Bites and stings in travellers

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Summary
As a rule, bites and stings in travellers are merely a nuisance. But it is sensible to be informed about the more serious possibilities which can result. Systemic diseases can be transmitted, the skin lesions from insects can be troublesome and finally, some bites and stings can cause envenoming. Thus, the bather may be harmed by venomous fish stings, sea urchins, jellyfish and in Asian-Pacific waters by sea-snakes. Land hazards include bites or stings by scorpions, spiders, ticks, centipedes, bees, wasps, caterpillars and snakes. The main clinical features of such bites and stings, including treatment and prevention, are outlined.

Introduction
Few, if any, travellers will escape bites or stings, particularly if they travel to the tropics or sub-tropics. The catalogue of disasters associated with insect troublemakers should alone be enough to make one stay at home (Harman, 1971). Other common animals potentially noxious to the traveller include jellyfish, sea urchins, venomous fish, sea snakes, ticks, mites, scorpions, spiders, centipedes and land snakes. However, the traveller may take heart from statistics in Rhodesia where the annual death rate from road accidents is seventeen times that caused by animals (Castle, 1971). Moreover most of the deaths caused by animals were provoked by man rather than by the animal. Undoubtedly, the greatest animal danger to travellers is man (followed in Rhodesia by the crocodile). Bites and stings can harm the traveller, causing (1) trauma, physical and mental (fright), (2) transmission of systemic disease, (3) skin lesions, (4) envenoming.

Transmission of systemic disease
Systemic disease is the most serious potential harm to the traveller from bites and stings. Although the diseases in Table 1 would be acquired in the tropics and sub-tropics, symptoms often start after the traveller has returned home. The most important disease is falciparum malaria, dealt with in the previous paper.

Skin lesions
Insect bites are a world-wide scourge to travellers who can become rapidly sensitized so that firm itching papules form within 24 hr of each bite—usually multiple bites. After further exposure immediate weals may develop and subsequent bites may provoke both types of reaction indefinitely. Alternatively, reactions may decrease; first, the delayed and then the immediate responses are reduced or lost (Harman, 1971). For local treatment calamine lotion is suitable; antihistamines can cause sensitization and photo-sensitization and are not recommended. But if lesions are widespread, antihistamines by mouth are probably helpful, such as chlorpheniramine maleate tablets (Piriton) 4–16 mg daily. For more severe local reactions a steroid such as betamethasone valerate cream (Betnovate) may be needed—provided there is no sepsis or ulceration. Infection of scratched lesions must be discouraged, if necessary by a bandage. If infection has been established, systemic and local antibiotics may be needed.

Table 1. Diseases acquired by bites and stings

<table>
<thead>
<tr>
<th>Vector</th>
<th>Systemic disease transmitted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class INSECTA</td>
<td></td>
</tr>
<tr>
<td>Order DIPTERA</td>
<td></td>
</tr>
<tr>
<td>Mosquitoes</td>
<td>Malaria, filariasis, arboviral infections</td>
</tr>
<tr>
<td>Sandflies</td>
<td>Leishmaniasis, bartonellosis</td>
</tr>
<tr>
<td>Blackflies</td>
<td>Onchocerciasl</td>
</tr>
<tr>
<td>Midges</td>
<td>Filariais</td>
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<tr>
<td>Tsetse flies</td>
<td>African trypanosomiasis</td>
</tr>
<tr>
<td>Horseflies</td>
<td>Loaiasis, tularaemia</td>
</tr>
<tr>
<td>Order ANOPLURA</td>
<td></td>
</tr>
<tr>
<td>Lice</td>
<td>Typhus, Q fever</td>
</tr>
<tr>
<td>Order SIPHONOPTERA</td>
<td></td>
</tr>
<tr>
<td>Fleas</td>
<td>Plague, typhus</td>
</tr>
<tr>
<td>Order HEMIPTERA</td>
<td></td>
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<tr>
<td>Bugs</td>
<td>American trypanosomiasis</td>
</tr>
<tr>
<td>Class ARACHNIDA</td>
<td></td>
</tr>
<tr>
<td>Order ACARINA</td>
<td></td>
</tr>
<tr>
<td>Mites</td>
<td>Typhus</td>
</tr>
<tr>
<td>Soft ticks</td>
<td>Relapsing fever</td>
</tr>
<tr>
<td>Hard ticks</td>
<td>Typhus, arboviral infections</td>
</tr>
<tr>
<td>Dogs, bats, etc.</td>
<td>Rabies</td>
</tr>
</tbody>
</table>
Clothing helps to prevent insect bites especially round the ankles. A useful repellant is diethyltoluamide in a liquid evaporating base such as 70% ethanol. An insecticidal aerosol spray is worth taking on travels.

Myiasis

This is the condition produced by fly larvae invading living tissue. The Tumbu fly, Cordylobia anthropophaga, is widely distributed in Africa South of the Sahara. The female fly lays eggs on sand or soil contaminated by urine or excreta; larvae from the eggs can live 1–2 weeks in the soil before they become attached to a host. They burrow just under the skin, leaving their posterior breathing openings on a level with the surface. The lesions may appear only on return from travelling and resemble boils but do not throb (although they itch), and each has a small hole in the centre. The larvae grow in the skin for about 2 weeks then wriggle out to form a puparium. The adult flies emerge 10 days later; they resemble bluebottles except for a yellowish colour. Man may acquire the larvae from contaminated sand, clothes or bed linen. An adhesive occlusive dressing kills the larvae which in time are shed naturally from the skin (Radcliffe, 1972). Larvae of several other flies can cause similar lesions including subconjunctival lesions.

Chiggers

The chigger sandflea, Tunga penetrans, occurs in tropical America, Africa and India. The impregnated female flea burrows into the skin of the first warm-blooded animal she encounters (in man, usually the foot), and grows to the size of a small pea. A painful boil-like lesion with a central black dot results, and secondary infection is common. The best treatment is to enlarge the entrance orifice with a needle and enucleate the entire insect. Walking bare-footed should be avoided.

Venomous bites and stings

General considerations

Venomous creatures can secrete venom in a gland or in specialized cells, and can deliver this venom during biting or stinging. All venoms contain a complex mixture of toxins which can be experimentally fractionated into enzymes and polypeptides with widely varying biological and pharmacological effects. Readers interested in details of venoms should consult the admirable review by Minton (1974). The complex and sometimes conflicting experimental effects of venom fractions are not always relevant to the clinical problem of envenoming in man. Indeed, some experimental findings can mislead clinicians. In man, the important clinical effects of venomous bites and stings are fairly simple and may be divided into:

1. Fright effects are present in virtually all venomous bites and stings. Often there is fear that rapid, painful death is inevitable.
2. Venom effects may be summarized:
   (i) Local, mainly vasculotoxic (pain, swelling, sometimes necrosis).
   (ii) Systemic vasculotoxic (bleeding, sometimes shock).
   (iii) Systemic neurotoxic (autonomic effects; neuromuscular block of muscles for vision, swallowing, breathing).
   (iv) Systemic cardiotoxic or myotoxic effects (less commonly).

Systemic vasculotoxic effects are typical of vipers, snakebite poisoning, and the neuromuscular blocking effects are typical of elapid snake bite poisoning. Venoms of sea snakes and of some Australian elapid snakes are mainly myotoxic. The venom effects of bites and stings other than snake bite chiefly involve local vasculotoxic features, autonomic toxic effects, and sometimes cardiotoxic effects. If poisoning is very severe, acute renal failure can occur in most types of envenoming.

Although the effects of venomous bites and stings in man depend on the type of venom, a much more important factor is the amount of venom injected. Clinicians should realize that, although fatalities and serious poisoning can occur, more commonly (and fortunately) little or no envenoming results from venomous bites and stings in man. In the latter cases, fear often dominates the clinical picture.

Venomous bites and stings in the sea

Fish stings

Venomous fish stings are common in the tropics. In coastal waters where the sea bed is mud or sand, catfish occur—so called because they have whiskery mouthparts. They have serrated spines in their fins which can inject venom. Stingrays have a flat triangular-shaped body varying in diameter from a few cm to 3 m. When trodden on they whip forward their tail which has a serrated sting on it. In coral reefs, even more venomous fish are found such as stonefish and scorpion fish. They have multiple venomous stings along their backs. The main effects of stings by these fish is an intense and often agonizing local pain. Sometimes the sting becomes detached and remains embedded, leading to a chronic discharge. Local necrosis sometimes occurs with scorpion fish stings.

The most effective treatment for the local pain of venomous fish stings is hot water. The part stung is immersed in water as hot as the patient can bear. The pain is relieved within seconds and the part
stung must be quickly removed from the water to avoid blistering. It should be reimmersed as pain recurs (within seconds at first, later within minutes). This procedure should be continued until the pain no longer recurs (usually about 30 min). It is important to explain details of this simple but highly effective treatment to the patient, who should be given a can of water recently brought to boiling point. This should be added to the immersing water to keep it as constantly hot as he can bear. If the water is not hot enough, the treatment is not effective, but if the stung part is immersed too long in very hot water, blistering will result. If the part stung is unsuitable for immersion in hot water (for example, the face or trunk), the area should be infiltrated through the puncture wound with 2–5 ml of 1% lignocaine hydrochloride.

Sea urchins

Sea urchins are globular, cushion-shaped, or discoidal relatives of the starfish and are abundant in shallow tropical waters. They have numerous sharp, brittle, articulated spines, 3–30 cm long. The spines are used for locomotion, digging and self-protection, and although no venom has so far been demonstrated, the pain can far exceed that produced by mere mechanical injury. Sometimes general symptoms may follow in addition—giddiness, difficulty in breathing, weakness of lips, tongue, eyelids and limbs lasting up to 6 hr. If the spines are not removed they may cause sepsis and, after 1–3 months, small, painless granulomatous nodules may appear. Penetration of bone may cause cyst formation, and joint injury can provoke severe destructive arthritis.

Spines should be removed as soon as possible. Over superficially embedded spines the thick keratin of the sole of the foot is excised and 2% salicylic ointment is applied for 24–48 hr. The spines can then be expelled through the soggy skin by vigorous, and not too painful, squeezing. More deeply embedded spines should be removed surgically after local anaesthesia. Antiseptic dressings or antibiotics may be required for a few days until the wounds heal.

Jellyfish stings

Jellyfish have myriads of microscopic stinging capsules called nematocysts on their tentacles. When touched, these capsules extrude a sting which can eject venom. However, only a small number of jellyfish have stings which can penetrate intact human skin. The most dangerous jellyfish, the cubomedusan or box jellyfish, sometimes called sea-wasp, is confined to tropical waters. Having a cuboidal body or float, up to 20 cm in diameter, and a leash of several tentacles growing from each of the four body corners, it is translucent and difficult to see in the water. Sea-wasps of the family Chironex, dropidae, have been found in tropical waters of all continents in the world. Physalia, or the Portuguese man-o’-war jellyfish, has a coloured float from which numerous minor tentacles hang, together with a single main tentacle which can be very long, up to 30 m in length. Although it has an evil reputation, severe poisoning seldom follows the stings of the Portuguese man-o’-war, fatal stings being extremely rare. In contrast, several deaths following stings by the box jellyfish, Chironex fleckeri, have been recorded from Australian waters. Death is due to rapid collapse within a few minutes of the sting; these rapid deaths appearing to be more due to heart failure than to respiratory failure. If the victim survives for 30 min, death does not occur but the parts stung become swollen and, later, necrosis can ensue. A smaller cubomedusan causes the Irukandji sting, so-named after an Australian aboriginal tribe. Local symptoms are minimal, but after 10–20 min, violent generalized pains ensue, with restlessness and sweating. These symptoms may continue for 1–2 days. The Irukandji sting is never fatal.

Stings by most jellyfish other than sea-wasps cause only local wealing with tingling and discomfort, usually lasting a few hours. Only a small proportion (about 10–20%) of the nematocysts discharge their stings and venom, and this has important implications for treatment. Methylated spirits (or any other alcohol) should be applied to the stung parts to kill the undischarged nematocysts. If no alcohol is available, dry sand or any dry powder should be thrown on the sting and then the tentacles and slime should be scraped off. Dry sand is better than wet sand. The sting should not be rubbed with wet hands, cloth, and so on, as this will spread and aggravate the sting. After the spirits have dried, calamine lotion is a suitable local application. In severe cases, with rapid collapse, the victim should be lain on his back, methylated spirits poured on the sting, and tourniquets put on affected limbs. If breathing stops, mouth-to-nose artificial respiration should be given; if the heart stops, closed-chest cardiac massage should be carried out. A potent sea-wasp antivenom has recently become available from the Commonwealth Serum Laboratories, Australia, and its administration has resulted in dramatic recovery from severe poisoning by C. fleckeri stings (Baxter and Marr, 1974).

Sea snake bite

Sea snakes have very short fixed fangs and characteristic flat tails. They are common only in Asian and western Pacific coastal waters, and do not occur in the Atlantic ocean or the Mediterranean. Although sea snake venom is extremely toxic, one
ever, if rather than was 50% had trivial or no poisoning. However, if poisoning were present, the mortality rate was 50% until effective antivenom became available (Reid, 1975a). It is thus very important for the clinician to distinguish sea snake bite from sea snake bite poisoning.

The great majority of victims are fishermen bitten at their work, but occasionally bathers and skin divers are bitten if they tread on or handle sea snakes. Sea snake bites have no important local effects—there is no swelling or pain and the pinprick marks of the bite may be difficult or even impossible to make out. If venom is injected characteristic generalized myalgic pains start 30–60 min after the bite followed in a few hours by the red-brown colour of myoglobin in the urine. In severe poisoning the myalgic pains merge into paresis with inability to move the eyes, open the mouth, protrude the tongue or swallow. Respiratory insufficiency and hyperkalaemia follow (tall peaked T-waves in chest leads of the electrocardiogram). Fatal respiratory failure may occur within a few hours of the bite from inhalation of secretions or vomit. In other cases where respiratory failure is due to weakness of respiratory muscles, death is usually 12–24 hr after the bite. In the absence of effective antivenom, the myotoxic effects are prolonged and full recovery may take several months. In such cases, acute renal failure is the rule.

In treating sea snake bites, adequate reassurance is most important and a placebo injection should be promptly given unless clear signs of poisoning are already evident; in this event effective antivenom should be given. If used correctly antivenom can be effective even though not given until hours after the bite (Reid, 1962). Both tiger snake antivenom and sea snake antivenom (Commonwealth Serum Laboratories, Melbourne) are effective (Baxter and Gallichio, 1974). Antivenom (1000–10,000 u) should always be given by intravenous infusion, which is the most effective and the safest route. Details of antivenom administration and supportive treatment are given elsewhere (Reid, 1972b; 1975a).

Bathers in regions where sea snakes can occur should follow the example of fishermen and shuffle, rather than walk, on the sea bed. Bathers are quite safe swimming in the sea, and therefore should keep swimming as far as possible. Divers should ignore sea snakes and on no account attempt ‘sportingly’ to catch them. Amateur fishermen should treat sea snakes with great respect; although they may seem docile most of the time, it would be extremely foolish to rely on their reluctance to bite man. In some regions, sea snakes may be caught by amateur fishermen using a line and hook, in which case the line should be cut and the sea snake dropped into the water—without handling. If a sea snake finds its way by net or line into a boat, no attempt should be made to kill it. The sea snake should be lifted sharply by the tail, held at arm’s length with regular shaking of the hand (this is most effective in keeping the sea snake vertical and stopping it ‘climbing up itself’ to bite the hand), and then thrown into the sea.

Venomous bites and stings on land
Scorpion stings

Scorpions have four pairs of legs, a pair of claws, a body with broader front part and a 6-jointed tail-like abdomen. The terminal segment, called the telson, contains two venom glands connecting with the curved, needle-sharp sting which is used either in defence or in obtaining food. The tail with its sting is always brought forward in front of the scorpion. Scorpions never sting backwards and this feature enables safe handling of scorpions (provided one is familiar with their habits). The length of adult scorpions varies from <2 cm up to about 20 cm, but the length of the scorpion does not relate to its danger to man. Some of the most dangerous scorpions to man are only 2–4 cm long. Most scorpions are nocturnal, and feed on spiders and insects. During the daytime, they hide under logs, rocks, in cracks, among debris, clothing, etc. There are some 350 different species of scorpion. Probably the five most dangerous scorpions to man are: Centruroides (southern United States, Central America), Tityus (South America), Androctonus (Africa), Leiurus (Africa and Middle East), and Buthus (Asia). There are no reliable statistics indicating the frequency and danger of scorpion stings. In Israel, Efrati (1949) recorded that despite seeing scores of stings by Leiurus scorpions every year, he had observed only twenty-two cases of serious generalized poisoning during 12 years. In south India, no fatal cases were seen at a hospital where 800–1000 scorpion sting victims attended each year (Roantree, 1961). However, in some parts of the tropics, scorpion stings appear to be more important clinically than snake bite. This is said to be so in Mexico where Centruroides is alleged to have killed over forty victims each year in Durango, a small city of only 40,000 inhabitants. In Trinidad and South America, stings by Tityus are common and are sometimes fatal.

The clinical features depend mainly on the amount of venom injected relative to the weight of the victim. Local pain and fright are the most common results and this is all that happens in the great majority of stings. Pain can be severe and may last some hours, even 1–2 days. Local redness and swelling are unusual. General symptoms are exceptional but are
more common in younger victims. They suggest an 'autonomic storm' and include rapid breathing, salivation, sweating, vomiting, abdominal and generalized pains, and, in very severe poisoning, falling blood pressure and pulmonary oedema probably due to cardiotoxic effects. The latter can be fatal in children. In such cases various electrocardiographic abnormalities have been recorded. Evidence of acute pancreatitis is common with *Tityus* stings—abdominal pain, glycosuria, raised blood sugar and raised serum amylase. In India, evidence of intravascular coagulation has been reported in *Buthus* stings.

If local pain is severe, the area should be infiltrated through the puncture wound with 2–5 ml of 1% lignocaine hydrochloride. Alternatively, intramuscular or intravenous pethidine should be given, 100 mg for an adult, 2 mg per kg for children. Local injection of emetine hydrochloride has been reported as successful in relieving pain but is not recommended because it sometimes causes local necrosis (Cutting, 1963). For the general systemic symptoms, specific scorpion antivenom is available in a few parts of the tropics (Reid, 1975b). When antivenom is not available, general supportive treatment is indicated. Subcutaneous atropine (for adults 2 mg; 6–12 years 0.5–1 mg; 1–5 years 0.25–0.5 mg; up to 1 year 0.25 mg) and intravenous calcium gluconate (1–2 g for adults; 0.6–1 g for 6–12 years; 0.3–0.6 for under 6 years) have been reported as effective by some clinicians.

**Spider bites**

Spiders have a pair of horny fangs (chelicerae) through which venom can be injected. But few spiders can pierce human skin, and most are reluctant to bite man. As with scorpions and many snakes, the harmfulness of a spider cannot be judged from its appearance. Many of the large brown tarantula-like spiders are harmless to man. Perhaps the most dangerous are the widow spiders of the genus *Latrodectus* found in most tropical and sub-tropical countries. Adult *Latrodectus* bodies measure only about 1 cm in length. Another biting genus, *Loxosceles*, is brown and about the same size as *Latrodectus*; it is common in the Americas. Both sexes are venomous.

Man encounters spiders in dark, neglected places such as barns, stables and unused buildings. Black widow spiders may spin webs across latrine seats, and it is reported that in Texas 90% of spider bites occur in outside privies. *Latrodectus* poisoning causes excruciating cramp-like pains in the limbs, chest, and abdomen. Sweating, breathing difficulty, salivation, nausea, and vomiting may follow. Ptosis has been recorded, and in severe poisoning an itching rash may erupt over the trunk or around the site of the bite. Symptoms usually start within a few minutes of the bite, and within 30 min the victim may be unconscious. Abnormalities in the electrocardiogram have been observed, and death may be due to cardiac or respiratory failure. Recovery is usually complete within 24 hr but may take up to a week. The clinical picture following *Loxosceles* bites is quite different. Local pain is severe, and a white, intensely ischaemic area at the site of the bite is surrounded by redness and extravasated blood. The white area turns violaceous, then black and dry. The eschar separates, leaving a deep ulcer. In severe cases, haemoglobinuria, intravascular coagulation, and acute renal failure may occur.

Treatment of spider bites is similar to that of scorpion stings. A multitude of remedies has been proposed but controlled studies are notably lacking. *Latrodectus* antivenom (available from Commonwealth Serum Laboratories, Melbourne; Merck-Sharp and Dohme, Philadelphia, U.S.A., and from the South African Institute for Medical Research, Johannesburg) has been reported as clinically useful, but the results were not as dramatic as with calcium gluconate. Other measures advocated for latrotoxism include muscle relaxants, adrenaline, atropine, and chlorpromazine. Steroids have received favourable and unfavourable comment. In *Loxosceles* poisoning, no benefit was noted with prednisone. Antibacterial measures including early excision of eschars and skin grafting are helpful. If renal failure develops, dialysis might be needed. *Loxosceles* antivenom is available in Brazil, but clinical reports of effectiveness are lacking.

**Tick paralysis**

Hard ticks have a neurotoxic saliva which can cause a rapid progressive flaccid paralysis in man and certain other mammals. Human tick paralysis has been reported from Canada, United States, south-east Europe, South Africa, Somalia, and Australia. Symptoms start 4–6 days after attachment of the causative tick and may therefore not develop until the traveller has returned home. Anorexia and ataxia are followed by flaccid paralysis of legs, trunk, arms, and neck in that order. Tendon reflexes are decreased or absent; sensory changes are minimal or lacking. Fatal bulb or respiratory paralysis may develop, mortality in some series of tick paralysis being 10–12%. Removal of the tick, which is usually attached to the scalp or neck, is curative, and improvement may begin within an hour, recovery usually being complete within 48 hr. Travellers to tick-infested areas who are likely to intrude on tick habitats by camping, picnicking and so on, should look for ticks at the end of each day, especially where children are concerned.
Centipede bites

The body of a centipede has a series of similar segments each possessing a pair of legs. The head has a pair of antennae and associated mouth parts. The segment immediately behind the head has a pair of claws through which venom is injected when the centipede bites. Numbers of leg pairs vary from fifteen to over a hundred; in the tropics some centipedes exceed 25 cm in length. Bites can cause intense pain and treatment is similar to that for scorpion stings.

Bee, wasp and hornet stings

Honey bees have a barbed sting which is left behind together with the venom sac; but wasp and hornet stings are not barbed and are therefore not left behind. Bee venom contains many toxic fractions, the most important being melittin which alters capillary permeability, causes local pain and inflammation, haemolyses erythrocytes, and lowers blood pressure. Local pain is the commonest clinical feature of these stings but with multiple stings, general symptoms with haemoglobinuria or myoglobinuria, and acute renal failure may develop. Deaths have been reported after only 30–60 stings by the honey bee, but usually 400–500 stings are necessary to produce death in an adult, and survival after 2243 stings has been reported (Murray, 1964).

The most serious aspect of wasp and bee stings is allergic reaction (Barr, 1974). In the United States there are more deaths from anaphylaxis following wasp or bee stings than from all other venomous bites or stings combined together. In very susceptible patients symptoms start after a few seconds with tingling of the scalp, vasodilatation, hypotension, and death within 1–2 min. In most patients, however, the reaction begins in 1–2 min with generalized urticaria, followed during the next hour by oedema of the glottis, bronchoconstriction, hypotension, and coma. Once an immediate reaction has occurred, the response to further stings starts progressively sooner and the severity of the shock increases.

Delayed allergic reactions may also occur, coming on 1–7 days after the sting—fever, urticaria, enlarged lymph nodes, joint pains and leucocytosis. These episodes usually last 1–2 weeks.

The bee sting should be removed. It is a tiny black shaft with the white poison sac attached to its free end. It should not be grasped by forceps or fingers as this can express more venom from the sac. The sting should be scraped from the flesh with the finger-nail or the blade of a knife. Local antiseptic is then applied; pethidine may be needed for pain. It has been claimed (Arnold, 1972) that meat tenderizer, available in most kitchens, applied in a dilute solution (quarter-teaspoonful of tenderizer mixed with a teaspoonful of tap water) rubbed into the skin relieves all pain within seconds. The effect presumably depends on the content of papain which breaks down venom and kinins at the sting site. Adrenaline is indicated for anaphylactic reactions, either by injection or by inhalation. Inhalation of an adrenaline aerosol (Medihaler EPI, Riker) from a pressurized can similar to that used for asthma is more rapid than administering an injection. Five inhalations of this aerosol are equivalent in effect to 0.5 ml of injected 1 : 1000 adrenaline. People who are known to be allergic to bee or wasp stings should always carry such a can. Desensitization can be done but this is usually temporary unless maintenance desensitizing injections every 1–4 months are continued indefinitely.

Caterpillars, moths and beetles

Some caterpillars and moths have sharp hairs or spines which contain venom. Examples are pine caterpillars (Thaumatopoea) and processionary caterpillars (Ctenocampa) in Europe and the Mediterranean area, puss caterpillars (Megalopyge opercularis) in the United States, Hylesia moths and caterpillars in South America. Outbreaks of caterpillar dermatitis usually occur in the summer months. Symptoms produced by contact with urticarial species may be restricted to reddening and itching but may include burning pain, swelling, blistering, conjunctivitis, vomiting, headache, paralysis and shock. Prompt application of scotch tape can remove many spines; lotions or creams with steroids relieve the skin reactions. Antihistamines are of little value.

Blister beetles of the Meloidae family can exude a vesicant on injury. Small rove beetles (Paederus) fly during the evening and are common in humid climates. When crushed or rubbed on the skin, erythema results about 10 hr later followed by a crop of small blisters which persist for about two days.

Land snake bites

Snake bite as a holiday hazard in Europe, the Near East, North Africa and the West Indies (Reid, 1971) and snake bite in the tropics (Reid, 1972a, b) have been considered elsewhere and the hazard of land snake bite to the traveller will only be summarized in this paper. Snake bite is a rural and occupational hazard and travellers, even to the tropics, can be reassured that they are most unlikely to see, let alone be bitten by snakes. If a traveller does get bitten it is usually his own fault (e.g. by picking a snake up).

Vipers and elapid. Medically important land snakes have fangs at the front of their mouths which enable them to inject venom. These are the 'poisonous' snakes of which there are two families
—elapids (neurotoxic), and vipers (vasculotoxic). Elapids have short fixed fangs (covered by a gumfold, the vagina dentis). Vipers have long, erectile fangs, triangular heads, and, usually, short fat bodies. Vipers are subdivided into crotaline or pit vipers, having a thermostensitive pit between eye and nose, and viperine vipers, without pits. The pit detects warm-blooded prey in the dark. All snake venoms are highly toxic, but when a venomous snake bites man it is a defensive or warning bite when little or even no venom is usually injected, hence most human victims do not have serious poisoning. Only about 25% will develop systemic poisoning. Therefore poisonous snake bite is not necessarily the same as snake bite poisoning. Fear in varying degrees is present in all victims bitten by snakes and often dominates the clinical picture.

**Early effects of envenoming.** Local swelling starts within a few minutes of a viper bite if venom is injected. It is a valuable clinical sign, because if swelling is absent and one knows the biting snake was a viper then poisoning can be immediately excluded. Local swelling is also a feature of poisoning by Asian cobra bites and African spitting cobra bites, though it may not appear for 1–2 hours. Other elapids (such as mambas and kraits) have no local effects. Local pain almost invariably follows cobra and viper bites if venom is injected, and it may be severe for several days. But local pain may be minimal or absent in severe poisoning, and considerable in bites not involving poisoning; thus local pain is extremely variable and of no help in diagnosis.

The important early signs of systemic poisoning are: *viper*—blood-stained spit, later non-clotting blood; *elapid*—ptosis, glossopharyngeal palsy. Non-clotting blood is best detected using a capillary tube with blood taken from a finger prick. The tube should be kept horizontal at room temperature for 20–30 min and then raised vertically. Non-clotting blood runs out of its own accord or can easily be blown out. There are exceptions to these generalizations. The tropical rattlesnake has neurotoxic, not haemorrhagic, effects although it is a pit viper. Some Australian elapid snakes have myotoxic, vasculotoxic, and coagulation effects although they are principally neurotoxic. Some vipers (such as the puff adder and the gaboon viper in Africa) do not affect clotting in human victims.

**Later effects of envenoming.** Blisters around the site of the bite are common in cobra and viper envenoming, and blisters extending up the limb suggest a large dose of venom and often precede necrosis. Local necrosis is characteristic of poisoning from Asian and African spitting cobra bites and some viper bites (such as the African puff adder). It becomes evident a few days after the bite and is shown by a darkening of the skin together with an offensive ‘putrid’ smell, which is particularly marked in cobra bite necrosis. Necrosis can be extensive but it is usually superficial; involvement of tendons, muscle and bones is exceptional. Bacterial infection follows necrosis and may spread to joints. But in the absence of necrosis or meddlesome local measures such as incision, application of dressings, etc., bacterial infection virtually never occurs.

**Prognosis.** Viperine poisoning is severe if within 1–2 hr of the bite swelling is above the knee or elbow, shock is evident, or haemorrhagic signs besides haemoptysis develop (gum bleeding, ecchymoses, positive tourniquet test and so on, do not usually appear for 4–5 hr). Elapid poisoning is severe if neurotoxic signs start within 1 hr or less of the bite and rapidly progress to respiratory failure. Mental confusion strongly suggests respiratory failure, although ptosis and glossopharyngeal palsy can make assessment of mental awareness difficult. Shock may also be a feature of severe elapid poisoning. The laboratory findings in snake bite are detailed elsewhere (Reid, 1974). Polymorph leucocytosis indicates serious poisoning. Even without specific treatment the mortality in snake bite is low. Generally speaking, deaths are most rapid after elapid bites, especially cobra bites (average death time is about 5 hr after the bite) and more protracted after viper bites (average is 2–3 days after the bite). In elapid bite, death is mainly due to cardiorespiratory failure; shock, and haemorrhage into vital organs are the main causes of death in viper bites.

**Clinical course of envenoming.** In the absence of necrosis, pain after viper bites rarely exceeds 2 weeks. Swelling usually resolves completely in 2–3 weeks, rarely in 2–3 months, and in exceptional cases the limb may remain permanently swollen. If blisters are left alone and no dressing is applied, they rupture spontaneously about two weeks after the bite and dry up in another 1–2 weeks. If dressings are applied, infection usually follows and greatly prolongs healing. Healing of local necrotic lesions varies according to the extent of the lesions and the treatment given, but requires at least a month, and may take 5–6 months or longer even with expert surgical attention. In patients who recover without receiving specific antivenom, systemic symptoms generally subside quickly. Neurotoxic features of elapid systemic poisoning resolve in 2–3 days as a rule, but exceptionally may persist as long as 2 weeks. In systemic viper bite poisoning shock and haemorrhagic features generally resolve within a week, but in bites by some vipers coagulation changes may persist for 2–3 weeks or even longer if specific antivenom is not given. Complications of systemic poisoning are rare. Renal failure may occur with bites by all types of poisonous snake—vipers, elapids, or sea snakes. Neglected local necrosis can result in
chronic osteomyelitis with sinuses discharging for years. Scarring may be extensive.

First-aid treatment. This comprises the measures taken by the victim or associates before receiving medical treatment. Recommendations (it will be rare indeed for the doctor to have to apply them personally) should be short, simple, practicable and more helpful than harmful. Reassurance is most important, as the danger of snake bite is greatly exaggerated. The site of the bite should not be incised, as this frequently introduces infection, delays recovery and aggravates bleeding. A firm, but not tight ligature should be applied just above the bite, using cloth, handkerchief or grass. The victim should then go to the nearest hospital. If available, aspirin or alcohol in moderation is helpful. If the snake has been killed it should be taken to hospital; otherwise it should be left alone, since attempts to find or kill it often result in further bites. Antivenom should play no part in first-aid treatment.

Hospital treatment. Details of the hospital treatment of snake bite are given elsewhere (Reid, 1974) but it must be emphasized that antivenom is indicated only to counteract systemic poisoning (and if correctly used, antivenom is very effective in doing this) and to prevent or to minimize local necrosis in bites by known necrotizing snakes such as the puff adder when the patient presents within two hours of the bite.

Prevention. This is a matter of common sense. Boots give more protection than shoes. When walking in the dark, a torch should be used. Camping should be on open ground and, preferably, trenches should be dug around the tent and filled with lime. Food should be kept separate from the sleeping area—it attracts rodents which in turn attract snakes. If a snake is encountered it is safer to keep perfectly still; the snake will then be unable to strike toward the movement it requires, and can be distracted by throwing down some object.

References


Reid, H.A. (1972b) Snake bite. Part II: Treatment. Tropical Doctor, 2, 159.


