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PAPER

PATHOLOGY/BIOLOGY

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Animal-Related Fatalities—Part II: Characteristic Autopsy Findings and Variable Causes of Death Associated with Envenomation, Poisoning, Anaphylaxis, Asphyxiation, and Sepsis

ABSTRACT: In addition to blunt and sharp trauma, animal-related fatalities may result from envenomation, poisoning, anaphylaxis, asphyxiation, and sepsis. Although the majority of envenomation deaths are caused by hornets, bees, and wasps, the mechanism of death is most often anaphylaxis. Envenomation resulting from the injection of a poison or toxin into a victim occurs with snakes, spiders, and scorpions on land. Marine animal envenomation may result from stings and bites from jellyfish, octopus, stonefish, cone fish, stingrays, and sea snakes. At autopsy, the findings may be extremely subtle, and so a history of exposure is required. Poisoning may also occur from ingesting certain fish, with three main forms of neurotoxin poisoning involving ciguatera, tetrodotoxin ingestion, and paralytic shellfish poisoning. Asphyxiation may follow upper airway occlusion or neck/chest compression by animals, and sepsis may follow bites. Autopsy analysis of cases requires extensive toxinological, toxicological, and biochemical analyses of body fluids.

KEYWORDS: forensic science, animal injuries, envenomation, poisoning, anaphylaxis, asphyxiation, sepsis, autopsy

Animal-related deaths involve a wide spectrum of injuries and lethal mechanisms. In Part I of this series (1), deaths because of blunt and sharp trauma were reviewed. In the second part, deaths because of other causes are analyzed. This includes envenomation, poisoning, anaphylaxis, asphyxiation, and sepsis.

Envenomation

A venom can be defined as a poison or toxin that is injected by some means into a victim, and envenomation as the injection of these substances. In Australia, encounters with venomous plants and animals accounted for 0.1 deaths per 100,000 of the population between 1998 and 2002 (2). In the United States between 1991 and 2001, a total of 1943 people died after encounters with venomous and nonvenomous animals, representing an average of 177 deaths per year. Of these fatalities, venomous animals caused 39% of deaths, 70.2% of which were caused by hornets, bees, and wasps (3). Envenomation is a more significant problem globally, with between 400,000 and 2 million snake attacks reported per year resulting in 20,000–100,000 deaths. This may be an underestimation, however, as not all envenomations may be reported, particularly if they occur in remote and rural locations. The highest rates are in South Asia, Southeast Asia, and sub-Saharan Africa (4,5). There are only two lizards recognized as being venomous to humans (the Gila monster and beaded lizard); however, studies have revealed that other lizards possess venomous glands (6).

A wide range of venomous snakes exist, both on land and in the sea (7). In Australia, approximately 1000–3000 people are bitten annually resulting in approximately two deaths (8). In the United States, there are approximately 8000 people bitten by venomous snakes with five to six deaths per year (3); with nine of 16 deaths between 2001 and 2005 being attributed to rattlesnakes (9). Bites most commonly occur in the spring and summer and involve young male victims aged from 20 to 30 years, with alcohol intoxication (blood levels more than 0.1%) (10). Evenomation can occur after the apparent death of an animal and even following decapitation (11).

There is a range of toxins in snake venom with differences between species. Typical effects are the production of a hemorrhagic state (through the actions of proteases and/or endothelial damage), hypotension, paralysis, renal failure, and muscle necrosis (12–15). Many variables affect the severity of envenomation. The clinical response may depend on the type of snake and its age and size, and also on the age, size, and current health status of the victim. Symptoms may take a number of hours to develop. This occurs with venom that is primarily neurotoxic which often results in only mild pain at the site of the bite (16). The time taken to administer antivenom may significantly affect the rate of progression of symptoms and signs (17). The principle underlying the use of antivenom involves antibodies to venom components that neutralize their actions (18). Although it is the only effective treatment, it is not without the risk of inducing allergic-type reactions, including anaphylaxis (19). Thus, if death occurs following antivenom

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treatment, it may not be possible to determine whether the fatality was caused by the toxic effects of the venom or to the consequences of therapy (15).

The local effects of snake bite may be minimal with no signs at autopsy other than puncture wounds (20), which can be easily overlooked if not suspected at the time of examination. However, local swelling at the bite site may occur, which is more common is some species such as the mulga snake (21). Other autopsy findings are similarly usually unrewarding, with no specific changes (20,22,23). A high degree of suspicion is, therefore, often required to detect cases. Snake bite in pregnancy may result in loss of both fetus and mother. Fetal death may occur with maternal survival, as was the case of a woman bitten by a viper at 33 weeks gestation (24). Overall fetal mortality rates of 43–58% have been reported (25). The outcome for the fetus in cases of envenomation from other animals during pregnancy is not known (26). If snake bite is suspected, photography of possible bites with a scale should be undertaken. The putative bite can be swabbed or excised and stored dry in a refrigerator with draining lymph nodes, serum, and urine for venom testing.

Spider bites also pose a risk to humans, with problems ranging from neurological disturbances following Black widow spider bites, to local tissue necrosis following bites from Brown Recluse spiders (27–29). Black widow spiders inhabit North America with only the female of the species being able to penetrate mammalian skin. Certain animal species such as horses, camels, and cats are particularly sensitive to Black widow spider venom. These spiders can control the amount of venom injected per bite, resulting in 15% of human bites being nonenvenomating (27). Macroscopically, there may be little evidence of a bite, although there may have been a history of severe muscle cramps (27,30). Rarely, Black widow bites can cause myocarditis, which can be fatal (31). Myocarditis has also been documented after a Katipo spider bite in New Zealand (32).

Black widow spider toxicity varies geographically, with two deaths reported in Albania in 2006 (33). In other areas of the world, deaths may result from different species. For example, in Australia, 13 deaths have been attributed to funnel-web spider bites, of which seven were in children. The increased vulnerability of children most likely relates to the greater venom load per kilogram body weight (34). Armed spiders in South America may also be lethal, particularly to children and the elderly (35). Lethality from spider bites has reduced considerably since the introduction of antivenom, for example, no fatalities from Redback spider bites have occurred in Australia since the 1950s, and no deaths from funnel web bites since the 1980s (36–38).

Scorpion venom contains a complex mixture of neurotoxic peptides and enzymes that may cause autonomic nervous system hyperactivity, on occasion with fatal respiratory and cardiac failure (39). Toxicity again varies with geographic locations, with studies in Australia demonstrating no deaths or major systemic symptoms following envenomation in that country, in contrast to other parts of the world (40).

In Australia, envenomation by marine animals poses a more serious risk. Box jellyfish found off the coast of Northern Queensland were responsible for the deaths of two children between 2000 and 2003. Stings can rapidly lead to hypotension, respiratory distress, and hypotension, with death reported within 60 sec (41) because of the effects of a myocardial toxin that also has neuromuscular blocking effects. Stings can also leave large scars should the victim survive, because of the effects of a dermatonecrotic toxin (42). The toxin is delivered by stinging cells (nematocysts), which can be blocked by topical application of acetic acid (vinegar). Despite having been termed the world's most venomous animal, most encounters do not result in significant harm (43). Contact can, however, result in Irukandji syndrome, which refers to life-threatening hypertension with intracranial hemorrhage and supraventricular tachycardia following stings from a range of tropical jellyfish (44,45). The mechanism of this syndrome is uncertain (43). The Portuguese Man-of-War is a jellyfish with long stinging tentacles that are difficult to see in the water. Stings are acutely painful, with tentacles that have been washed up on beaches still able to sting many months after becoming detached. Exposure to a large number of stings may be fatal (41,44,45).

The blue-ringed octopus native to the Indian and Pacific Oceans has a potent neurotoxic venom, with an often painless sting. The animals live in coastal rock pools and can cause a victim to become rapidly incapacitated with death from flaccid muscle paralysis and respiratory failure (46,47). Stonefish may also be encountered in shallow waters around Australia and are the only marine life with venom used purely for defense; however, they are considered the world's most dangerous stinging fish and pose a considerable risk to anyone stepping on their spines (48,49). Cone shells are venomous Indo-Pacific marine snails found in shallow tropical waters that may cause painful stings with reports of approximately 15 deaths (46,48).

As was described in Part I (1), deaths caused by stingrays are most often the result of penetrating injury, although envenomation may rarely occur. The venom is contained within a sheath that covers the tail spine that tears after penetration of the victim's tissues. Toxins include serotonin, 5′-nucleotidase, and phosphodiesterase with effects including severe local pain, seizures, hypotension, arrhythmia, and death (50,51). Parts of the sheath sometimes with a portion of broken barb may remain in the wound causing infection. However, as many rays have lost or torn this sheath, there may not be significant amounts of stored venom to present a danger to humans (52). A rare case of myocardial necrosis was caused by the venom in a young boy who sustained a wound to the heart (53).

Sea snakes are found in all oceans with a concentration in tropical and subtropical waters. Although <10% of sea snakes inject venom, it is estimated that 150 deaths occur per year from envenomation, mainly in countries in the Indo-Pacific region (46).

Poisoning

As opposed to envenomation, which is usually an active process requiring an attack by an animal to inject toxin, poisoning is a passive process in which the animal produces a poison that is absorbed by the victim—usually by ingestion. The ingestion of a variety of animal substances may result in symptoms of poisoning. This is particularly so for certain varieties of fish with three main forms of neurotoxin poisoning involving ciguatera, tetrodotoxin ingestion, and paralytic shellfish poisoning. It is estimated that 1200 cases of seafood poisoning per 100,000 population occur around the Pacific each year (54).

Ciguatera is the most common and is rarely fatal. It results from the ingestion of finfish that contain ciguatoxins, which are neurotoxins (55) that have been ingested while feeding on algae or smaller fish. The viscera of the fish are the most toxic parts and symptoms include vomiting, diarrhea, and abdominal cramps (56,57). Although it is estimated that there are 10,000–50,000 case of ciguatera poisoning worldwide annually, fatal cases are very rare (58,59).

Puffer fish poisoning results from the ingestion of tetrodotoxin and is the most common lethal marine poisoning. The neurotoxin interferes with sodium channels, blocking the action potential of neuronal transmission; it is both water and heat stable. Puffer fish is a delicacy in countries such as Japan where 100 fugu-related

deaths per year were reported in the first part of last century. Death results from rapidly developing paralysis with hypotension and respiratory failure. There is no antidote and treatment is supportive (54,60,61).

Paralytic shellfish poisoning is the most common form of shellfish poisoning and follows the ingestion of oysters, mussels, or clams contaminated with toxic marine microalgae dinoflagellates. Death may result from respiratory failure. Other types of shellfish poisoning include neurotoxic and encephalopathic forms (54). The autopsy diagnosis may depend on a clear history of signs and symptoms of poisoning following ingestion.

Scombroid fish poisoning results from eating fish such as tuna and mackerel that have elevated levels of histamine because of storage at high temperatures. The resultant tachycardia, diarrhea, and headache are usually transitory although fatalities have occurred, usually in the presence of co-existent disease (62–64).

Anaphylaxis

Anaphylaxis is a potentially fatal type I hypersensitivity reaction caused by immunoglobulin E (IgE)-mediated sensitivity to animal venom resulting in the release of chemical mediators from basophils and tissue mast cells (65). Although anaphylaxis has been reported without previous exposure to a specific allergen (66), it is likely that allergic reaction to venoms results from prior contact, which may occur by inhalation or mucosal contact (67,68). The majority of deaths caused by ants, bees, and wasps are attributed to anaphylaxis, as opposed to the direct toxic effects of venom. Multiple stings are usually required to cause death by envenomation. Rarely anaphylaxis may follow envenomation by lizards such as the Gila monster and the Mexican beaded lizard (69); it can also follow tick bites (70). In addition to anaphylaxis to venom, reactions may occur following the ingestion of seafood (71).

At autopsy, the findings may be nonspecific and not particularly helpful in establishing the diagnosis, although there may be evidence of a sting, cutaneous urticaria and swelling, upper airway edema, and hyperinflation of the lungs with mucus plugging. Swelling of the upper airways may be limited to the oropharynx, nasopharynx, epiglottis, larynx, or upper trachea, or it may be generalized. There are no specific macroscopic or microscopic postmortem features, usually due to the rapidity with which death associated with anaphylaxis occurs (72–76). Deaths may also be contributed to by underlying significant disease such as coronary artery atherosclerosis (77).

The most useful tests to support a diagnosis of lethal anaphylaxis are measurements of allergen-specific IgE and serum tryptase. A tryptase level of 10 μg/L or greater has been found to be a sensitive (86%) and specific (88%) marker for individuals suffering from anaphylaxis, although it has been suggested that postmortem levels should be higher for the diagnosis to be made. The diagnosis of fatal anaphylaxis usually relies on a history of a bite or sting followed by dyspnea and collapse in a sensitized individual (65).

An Australian study from 1980 to 1999 identified six cases of fatal ant sting (78). Victims are often aware of an allergy to the venom but have neglected to carry injectable epinephrine with them. Of the approximately 8800 species of ants worldwide, Australia is home to 2750; however, most of these are not harmful. The ants that most commonly cause death are the ''jumper'' and ''bulldog'' types (78). Within the United States, fire ants pose a large problem as they attach to a victim and repeatedly sting. At least 44 deaths in the last 50 years have been attributed to these types of ants. At autopsy, anti-fire ant venom IgE may be demonstrated (79).

Bees and wasps pose a significant problem to those with allergies. Langley found that of 39 insect sting fatalities, 16 were attributable to bees and eight to wasps; one victim sustained over 300 bee stings (64). Of seven cases of fatal wasp stings, all were in rural settings involving farmers in four cases (80). Death may not always be due to anaphylaxis as asphyxia may develop if there has been a sting involving the upper airway.

Asphyxiation and Suffocation

A variety of animals have caused respiratory compromise owing to significant narrowing of the upper aerodigestive tract. As noted above, any sting that causes mucosal edema may reduce the diameter of major airways. An alternate mechanism involves foreign body inhalation and this has occurred with several types of small fish. A particular scenario occurs when a fisherman places a fish in his mouth for holding purposes to free up both hands, and the fish wriggles free and is inhaled (81–83). In other reports, fish have accidentally entered the mouth and obstructed the airway, or an individual has choked while trying to swallow a large fish (84–86). On occasion, the fish may still be alive when extracted (83).

An alternative form of airway narrowing may occur when leech-infested water has been drunk. Although initially small, continued ingestion of blood by the leech will result in considerable distention with resultant narrowing of the airway. Leeches within the nasal cavity or mouth may present with epistaxis or with spitting of blood; however, when lodged within the airway, there may be stridor, respiratory distress, and cyanosis (87–90). Significant anemia may also result from the continued presence of these parasites (91).

External covering of the mouth and nose by a sleeping cat may result in smothering in infants (92,93). An unusual variation of café coronary syndrome involved a psychotic 39-year-old woman who was found dead having consumed her pet cat, with her upper airway occluded by feline adipose tissue and a kidney (94).

External compression of the chest may occur with crush asphyxia because of large animals such as elephants. In addition, lethal asphyxia has been reported from pressure on the neck from an elephant trunk (95) and from constriction by a pet python in a 21-month-old boy (96). Ligature strangulation with other injuries has resulted from ropes that were tied to cows accidentally wrapping around the neck and tightening (97,98).

Sepsis

Animal bites and stings may transmit a wide variety of microorganisms that may lead to significant local or disseminated sepsis. Not all animal bites have the same rate of sepsis with some of the most dangerous bites arising from Komodo dragons and camels (99). There is always the risk of tetanus in those who have not been immunized (100). There is a higher rate of infection following cat bites compared with dogs (101,102), due possibly to the slender nature of cat teeth leading to a higher proportion of puncture wounds (103). High-risk individuals are those with diabetes mellitus or who are immunocompromised (99). For example, those with asplenia or hyposplenia are at risk of overwhelming sepsis following minor trauma, as was the case of death caused by disseminated *Capnocytophaga canimorsus* infection shortly after a minor dog bite in a man who had undergone previous splenectomy following a fall (104).

Rabies is a viral infection transmitted by bites from infected animals such as dogs and bats (105). After the initial bite, there is a latent period that can last for many months during which time the person feels well (106). Once the victim becomes unwell, there

is steady decline over a number of days, terminating in coma and death (105,106). Negri bodies may be found with a near absence of inflammation in the brain (107). Rabies inoculation without post-exposure prophylaxis was thought to be universally fatal; however, survival may rarely occur (108). Rabies has also been transmitted in transplant organs, from a donor who was subsequently shown to have had the disease (109). A range of other diseases are transmitted by insect bites including malaria, plague, and various types of encephalitis (110). Diseases may also be contracted by eating raw meat, such as eosinophilic meningitis caused by angiostrongyliasis following ingestion of live leopard slugs (111).

Consumption of animal (including fish) flesh is not without its risks. In addition to anaphylaxis (71) as noted earlier, infection may be acquired resulting in food poisoning (112), hydatid disease (113), trichinella (114), and spongiform encephalopathy (115,116). In rare cases, bites from poisonous animals may result in death from infection rather than envenomation (117).

Conclusion

This review has demonstrated the wide range of situations that may be identified at autopsy in deaths related to animal activity. In addition to blunt and sharp trauma, which would be regarded as more common (reviewed in Part I), deaths may be related to envenomation, poisoning, anaphylaxis, asphyxiation, and sepsis. Deaths may not occur immediately after contact with the offending animal but may be delayed for considerable periods, even months. At autopsy, the macroscopic findings may be extremely subtle, and so an adequate history detailing exposure to a particular animal is required. Pathologists working in a medical examiner system are required to determine manner of death (accident, homicide, suicide, or undetermined) (118). There are no guidelines with regard to deaths related to encounters with animals. The National Association of Medical Examiners requires death to have occurred at the hands of another person for the manner to be judged as homicide, and a self-inflicted act intended to take life for a determination of suicide. Although a suicide by self-injection of snake venom has been recorded (119), in most deaths it may be unclear how these should be classified (e.g., as natural or accidental). Consideration of the circumstances may be required, possibly necessitating a scene examination, in addition to full postmortem examination.

References

1. Bury D, Langlois NEI, Byard RW. Animal-related fatalities—Part I: characteristic autopsy findings and variable causes of death associated with blunt and sharp trauma. J Forensic Sci 2011. In press. doi: 10.1111/j.1556-4029.2011.01921.x.
2. Australian Bureau of Statistics. Deaths from external causes 1998–2002. Australian Bureau of Statistics, 2004, http://www.abs.gov.au/ausstats/abs@.nsf/mediareleasesbytitle/5B78E9004550E0BECA256E31007ED39A?OpenDocument (accessed April 25, 2010).
3. Langley RL. Animal-related fatalities in the United States—an update. Wilderness Environ Med 2005;16(2):67–74.
4. Kasturiratne A, Wickremasinghe AR, de Silva N, Gunawardena NK, Pathmeswaran A, Premaratna R, et al. The global burden of snakebite: a literature analysis and modelling based on regional estimates of envenoming and deaths. PLoS Med 2008;5(11):e218.
5. Hayes WK, Mackessy SP. Sensationalistic journalism and tales of snakebite: are rattlesnakes rapidly evolving more toxic venom? Wilderness Environ Med 2010;21:35–45.
6. Fry BG, Vidal N, Norman JA, Vonk FJ, Ramjan SFR, Kuruppu S, et al. Early evolution of the venom system in lizards and snakes. Nature 2006;439:584–8.
7. van den Enden E. Bites by venomous snakes. Acta Clin Belg 2003;58:350–5.
8. White J. Envenoming and antivenom use in Australia. Toxicon 1998;36:1483–92.
9. Langley RL. Animal bites and stings reported by United States poison control centers, 2001–2005. Wilderness Environ Med 2008;19(1):7–14.
10. Wingert WA, Chan L. Rattlesnake bites in Southern California and rationale for recommended treatment. West J Med 1988;148:37–44.
11. Suchard JR, LoVecchio F. Envenomation by rattlesnakes thought to be dead. N Engl J Med 1999;340:1930.
12. Mebs D, Ownby CL. Myotoxic components of snake venoms: their biochemical and biological activities. Pharmacol Ther 1990;48:223–36.
13. Fox JW, Serrano SMT. Exploring snake venom proteomes: multifaceted analyses for complex toxin mixtures. Proteomics 2008;8:909–20.
14. Cruz LS, Vargas R, Lopes AA. Snakebite envenomation and death in the developing world. Ethn Dis 2009;19(1 Suppl. 1):S1-42–6.
15. Warrell DA. Snake bite. Lancet 2010;375:77–88.
16. Peterson ME. Snake bite: coral snakes. Clin Tech Small Anim Pract 2006;21(4):183–6.
17. Peterson ME. Snake bite: pit vipers. Clin Tech Small Anim Pract 2006;21(4):174–82.
18. Chippaux J.-P. Snake-bites: appraisal of the gobal situation. Bull World Health Organ 1998;76:515–24.
19. Ibister GK, Brown SG, MacDonald E, White J, Currie BJ. Current use of Australian snake antivenoms and frequency of immediate-type hypersensitivity reactions and anaphylaxis. Med J Aust 2008;188:473–6.
20. White J, Pounder DJ. Fatal snakebite in Australia. Am J Forensic Med Pathol 1984;5(2):137–43.
21. Currie BJ. Snakebite in tropical Australia: a prospective study in the ''Top End'' of the Northern Territory. Med J Aust 2004;181:693–7.
22. Sutherland SK. Deaths from snake bite in Australia, 1981–1991. Med J Aust 1992;157(11–12):740–6.
23. Sutherland SK, Leonard RL. Snakebite deaths in Australia 1992–1994 and a management update. Med J Aust 1995;163(11–12):616–8.
24. Pant HP, Poudel R, Dsovza V. Intrauterine death following green tree viper bite presenting as antepartum hemorrhage. Int J Obstet Anesth 2009;19(1):102–3.
25. Pantanowitz L, Guidozzi F. Management of snake and spider bite in pregnancy. Obstet Gynecol Surv 1996;51(10):615–20.
26. Langley RL. A review of venomous animal bites and stings in pregnant patients. Wilderness Environ Med 2004 Fall;15(3):207–15.
27. Peterson ME. Black widow spider envenomation. Clin Tech Small Anim Pract 2006;21(4):187–90.
28. Peterson ME. Brown spider envenomation. Clin Tech Small Anim Pract 2006;21(4):191–3.
29. Andersen RJ, Campoli J, Johar SK, Schumacher KA, Allison EJ Jr. Suspected brown recluse envenomation: a case report and review of different treatment modalities. J Emerg Med 2011;41(2):e31–7.
30. Rogers JJ, Stanford C, Dart RC. The use of visual analog pain scales in black widow spider envenomation. J Med Toxicol 2006;2(1):46–7.
31. Sari I, Zengin S, Davutoglu V, Yildirim C, Gunay N. Myocarditis after black widow spider envenomation. Am J Emerg Med 2008;26(5):630.e1–3.
32. Crook R, Harrison N, Gibbons D. Myocarditis following Katipo spider bite. N Z Med J 2010;123(1314):119–21.
33. Hoxha R. Two Albanians die from black widow spider bites. BMJ 2006;333(7562):278.
34. Isbister GK. Prospective cohort study of definite spider bites in Australian children. J Paediatr Child Health 2004;40(7):360–4.
35. Vetter RS, Isbister GK. Medical aspects of spider bites. Annu Rev Entomol 2008;53:409–29.
36. Braitberg G, Segal L. Spider bites—assessment and management. Aust Fam Physician 2009;38(11):862–7.
37. Nicholson GM, Graudins A, Wilson HI, Little M, Broady KW. Arachnid toxinology in Australia: from clinical toxicology to potential applications. Toxicon 2006;48(7):872–98.
38. Nimorakiotakis B, Winkel KD. The funnel web and common spider bites. Aust Fam Physician 2004;33(4):244–51.
39. Petricevich VL. Scorpion venom and the inflammatory response. Mediators Inflamm 2010;2010:903295.
40. Isbister GK, Volschenk ES, Balit CR, Harvey MS. Australian scorpion stings: a prospective study of definite stings. Toxicon 2003;41(7):877–83.
41. McGoldrick J, Marx JA. Marine envenomations Part 2: invertebrates. J Emerg Med 1992;10:71–7.

42. Holmes JL. Marine stingers in far North Queensland. Aust J Dermatol 1996;37(Suppl. 1):S23–6.
43. Bailey PM, Little M, Jelinek GA, Wilce JA. Jellyfish envenoming syndromes: unknown toxic mechanisms and unproven therapies. Med J Aust 2003;178:34–7.
44. Nimorakiotakis B, Winkel KD. Marine envenomations. Part 1—jellyfish. Aust Fam Physician 2003;32(12):969–74.
45. Auerbach PS. Envenomations from jellyfish and related species. J Emerg Nurs 1997;23(6):555–65, quiz 66-7.
46. Fenner PJ. Dangers in the ocean: the traveler and marine envenomation. II. Marine vertebrates. J Travel Med 1998;5(4):213–6.
47. Cavazzoni E, Lister B, Sargent P, Schibler A. Blue-ringed octopus (*Hapalochlaena* sp.) envenomation of a 4-year-old boy: a case report. Clin Toxicol (Phila) 2008;46(8):760–1.
48. Nimorakiotakis B, Winkel KD. Marine envenomations. Part 2—other marine envenomations. Aust Fam Physician 2003;32(12):975–9.
49. Brenneke F, Hatz C. Stonefish envenomation—a lucky outcome. Travel Med Infect Dis 2006;4(5):281–5.
50. Kizer KW. Marine evenomations. J Toxicol Clin Toxicol 1983;21: 527–55.
51. McGoldrick J, Marx JA. Marine envenomations; Part 1: vertebrates. J Emerg Med 1991;9:497–502.
52. Russell FE, Panos TC, Kang LW, Warner AM, Colket TC III. Studies on the mechanism of death from stingray venom; a report of two fatal cases. Am J Med Sci 1958;235(5):566–84. passim.
53. Fenner PJ, Williamson JA, Skinner RA. Fatal and non-fatal stingray envenomation. Med J Aust 1989;12:621–5.
54. Isbister GK, Kiernan MC. Neurotoxic marine poisoning. Lancet Neurol 2005;4(4):219–28.
55. Burnett JW, Weinerich D, Williamson JA, Fenner PJ, Lutz LL, Bloom DA. Autonomic neurotoxcity of jellyfish and marine animal venoms. Clin Auton Res 1998;8:125–30.
56. Dickey RW, Plakas SM. Ciguatera: a public health perspective. Toxicon 2010;56:123–6.
57. Friedman MA, Fleming LE, Fernandez M, Bienfang P, Schrank K, Dickey R, et al. Ciguatera fish poisoning: treatment, prevention and management. Mar Drugs 2008;6(3):456–79.
58. Lehane L, Lewis RJ. Ciguatera: recent advances but the risk remains. Int J Food Microbiol 2000;61(2–3):91–125.
59. Hamilton B, Whittle N, Shaw G, Eaglesham G, Moore MR, Lewis RJ. Human fatality associated with Pacific ciguatoxin contaminated fish. Toxicon 2010;56(5):668–73.
60. Lange WR. Puffer fish poisoning. Am Fam Physician 1990;42:1029–33.
61. Ahasan HANN, Mamun AA, Karim SR, Bakar MA, Gazi EA, Bala CS. Paralytic complications of puffer fish (tetrodotoxin) poisoning. Singapore Med J 2004;45:73–4.
62. Davis J, Henry SA, Rowland J, Ripley D, Jacobson G, Brunkard JM, et al. Scombroid fish poisoning associated with tuna steaks—Louisiana and Tennessee, 2006. MMWR Morb Mortal Wkly Rep 2007;56(32):817–9.
63. Guly HR, Grant IC. Case of the month: lesson of the week: don't forget scombroid. Emerg Med J 2006;23(12):955–6.
64. Langley RL. Fatal animal attacks in North Carolina over an 18-year period. Am J Forensic Med Pathol 1994;15(2):160–7.
65. Riches KJ, Byard RW. The detection of fatal anaphylaxis at autopsy—an overview. Scand J Forensic Sci 2004;10:61–3.
66. Rancé F, Bidat E, Biurrier T, Sabourand D. Cashew allergy: observations of 42 children without associated peanut allergy. Allergy 2003;58:1311–4.
67. de Medeiros CR, Barbaro KC, de Siqueira FO, Zanotti FAP, Castro FFM. Anaphylaxis reaction secondary to *Bothrops* snakebite. Allergy 2008;63:242–9.
68. de Medeiros CR, Barbaro KC, Lira MS, França FOS, Zaher VL, Kokron CM, et al. Predictors of Bothrops jararaca venom allergy in snake handlers and snake venom handlers. Toxicon 2008;51:672–80.
69. Cantrell FL. Envenomation by the Mexican beaded lizard: a case report. J Toxicol Clin Toxicol 2003;41(3):241–4.
70. Brown AFT, Hamilton DL. Tick bite anaphylaxis in Australia. J Accid Emerg Med 1998;15:111–3.
71. Lopata AL, Lehrer SB. New insights into seafood allergy. Curr Opin Allergy Clin Immunol 2009;9:270–7.
72. Ansari MQ, Zamora JL, Lipscomb MF. Postmortem diagnosis of acute anaphylaxis by serum tryptase analysis. Am J Clin Pathol 1993;99:101–3.
73. Pumphrey RS, Roberts IS. Postmortem findings after fatal anaphylactic reactions. J Clin Pathol 2000;53(4):273–6.
74. Roberts ISD, Pumphrey RSH. Diagnosing anaphylaxis at autopsy. Cell Pathol 2001;3:136–8.
75. Fineschi V, Cecchi R, Centini F, Reattelli LP, Turillazzi E. Immunohistochemical quantification of pulmonary mast-cells and post-mortem blood dosages of tryptase and eosinophil cationic protein in 48 heroin-related deaths. Forensic Sci Int 2001;120:189–94.
76. Low I, Stables S. Anaphylatic deaths in Auckland, New Zealand: a review of coronial autopsies from 1985 to 2005. Pathology 2006;38:328–32.
77. Brown SG, Wu QX, Kelsall GR, Heddle RJ, Baldo BA. Fatal anaphylaxis following jack jumper ant sting in southern Tasmania. Med J Aust 2001;175(11–12):644–7.
78. McGain F, Winkel KD. Ant sting mortality in Australia. Toxicon 2002;40(8):1095–100.
79. Prahlow JA, Barnard JJ. Fatal anaphylaxis due to fire ant stings. Am J Forensic Med Pathol 1998;19(2):137–42.
80. McGain F, Harrison J, Winkel KD. Wasp sting mortality in Australia. Med J Aust 2000;173(4):198–200.
81. Pinheiro J, Cordeiro C, Vieira DN. Choking death on a live fish (Dicologoglossa cuneata). Am J Forensic Med Pathol 2003;24(2): 177–8.
82. Rajendran KM, Satyanand S. An unusual case of live fish obstructing the airway. A case report. Br J Anaesth 1966;38(6):492–3.
83. Khan AA. Live fish in the pharynx. J Laryngol Otol 1969;83(11):1119–22.
84. Pritchard JR. Sudden death by Lepomis macrochirus (the killer fish). Am J Forensic Med Pathol 1989;10(3):245–6.
85. Lowis RM. [Foreign body in pharynx, live fish]. Br Med J 1947; 2(4518):211.
86. Deidiker R. Return of the killer fish: accidental choking death on a bluegill (Lepomis macrochirus). Am J Forensic Med Pathol 2002;23(2):197–8.
87. Labadi MH, Jamal MN. Leeches in the larynx. J Laryngol Otol 1997;111(10):980–1.
88. Mohammad Y, Rostum M, Dubaybo BA. Laryngeal hirudiniasis: an unusual cause of airway obstruction and hemoptysis. Pediatr Pulmonol 2002;33(3):224–6.
89. Kaygusuz I, Yalcin S, Keles E. Leeches in the larynx. Eur Arch Otorhinolaryngol 2001;258(9):455–7.
90. Pandey CK, Sharma R, Baronia A, Agarwal A, Singh N. An unusual cause of respiratory distress: live leech in the larynx. Anesth Analg 2000;90(5):1227–8.
91. Cundall DB, Whitehead SM, Hechtel FO. Severe anaemia and death due to the pharyngeal leech Myxobdella africana. Trans R Soc Trop Med Hyg 1986;80(6):940–4.
92. Karkola K, Mottonen M, Raekallio J. Deaths caused by animals in Finland. Med Sci Law 1973;13(2):95–7.
93. Heaton PA, Sage MD. Fatal smothering by a domestic cat. N Z Med J 1995;108(994):62–3.
94. Redpath M, Sauvageau A. An unusual case of smothering secondary to ingesting raw pet cat. Am J Forensic Med Pathol 2011;32(2):190–2.
95. Begoian AG. Death from compression of the neck by an elephant's trunk. Sud Med Ekspert 1979;22(2):56–7.
96. McCarty VO, Cox RA, Haglund B. Death caused by a constricting snake—an infant death. J Forensic Sci 1989;34(1):239–43.
97. Verma SK, Aggarwal NK, Kohli A. Accidental ligature strangulation deaths in East Delhi (India). Med Sci Law 2005;45(1):47–51.
98. Mitrani I. Accidental strangulation. J Forensic Med 1962;9:20–4.
99. Morgan M. The microbiology of animal bites. Bull R College Pathol 2004;128:16–9.
100. Fleisher GR. The management of bite wounds. N Engl J Med 1999;340:138–40.
101. Aghababian RV, Conte JE Jr. Mammalian bite wounds. Ann Emerg Med 1980;9:79–83.
102. Talan DA, Citron DM, Abrahamian FM, Morgan GJ, Goldstein EJC. Bacteriologic analysis of infected dog and cat bites. N Engl J Med 1999;340:138–40.
103. Dire DJ. Cat bite wounds: risk factors for infections. Ann Emerg Med 1991;20:973–9.
104. Stiegler D, Gilbert JD, Warner MS, Byard RW. Fatal dog bite in the absence of significant trauma: *capnocytophaga canimorsus* infection and unexpected death. Am J Forensic Med Pathol 2010;31(2):198–9.
105. Warrell MJ, Warrell DA. Rabies and other lyssavirus diseases. Lancet 2004;363(9413):959–69.

106. Hanna JN, Carney IK, Smith GA, Tannenberg AE, Deverill JE, Botha JA, et al. Australian bat lyssavirus infection: a second human case, with a long incubation period. Med J Aust 2000;172(12):597–9.
107. Sandhyamani S, Roy S, Gode GR, Kalla GN. Pathology of rabies: a light- and electron-microscopical study with particular reference to the changes in cases with prolonged survival. Acta Neuropathol 1981;54:247–51.
108. Willoughby RE, Rotar MM, Dhonau HL, Ericksen KM, Cappozzo DL, Kazmierczak JJ, et al. Recovery of a patient from clinical rabies—Wisconsin, 2004. MMWR Morb Mortal Wkly Rep 2004;53(50):1171–3.
109. Srinivasan A, Burton EC, Kuehnert MJ, Rupprecht C, Sutker WL, Ksiazek TG, et al. Transmission of rabies virus from an organ donor to four transplant recipients. N Engl J Med 2005;352(11):1103–11.
110. Prentice MB, Rahalison L. Plague. Lancet 2007;369(9568):1196–207.
111. Senanayake SN, Pryor DS, Walker J, Konecny P. First report of human angiostrongyliasis acquired in Sydney. Med J Aust 2003; 179(8):430–1.
112. Fujioka M, Nishimura G, Miyazato O, Yamamoto T, Okamoto F, Tsunenori K. Necrotising fasciitis and myositis that orginated from gastrointestinal bacterial infection: two fatal cases. Scand J Plast Reconstr Surg Hand Surg 2003;37:239–42.
113. Byard RW. An analysis of possible mechanisms of unexpected death occurring in hydatid disease (echinococcosis). J Forensic Sci 2009;54:919–22.
114. Clausen MR, Meyer CN, Krantz T, Moser C, Gomme G, Kayser L, et al. *Trichinella* infection and clinical disease. Quart J Med 1996;89:631–6.
115. Collins S, McLean CA, Masters CL. Gerstmann–Sträussler–Scheinker syndrome, fatal familial insomnia, and kuru: a review of these less common human transmissible spongiform encephalopathies. J Clin Neurosci 2001;8:387–97.
116. Harman JL, Silva CJ. Bovine spongiform encephalopathy. J Am Vet Assoc 2009;234:59–72.
117. Singh S, Dass A, Jain S, Varma S, Bannerjee AK, Sharma BK. Fatal non-bacterial thrombotic endocarditis following viperine bite. Intern Med 1998;37:342–4.
118. Davis GG. Mind your manners. Part I: history of death certification and manner of death classification. Am J Forensic Med Pathol 1997;18:219–23.
119. Knight B, Barclay A, Mann R. Suicide by injection of snake venom. Forensic Sci 1977;10:141–5.

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