hourly Or Ticarcillin/clavulanic acid 3/0.1 g (child: 50/1.7 mg/kg up to 3/0.1 g) intravenously, 6-hourly Or the combination of Metronidazole 400 mg (child: 10 mg/kg up to 400 mg) orally, 12-hourly Plus either Ceftriaxone 1 g (child 1 month or older: 50 mg/kg up to 1 g) intravenously, daily Or Cefotaxime 1 g (child: 50 mg/kg up to 1 g) intravenously, 8-hourly

* Total treatment duration is usually 14 days (intravenous plus oral therapy) for severe and penetrating wounds, but a longer duration of directed therapy is needed for injuries involving bones, joints and/or tendons, and specialist referral is recommended.

TABLE 2. EMPIRICAL ANTIBIOTIC THERAPY FOR SPECIFIC ANIMAL BITES

Animal	Examples of pathogens	Empirical therapy
Humans[5-8]	*Eikenella corrodens* Viridans streptococci *Streptococcus pyogenes* *Staphylococcus aureus* Coagulase-negative *Staphylococcus* Anaerobes Hepatitis B, C virus and HIV	Refer to Table 1 and text
Dogs[10-11]	*Pasteurella dagmatis,* *canis, septica* *Capnocytophaga canimorsus* *S. aureus* *Streptococcus* spp. *Moraxella* spp. Anaerobes	Refer to Table 1
Cats[12-14]	*Pasteurella multocida* *Streptococcus* spp. *S. aureus* Anaerobes *Bartonella henselae*	Refer to Table 1 Plus Consider azithromycin for *B. henselae*
Rats[15-20]	*Streptobacillus moniliformis* *Spirillum minus* *Pasteurella* spp. *Leptospira* spp.	Refer to Table 1 Plus Consider doxycycline for *Leptospira* spp.
Ungulates[13, 21-30]	*Actinobacillus* spp. Mixed aerobes/anaerobes *Pasteurella* spp. *Streptococcus equi, suis* Parapoxviruses	Refer to Table 1
Monkeys[31]	Mixed aerobes/anaerobes *Neiserria* spp. *Streptococcus* spp. *Haemophilus influenzae* Simian herpes B virus	Refer to Table 1 Plus Consider valaciclovir 1 g three times daily for 14 days[32]
Lizards[33-36]	*Salmonella* spp. *Pseudomonas aeruginosa* *Serratia marcescens*	Ciprofloxacin
Crocodiles[37,38]	*Aeromonas* spp. *P. aeruginosa* *Enterobacteriaceae* *Burkholderia pseudomallei* *Salmonella* spp. Anaerobes (incl. *Clostridium*)	Ceftazidime Plus Penicillin Plus Metronidazole
Marine animals[39-44]	*Vibrio* spp. *Aeromonas hydrophila* *Erysipelothrix rhusiopathiae* *Mycobacterium marinum* *Edwardsiella tarda*	First-generation cephalosporin Or clindamycin (if beta-lactam allergy) Plus Ciprofloxacin Plus Doxycycline if seawater exposure Plus Metronidazole if sewerage or soil contaminated water/wound (not required if clindamycin used) Refer to text for empirical management of suspected *M. marinum* infection
Seals[45]	*Mycoplasma* spp.	Doxycycline

Children are especially vulnerable to dog bites, and have increased risk of head and neck injury due to their smaller size, and possibly also due to increased provocation.[2] Most dog bites occur in males (56%), who also suffer most animal-bite fatalities.[2]

Mixed anaerobic and aerobic cultures are often present in dog bites.[10] Almost one-fifth of dog bites become infected, especially when the hand is involved.[10] The most common pathogens in dog and cat bites are *Pasteurella* spp., isolated in most cat bites and in half of all dog bites.[10] *Pasteurella canis* is most often isolated from dog bites, whereas *Pasteurella multocida* and *Pasteurella septica* are more often isolated from cat bites.[10] This is relevant for treatment, as *Pasteurella* spp. are generally resistant to many antibiotics used to treat cellulitis, including anti-staphylococcal penicillins (such as flucloxacillin), first-generation cephalosporins and clindamycin.[10] However, *P. multocida* is usually susceptible to penicillin, amoxicillin, quinolones, doxycycline and trimethoprim-sulfamethoxazole.[11]

Although *Capnocytophaga canimorsus* is rarely associated with dog bite wounds,[10] it can invade the host and cause a fulminant infection, including disseminated intravascular coagulation and septic shock, usually affecting immunocompromised and asplenic patients.[6] *C. canimorsus* has been described as an emerging infection with a case fatality rate of 26% in one recent study.[11] Penicillin is the drug of choice, and third-generation cephalosporins and beta-lactamase inhibitor combinations are other options with good activity.

Cat bites

Cat bites are the second most common type of animal bite, responsible for more than 7% of mammalian bites in Victoria, most of which occur in females.[2] Although less prevalent, cat bites become infected in up to 80% of cases, with *P. multocida* isolated in three-quarters of infections.[12] One proposed mechanism for the very high infection rate is that the sharp teeth of the cat may result in deep tissue puncture and

Figure 1. Cellulitis following a cat bite.

subsequent inoculation of bacteria.[12] Patients with *P. multocida* infection may, therefore, present with deep infection, including tenosynovitis and septic arthritis,[12] which may initially be underestimated. In contrast, *Pasteurella* spp. cellulitis is often characterised by rapid onset of spreading cellulitis (Figure 1), usually within 24 hours of the bite injury.[13]

Bartonella henselae is a fastidious, Gram-negative bacillus, and the aetiological agent of cat-scratch disease (CSD).[14] Although classically associated with cat (and particularly kitten[50]) scratches, it has been described following cat bites.[51] CSD is characterised by the development of a vesicle at the inoculation site, followed by a self-limited regional lymphadenopathy about two weeks later that can persist for weeks to several months. Low-grade fever, malaise and myalgia are often reported by affected patients.[14] Most cases of CSD are self-limited and do not require antibiotic therapy,[14] although a short course of azithromycin may be used for severe or unresolving lymphadenitis. Other manifestations include culture-negative endocarditis, neuroretinitis, pyrexia of unknown origin and bacillary angiomatosis in immunocompromised individuals, particularly those with AIDS.[52] Specialist referral is required for patients with these complex conditions.

Rodent bites

Rat bite fever (RBF) is caused by infection with either *Streptobacillus moniliformis*, a fastidious, slow-growing organism, or

Spirillum minus, so-called due to the tightly coiled spiral shape of this unculturable Gram-negative organism.[15] Although *S. moniliformis* occurs predominantly in North America, Australian cases have been reported.[16] *S. minus* occurs predominantly in Asia, where the disease is known as 'sodoku' (Japanese: so, rat; doku, poison). Rats primarily transmit the disease, although other rodents such as mice, guinea pigs and ferrets are also associated with transmission.[17] RBF occurs in about 10% of all rat bites, and those at risk include the homeless, children with pet rats, pet store workers and laboratory technicians.[17]

Symptoms of RBF usually present within one week of the bite and include fever, myalgia, sore throat, migratory arthralgia, headache and maculopapular rash.[17] In *S. moniliformis* infection, the original wound and adenopathy have usually resolved before presentation, which may hamper the diagnosis.[15] In contrast, *S. minus* has a longer incubation period of one to three weeks, and the indurated and painful wound often reappears or persists during systemic illness.[17] Complications of infection may include meningitis, pneumonitis and endocarditis.[18]

Diagnosis requires a high index of suspicion, and *S. moniliformis* requires enriched media with prolonged incubation,[17] thus the microbiologist must be informed if this diagnosis is being considered. The mortality rate of RBF is about 13% in untreated patients, and a lack of antibiotic therapy is associated with death.[16] Although penicillin is the treatment of choice for *S. moniliformis*, it is also usually sensitive to cephalosporins and tetracyclines, but may demonstrate resistance to fluoroquinolones and trimethoprim-sulfamethoxazole.[17] Antibiotic susceptibility of *S. minus* is much less studied, given the difficulty in culturing the organism.[15]

A differential diagnosis which should be considered in unwell patients following rat exposure is leptospirosis, which is the most common zoonosis and distributed globally. It is classically transmitted following transcutaneous or mucous exposure

to rat urine, although cases of transmission following rat bite have been reported.[19-20] Doxycycline remains the drug of choice to treat leptospirosis.

Monkey bites

Although monkey bites are uncommon, they do pose major health risks requiring prompt medical follow up. Theoretically, emerging infections resulting from monkey-to-human cross-species transmission could occur quite easily given the genetic similarity.[53] Travellers, zoo workers and laboratory personnel are at risk of monkey bite, and travellers are at risk of the almost universally fatal rabies virus infection, which can be transmitted by monkeys. The increasing incidence of rabies in Indonesia, and in particular Bali,[54] has ensured that Australian travellers remain at risk of exposure. Rabies post-exposure prophylaxis must therefore be offered to all returned travellers from a rabies endemic area with a history of monkey bite.[47]

Simian herpes B virus is a herpes simplex virus-like infection of macaque monkeys. It is transmissible to humans and has caused cases of fatal encephalomyelitis.[55] A few cases have occurred in laboratory personnel following macaque bites, scratches, needle-stick exposures and handling of infected bodily tissues. Travellers may be exposed to herpes B virus, with 81% of macaques in the Bali 'monkey forest' known to harbour the virus.[56] Despite this frequent human–monkey interaction, no infections have ever been reported from any of these destinations.[57] Given the unknown risk and potentially fatal complications, patients bitten by monkeys should be referred to an infectious diseases physician for consideration of prophylaxis.[32]

Bat bites, rabies and Australian bat lyssavirus

Despite being free of rabies, Australian bats are enzootic for the closely-related Australian bat lyssavirus (ABLV), which carries a similarly high mortality rate. Three cases of fatal encephalitis due to

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ABLV have been reported, with the most recent case occurring in 2013, and a case in 2000 with a reported incubation period of two years.[58] All people exposed to bats should, therefore, be assessed for post-exposure prophylaxis, regardless of the time elapsed since exposure. Recipients of bites from several other mammalian species, especially dogs, cats and monkeys, also require post-exposure prophylaxis if they have returned from rabies-endemic areas.

Rabies and ABLV exposures are categorised according to the WHO classification system (Box 1).[47] People with category I exposures require no prophylaxis if the contact history is reliable.[47] Immunocompetent patients with category II exposures require vaccination without human rabies immunoglobulin (HRIG).[47] Post-exposure rabies vaccination in immunocompetent people is given in a four-dose schedule (on days 0, three, seven and 14). An additional dose is suggested on day 28 in the immunocompromised, although deviations of a few days are probably unimportant.[47] The vaccine should be given in the deltoid muscle, as neutralising antibodies may be reduced if administered in other areas.[5] Both immunocompetent and immunocompromised patients who have received a pre-exposure rabies vaccine course only require two further doses (on day 0 and three) in the event of an exposure.[47]

HRIG is recommended for non-immune patients with category II exposures if:
- they are immunocompromised and exposed to rabies, or
- the exposure is ABLV, regardless of their immunocompromised or immunocompetent status.[47]

In addition to vaccination, HRIG is recommended for all non-immune patients with category III exposures, as long as no more than seven days has elapsed since administration of the first dose of vaccine (or the vaccine may be inactivated by immunoglobulin).[47] HRIG should be infiltrated in and around the wound, and any remaining HRIG should be administered in an area proximal to the wound.[47]

Bites from ungulates

Ungulates (hoofed animals) include horses, cows, sheep and pigs. Bites from these animals are often polymicrobial, including mixed anaerobes, Gram-positive bacteria including *Staphylococcus* and *Streptococcus* spp. and Gram-negative bacteria including *Pasteurella* and *Actinobacillus* species.[13]

Actinobacillus spp. are Gram-negative bacilli that are part of the normal flora of many ungulates (including horses, sheep, cattle and pigs).[13] Numerous cases of bites infected by *Actinobacillus* spp. have been reported, and they may result in a purulent, malodorous abscess at the site of injury.[21] *Actinobacillus* spp. are closely related to *Pasteurella* spp., so additional molecular testing to identify the organism should be requested in the appropriate setting.[22] *Actinobacillus* spp. are broadly sensitive to many classes of antibiotics used for bite-associated skin and soft tissue infection.[13]

Cattle, sheep and goat bites

Parapoxviruses include the orf virus, transmitted from sheep and goats ('scabby mouth'), and bovine papular stomatitis virus, transmitted from cattle.[23] These viruses may produce a lesion at the site of inoculation (usually on the fingers or hands), developing from a papule to a vesicle and eventually a red target-shaped ulcer over the course of one to two months.[23] The lesion often resolves by six weeks, although infection in the immunocompromised may require antiviral therapy and requires specialist referral.

Horse bites

Similar to other ungulates, horse bite infection is often polymicrobial and associated with several infections, particularly *Actinobacillus* spp. infection. Horse bite infection was shown to be the third leading cause of animal bite injury in one Victorian study.[2]

Streptococcus equi is a commensal of the upper respiratory tract of horses; infection in horses results in massive lymphadenopathy and airway obstruction known as 'strangles'.[21] Human disease due to this

organism has rarely been reported but has resulted in serious disease. Transmission of *S. equi* subspecies *zooepidemicus* after horse bite has been reported, causing meningitis and brain abscesses with long-term neurological sequelae.[24]

Rhodococcus equi is a Gram-positive bacteria carried in the gut of many domesticated animals and distributed worldwide.[25] It is weakly acid-fast and related to *Nocardia* spp. *R. equi* infection is typically associated with horses and particularly foals. Transmission is incompletely understood, but may occur via inhalation or inoculation into a wound or mucous membrane.[25] Various symptoms may occur, although many have pulmonary involvement.[22] The incidence of this infection has increased markedly, particularly as an opportunistic pathogen in the immunocompromised host.[25]

Hendra virus is transmitted by physical contact with oral and nasal secretions of an ill or dead horse. Only seven cases have occurred since the first detection of this virus in 1994, and four people have died.[26] Although transmission via horse bite has not been reported, personal protective equipment should be used when handling an unwell horse and prevention with equine vaccination is vital.[27] Given the high case fatality rate, patients with suspected Hendra virus infection should be referred to hospital for specialist assessment. Diagnosis

relies on polymerase chain reaction (PCR) identification and serology, which is available at reference laboratories.[26] No specific treatment or human vaccine is available.

Pig bites

Of all ungulates, pigs are the most likely to bite and are often associated with polymicrobial infection including *Pasteurella* and *Actinobacillus* spp.[28] *Streptococcus suis* is an emerging zoonotic infection, responsible for septicaemia and meningitis in pigs and humans with significant occupational exposure.[29] More than 1600 cases in humans have been reported, predominantly in Asia, with transmission occurring cutaneously,[29] including following pig bite.[30]

Bites from possums and other native species

Wombats, koalas and kangaroos seldom bite, although the rising urban exposure to possums increases the risk of bites.[31] Possum bites have been associated with a number of infections, including tularaemia.[59] Tularaemia is caused by the Gram-negative intracellular bacterium *Francisella tularensis*, which is widespread in the northern hemisphere and rarely causes infection in Australia. Tularaemia may be transmitted from a wide variety of animals, causing several classical syndromes depending on the route of exposure.[59] The most common form (about 80% of cases) is ulceroglandular tularaemia, with ulcerated skin lesions and painful lymphadenopathy.[59] Only a handful of cases have ever been reported in Australia, most recently in 2011, when two women were bitten by a ringtail possum in Tasmania and developed the ulceroglandular syndrome.[59] This organism's high virulence and infectivity (as few as 10 organisms required for infection) has led to its reputation as a potential bioterrorism agent.[60]

As its name suggests, the Tasmanian devil is a more aggressive marsupial which has resulted cases of bite infection, including *P. multocida*.[61] The devastating devil facial tumour disease (DFTD), a unique infectious cancer in which tumour cells themselves transmit between biting hosts, has thankfully not been reported in humans.[62]

Reptile bites

Lizards are becoming increasingly common as pets, and are known to transmit *Salmonella* species.[33] Generally, lizard oral flora is polymicrobial and reflective of the gut and skin flora of their recent meals. Although most lizards do not bite, case reports have described *Serratia marcescens* cellulitis following iguana bite,[34] and *Pseudomonas aeruginosa* septic arthritis following a monitor lizard bite.[35] Until recently, Komodo dragons were thought to harbour a deadly cocktail of oral flora, resulting in the gradual death of its prey following a bite. This has been challenged with data suggesting Komodo flora is comparable to that of other carnivorous species, with injury resulting from venom produced in previously unidentified venom glands in the lower jaw.[36]

Due to their territorial nature, crocodile attacks are often unprovoked, and survivors are likely to suffer deep and complex infections following the powerful bite of a crocodile.[37] If the patient survives the injury (noting a 27% mortality rate in Australian attacks[37]), polymicrobial infection may result, including *Clostridium* spp, *Aeromonas hydrophila*, *P. aeruginosa* and *Salmonella* spp. involvement.[37] In Australia crocodile bite wounds are also at risk of becoming infected with the environmental bacterium *Burkholderia pseudomallei*, which causes the potentially fatal infection melioidosis.[37] Some Australian authors have therefore suggested that patients with crocodile wound infections be treated broadly with antibiotics, including ceftazidime for *B. pseudomallei* (and most *Aeromonas* spp.), penicillins for *Clostridium* spp. and metronidazole for anaerobes.[38]

Snake bites in Australia rarely lead to severe localised tissue necrosis, and Australian snake venom is believed to be antibacterial in nature.[31] Routine use of antibiotic prophylaxis is not recommended, although referral of patients for antivenom should always be considered.[63]

Fresh and marine animal bites

Girt by sea, Australians may sustain wounds in marine environments, exposing them to a unique range of organisms not often encountered in everyday practice. These include *Vibrio* spp., *A. hydrophila*, *Erysipelothrix rhusiopathiae* and *Mycobacterium marinum*.[39] Bites transmitting these organisms may result in a range of presentations, from simple cellulitis to necrotising fasciitis and septic shock. Although bites are an uncommon cause of marine animal injury in Australia,[40] the significant morbidity and high mortality of subsequent infection warrants further consideration.

Vibrio spp. are classically located in warm estuarine waters, with a worldwide distribution. *Vibrio vulnificus* is a highly virulent pathogen associated with rapid and severe necrotising skin and soft tissue infection following traumatic injury, often requiring aggressive surgical debridement.[39] Cases have been reported following shark and stingray bites.[41,42] Blood cultures are positive in 30% of wound infections,[43] and should be taken before antibiotic therapy is started. On the basis of animal studies demonstrating efficacy, the US Centers for Disease Control and Prevention recommends combination treatment with a third-generation cephalosporin plus doxycycline as initial empirical antibiotic for *V. vulnificus* infection,[39] although other authors suggest doxycycline monotherapy for empirical *Vibrio* spp. therapy.[44]

A. hydrophila and other *Aeromonas* spp. are ubiquitous in aquatic environments, particularly fresh and brackish water, and are universally resistant to penicillins, amoxicillin/clavulanic acid and first-generation cephalosporins.[39] Carbapenems, fluoroquinolones and aminoglycosides usually demonstrate activity against these organisms. Empirical therapy for bite infection following aquatic exposure includes a carbapenem or fluoroquinolone to cover these organisms and other co-infecting Gram-negative bacteria.[39]

Infection with *E. rhusiopathiae*, a Gram-positive bacillus, is mainly seen following

Figure 2. Nodular lesions of fish tank granuloma.

occupational cutaneous exposure to fish and shellfish,[64] and is known as 'shrimp picker's disease', 'crab poisoning' or 'fish poisoning'. The bacterium is also ubiquitous in birds, reptiles and mammals, including pigs (which are thought to be a major reservoir), cats and dogs, which have also transmitted it following bites.[10] *E. rhusiopathiae* also causes erysipeloid, a disease characterised by well-circumscribed, violaceous plaques, most often on the hands. Unlike erysipelas, erysipeloid occurs slowly over the course of one week and usually resolves within four weeks, even without treatment.[65] Rarely, bacteraemia and endocarditis may also occur.[39] Diagnosis is confirmed by isolating the organisms in culture, and it remains susceptible to penicillins, cephalosporins, clindamycin and ciprofloxacin. Of note, *E. rhusiopathiae* is intrinsically resistant to vancomycin, which is often used to empirically treat Gram-positive bacteraemia.[39]

'Fish tank granuloma' is caused by *M. marinum*, a slow-growing acid-fast bacteria, characterised by nodular, erythematous lesions most often on the hands and arms (Figure 2). It occurs in fish handlers and aquarium workers with a history of lacerations or punctures. Bites are one mechanism of inoculation, with case reports including dolphin bite and exotic fish bite.[66,67] Diagnosis includes mycobacterial culture of biopsy samples or of the wound discharge, and/or PCR

identification.[45] Treatment with a prolonged course of combination clarithromycin and ethambutol is recommended,[45] and usually continued for two months after symptoms have resolved.[39]

Although uncommon, seal bites are known to cause 'seal finger' due to *Mycoplasma* spp., including *Mycoplasma phocacerebrale*, which has been isolated from the bite of a seal-trainer's finger.[68] Infected bites present as painful, erythematous lesions, which respond rapidly to doxycycline monotherapy.[68]

Assessment and management
Steps in the assessment and management of human and animal bite wounds are discussed below and summarised in Box 2.

Initial management
All recipients of bite injuries should be assessed for haemodynamic instability and penetrating traumatic injuries should be managed emergently, with a focus on haemorrhage control with direct pressure and assessment of neurovascular supply to distal structures. Even apparently minor injuries may cause deep penetration (e.g. cat bites) and may need exploration, which also aids in identifying foreign bodies or broken animal teeth.

Musculoskeletal injuries should be assessed with radiography for bony injury or foreign bodies, and clinical assessment (e.g. joint range of motion) with ultrasonography for injuries involving joints, deep ligaments or tendons. Patients with these injuries should be referred for surgical assessment and repair.

Wound care
Meticulous wound care includes cleaning the surface and flushing the injury for 15 minutes with soap and water, or povidone iodine if available,[13] and physical removal of major contamination. Vigorous irrigation and debridement should be performed to reduce the concentration of bacteria. Elevating the injury and applying an ice pack may provide analgesia and reduce swelling.[13]

Wound closure
The subject of primary closure, delayed closure or healing by secondary intention is controversial, as evidence is limited and recommendations differ.[13] Indications for primary closure include bite wounds where cosmesis is important (especially the face). Bite wound infection of the head and neck is less common, likely due to the excellent blood supply and lack of dependent oedema. Wounds at high risk of infection should not be closed by primary closure.[13,69]

Use of prophylactic antibiotics
Patients with wounds at high risk of infection require prophylactic antibiotics. Such wounds include:[9,13]
- crush injuries
- puncture wounds, especially from cat and human bites
- wounds overlying bone, joints, tendons and prostheses
- wounds in patients whose presentation is delayed more than eight hours
- wounds on the hands and feet
- wounds in immunocompromised hosts (including those with anatomical and functional asplenia)
- wounds in areas of venous or lymphatic compromise (e.g. lymphoedema, diabetic feet).

Treatment of infected wounds
Gram stain, culture and sensitivity testing guide subsequent antibiotic therapy; however, there is no need to take wound cultures unless the wound appears clinically infected. Clinical signs of infection include fever, erythema, oedema, wound discharge and surrounding lymphangitis. It is prudent to obtain blood cultures (for both aerobic and anaerobic organisms) and wound cultures before antibiotics are started. Full blood count and inflammatory markers (e.g. C-reactive protein level) should also be measured. Surgical consultation of patients is often required to consider debridement, exploration to assess involvement of underlying structures, abscess drainage and removal of suture material, if present.

Recommended antibiotics for empirical therapy of bite wound infections are listed in Tables 1 and 2. Patients with complex infections that involve deeper structures, such as tenosynovitis, septic arthritis and osteomyelitis, require treatment for prolonged durations (usually with an initial parenteral course for two to four weeks, depending on the structure involved and duration of infection),[9] and infectious diseases specialist referral is recommended. Cases involving infected wounds that respond poorly to empirical antibiotics or patients with multiple allergies or drug intolerances should also be discussed with an infectious diseases physician.

Tetanus prophylaxis

Tetanus is caused by the neurotoxin produced by *Clostridium tetani*, leading to tetanic contractions, myotonia and trismus. It is rare in Australia, with 156 hospitalisations between 2001 and 2006 and three deaths (case fatality rate, about 2%). In addition to animal bites, human bites have also been reported to transmit tetanus.[70] Tetanus vaccination is only available in combination with other agents (such as diphtheria, tetanus, acellular pertussis; DTPa), and given as a three-dose primary schedule at 2, 4 and 6 months of age.[47] Tetanus may occur even after apparently trivial wounds, although bites are categorised as tetanus-prone wounds requiring assessment for tetanus vaccination (Table 3).

Follow up

If patients are managed in the community, they should be educated regarding the symptoms of infection, should they develop (or worsen despite antibiotic treatment). Patients should be clinically reviewed between 24 and 48 hours after presentation, not only to assess the wound, but to monitor tolerance and compliance with antibiotic therapy, if initiated.

If empirical antibiotics were commenced, the results of Gram stain, aerobic and anaerobic cultures should be used to modify therapy based on organism and susceptibility testing.[9] If intravenous

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2. STEPS IN THE ASSESSMENT AND MANAGEMENT OF HUMAN AND ANIMAL BITE WOUNDS

1. Resuscitation
- Immobilise and elevate the bite injury
- Assess for neurovascular compromise and compress the bleeding
- Treat life-threatening injuries according to standard guidelines
- Assess for damage to deeper structures, such as tendons, ligaments and bone

2. Wound care
- Remove debris and foreign bodies, debride any devitalised tissue
- Vigorously flush the wound with soap and water or povidone iodine
- Take wound cultures only if evidence of infection and communicate with laboratory staff about suspected pathogens
- If infected, take blood cultures (aerobic and anaerobic) as well as wound cultures

3. Clarify and document history
- Confirm the species of the animal, document and report to authorities as required
- Document the injury (site, depth, pattern of injury) and circumstances of the attack
- Clarify past medical history, medications, allergies and immunisation status
- Clarify whether patient is immunocompromised

4. Wound closure
- There is limited evidence in this area and recommendations vary
- Consider closure for areas where cosmesis is an issue (usually for facial wounds)
- Avoid primary closure in wounds at high risk of infection
- Apply appropriate dressings if closure is not performed

5. Antibiotics
- Assess the need for empirical antibiotics for patients at high risk of infection
- Provide cover for aerobes and anaerobes broadly, including isolated species from infected wounds
- Assess history of allergies, intolerances, drug interactions, renal dysfunction
- Consider contraindications (e.g. prolonged QTc interval and ciprofloxacin)

6. Tetanus prophylaxis
- Assess previous history of tetanus immunisation status
- Consider tetanus immunoglobulin in patients with high-risk wounds if unvaccinated
- Provide tetanus prophylaxis even for patients with apparently low-risk wounds
- Document vaccinations given, including brand, batch number, dose number, date, time, site

7. Transmissible virus prophylaxis
- Blood-borne viruses: consider HIV and HBV PEP and HCV follow-up serology
- Rabies/Australian bat lyssavirus: administer PEP, including vaccination and HRIG
- Herpes B virus: consider specialist referral of patients for valaciclovir following macaque monkey bites
- Document PEP given, including brand, batch number, dose number, date, time, site

8. Follow-up care
- Review patient at 24 to 48 hours
- Provide education on the importance of medication compliance and signs and symptoms of infection
- Advise patient to return if features of cellulitis, sepsis or clinical deterioration occur
- Provide, or refer patient for, counselling, psychological assessment if needed and prevention strategies

Abbreviations: HBV = hepatitis B virus; HCV = hepatitis C virus; HIV = human immunodeficiency virus; HRIG = human rabies immunoglobulin; PEP = post-exposure prophylaxis.

TABLE 3. INDICATIONS FOR TETANUS TOXOID AND IMMUNOGLOBULIN[47]

Tetanus vaccination history	Clean, minor wounds		All other wounds	
	Toxoid	Immunoglobulin	Toxoid	Immunoglobulin
Three or more doses <5 years ago	No	No	No	No
Three or more doses 5 to 10 years ago	No	No	Yes	No
Three or more doses >10 years ago	Yes	No	Yes	No
Unknown or less than three doses	Yes	No	Yes	Yes

therapy was initiated, switching to oral therapy is reasonable once the patient is stable. If the pathogen remains unknown, oral antibiotic therapy should be continued as for mild infection (Table 1), remembering that polymicrobial infections are predicted and should be covered broadly.

Immunocompromised patients, who are at increased risk of transmission and more severe illness after a bite, are particularly vulnerable. An Australian study recently found that more than half of immunocompromised patients surveyed owned a pet, most exhibited risky behaviour and 30% had been scratched or bitten by their pet.[3] Immunocompromised patients with bite injury should be referred to hospital for assessment, and education remains the key to bite prevention in this group.

Another important, but underappreciated, complication of animal bites that must be addressed at follow up is post-traumatic stress disorder (PTSD). In particular, children who have been attacked by a dog may suffer from PTSD later in life.[71] Half of adults in one Australian survey lived in fear of future dog attacks, and many modified their behaviour towards dogs thereafter.[72]

Prevention

Although animals have become our much-loved companions, responsible pet ownership should always be encouraged. GPs have an important role in preventing bite wounds, and follow up represents an important opportunity to educate and offer vaccinations to people at risk of animal and human bites.

Strategies to minimise harm in the future should include the following measures.

- **Behavioural measures.** Avoid patting or touching dogs that demonstrate territorial behaviour and ensure that children are not left alone with dogs or other animals at risk of biting.[73] Avoid dogs that are eating, sleeping or caring for puppies. Dogs should not be greeted with an outstretched hand, an unfamiliar dog should be allowed to smell the person's hand first.[73] Some breeds of dogs are known to have a higher attack rate despite training, so families should consider this when purchasing a new dog.[73]
- **Rabies and ABLV prevention.** Avoidance of close contact with either wild or domestic animals is strongly encouraged, particularly for children travelling overseas. In Australia, unwell, injured or trapped bats and flying foxes should not be handled, and local wildlife rescue services should be contacted immediately for assistance. Pre-exposure vaccination should be offered to people at high risk of exposure, including those in occupations at high risk, risk-averse travellers and particularly children travelling to endemic areas.[47]
- **Tetanus immunisation.** A booster dose should be offered to those who travel and are at risk of sustaining a bite wound if more than five years have elapsed since their last dose, especially if access to travel services will be difficult.[47]

- **Hepatitis B vaccination.** Those at occupational risk of human bites, such as police, staff of correctional facilities and those who work with people with intellectual disabilities, may be at higher risk of human bites, and should receive HBV vaccination on employment.[47]
- **Immunocompromised and asplenic patients** should be educated about the risk of life-threatening infection in the event of an animal bite, and the need to seek urgent medical attention.[3]

Conclusion

Animal bites are a growing public health risk, which may lead to serious injury and transmission of infection. The nature of the bite, the species of the animal and the conditions of the host are all factors to consider when managing a patient with a bite injury. Management includes immediate wound care, prevention of tetanus transmission and antibiotic prophylaxis. Prevention of infection with education, behavioural modification and timely vaccination are also key to reducing the risk of transmission of infection in the future. MT

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A list of references is included in the online version of this article (www.medicinetoday.com.au).

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Human and animal bites
Managing and preventing infection

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