This article is intended as a general overview of snakebites, as presented in my lecture at the B.H.S. 1989 AGM.

Snakebite is largely a problem of the rural tropics. It is difficult to estimate the full scale of bites and deaths worldwide, but epidemiological studies are certainly interesting. In Surinam, the incidence of bites was found to be 45:100,000 in urban areas and 600:100,000 in rural areas. A similar figure was found in rural parts of Nigeria where snake-bite victims can occupy 10% of hospital beds. A study of the rural Waorani tribe in Equador showed that 78% of the population had venom antibodies, indicating having been bitten at some time, and that 4.9% of deaths were attributable to snakebites.

What makes a snake dangerous? One criterion is the toxicity of its venom. Secondly, the population densities of snakes and men in any area, and thereby the extent of snake:human contact, and thirdly, the habits and temperament of the species must be considered. A snake that tends to be readily flightable, reclusive or disinclined to bite, albeit potentially lethal, is less of a problem than another that holds its ground, relies on camouflage and is aggressive by nature. The World Health Organisation has advocated five species as “medically important”.

These are:–

- *Echis carinatus* (Carpet Viper) – Africa & Asia.
- *Vipera russelli* (Russell’s Viper) – Asia.
- *Bothrops atrox* (Fer-de-lance) – Central & South America.
- *Bitis arietans* (Puff Adder) – Africa.
- *Calloselasma rhodostoma* (Malayan pit viper) – South East Asia.

In addition, cobra bites are not uncommon in parts of Africa & Asia, and kraits may cause significant morbidity and mortality in parts of Asia. (77% mortality in a survey of 35 cases of *Bungarus caeruleus* bites in India).

The species that is probably responsible for the greatest number of bites worldwide is *Echis carinatus* throughout its large range across Africa & Asia and variable sub-types and morphs.

The pathological effects of snake venom can be generalised by family. Elapid venom is mainly neurotoxic (acting on nerves), vipersid venom is vasculo or haemotoxic (acting on blood vessels) and sea snake venom is myotoxic (acting on muscle). There are exceptions to these rules. For example, *Naja nigricollis* (Black-necked Spitting Cobra) venom has no neurotoxic effects, whereas *Crotalus durissus terrificus* (Tropical Rattlesnake) and *Bitis atropos* (Night Adder) venoms are primarily neurotoxic. Some Australian elapid venoms are haemotoxic or myotoxic. Venom contains many components which together produce a clinical picture of local features (at the site of...
bite) and systemic features (indicating widespread envenomation). Elapid venoms consist of small protein molecules which are absorbed into venous blood and spread into the body more rapidly than the larger proteins of viperid venom which tend to be absorbed via the lymphatic drainage. Human victims of snakebites do not collapse and die in minutes as some may believe. In fact, at least 50% of bites from potentially lethal species result in little or no envenomation. Fatal bites from elapids may lead to death in 5–20 hours, sea snakes in about 15 hours and vipers in 48 hours or longer.

Viper bites can produce local pain and swelling within minutes which may continue to increase with bruising and blistering for two to three days. This may either slowly resolve completely or leave local necrosis and gangrene. Systemic features include spontaneous bleeding and haemorrhage caused firstly by leakage from damaged blood vessels and secondly by non-clotting blood due to a deranged clotting mechanism. Death may be secondary to shock (loss of circulating fluid and low blood pressure) or haemorrhage into vital organs. Haemorrhage into the pituitary gland has been recognised following Russell’s Viper bite and leads to long-term morbidity from impaired control of the endocrine system. *Vipera berus* (European Adder) and *Bitis arietans* (Puff Adder) bites cause marked pain and swelling with limited systemic features, whereas systemic features are predominant in *Echis carinatus* (Carpet Viper) bites. Local and systemic features are variable with *Vipera russelli* and in addition conjunctival oedema and renal (kidney) failure are noted in Burma and rhabdo-myolysis (muscle breakdown) in Sri Lanka.

Gangrene can also occur with some elapid bites but local features are generally less than those of vipers. The appalling gangrene that often follows *Naja nigricollis* bites, is mostly a result of direct cytotoxicity (killing of cells), whereas that following viper bite is mainly secondary to local vessel damage. Neurotoxicity can present within 15 minutes of an elapid bite with weakness of the eyelids and throat muscles. This may extend to affect the respiratory musculature causing death from suffocation. The mechanism of poisoning is blockage of impulse transmission at the nerve-muscle junction caused by the venom toxin reversibly combining with the normal receptor but not inducing transmission. Krait bites, e.g. *Bungarus candidus*, have minimal local effects but may cause extensive paralysis and thus a high mortality rate.

Sea snakes are not common biters, usually only attacking fishermen when trapped in nets or tipped on board, but are worth mentioning as they represent the third venom type. A bite can cause some swelling but generalised muscle pain and stiffness occurs as muscle cells are destroyed (rhabdomyolysis). The consequential release of potassium ions may lead to cardiac arrest or arrhythmia and death.

PLATE 2. Bleeding gums: a systemic effect from the bite of *Echis carinatus*. 
Recommended first aid should employ the use of a compressive bandage rather than tourniquet, and to immobilise the bitten limb. The aim being to reduce the superficial venous flow and slow down absorption of venom without compromising the arterial supply. The wound should not be incised. Reassurance is obviously important in a frightened patient. The victim should be dispatched to hospital as soon as possible, preferably with the corpse of the offending snake for positive identification. Assessment in hospital for signs of systemic envenoming will decide when and if anti-venom (if available) is required. General supportive measures may also be required such as intravenous fluids for shock or artificial ventilation for respiratory paralysis. Further medical or surgical treatment may be needed in the recovery stage to prevent or treat infected wounds or debride necrotic tissue prior to healing by granulation or skin grafting.

ACKNOWLEDGEMENTS

I am grateful to Dr. R.D.G. Theakston of W.H.O. Collaborative Centre for Anti-venoms, Liverpool School of Tropical Medicine, for advice and use of slides for my lecture.

REFERENCES


