British Herpetological Society Bulletin, No. 21, 1987.

SNAKE BITES IN THAILAND AND BURMA

SAUL M. HALPERN

The London Hospital, Whitechapel, London, E1

INTRODUCTION

Many common South-east Asian snakes are venomous. Snakebite is a serious problem in the tropics. I have recently spent two months in South-East Asia, where I studied snake bites at Chulalongkorn Hospital, Bangkok, Thailand, The Department of Medical Research, Rangoon and Tharrawaddy District Hospital, Burma.

Most venoms contain several or many components designed to rapidly kill prey, but which cause a variety of problems in human victims. Clinical features can be divided into the local effects, (at the site of the bite) and systemic effects, (acting on the body in general or distant sites). In general systemic features of elapid venom (cobras, kraits) are primarily neurotoxic (affecting the nervous system), whereas those of viperid venom (viper) are primarily haemotoxic (acting on the blood). Contrary to popular belief, more than half of bites by all potentially lethal snakes result in slight or even no poisoning. Medical management of snake bite consists of reassurance, identification of the responsible species, assessment of the severity of envenoming, administration of antivenom if necessary, and supportive therapy.

The important species responsible for bites in Thailand and Burma are the Russell's Viper (*Vipera russelli siamensis),* Malayan Pit Viper *(Calloselasma rhodostoma),* Green Pit Viper *(Trimeresurus albolabris),* Thai Monocellate Cobra *(Naja kaouthia)* and the Malayan Krait *(Bungarus candidus).* Bites from sea snakes are rare.

RUSSELL'S VIPER (*Vipera russelli)*

The Russell's Viper is most commonly found in the central rice growing regions of Thailand and Burma. In Burma, more than 90% of bites are due to *Vipera russelli,* compared to 5% due to cobras. It is estimated that this species is responsible for over 10,000 bites per year in Burma and at least 1,000 deaths. This considerable morbidity and mortality has made snake bite between the fifth and seventh leading single cause of death in Burma, even in recent years.

The rural population is most at risk, particularly the young and fit breadwinners who are often bitten in the fields, especially at harvest time (November - December), where snakes come in search of rodents. In Burma, Russell's Viper bite is considered as an occupational hazard. The majority of bites occur in the early morning or in the evening. The foot being the commonest site, followed by the hand and ankle.

After being bitten, pain usually starts within minutes and swelling of the affected limb continues to a maximum at 1-4 days. Unlike other viperid bites, such as *Calloselasma rhodostoma,* bruising, blistering and necrosis (tissue death) are rare. The regional lymph nodes (eg in the groin), usually enlarge and are tender as a result of lymphatic absorption of the venom.

The earliest sign of systemic envenoming is non-clotting blood. This can be detected by leaving a blood sample to stand for 20 minutes (20 minute clotting test). In up to 40% of cases with systemic envenoming there may be continuous oozing from the bite wound, spontaneous bleeding from the gums, or bleeding into the conjunctivae, gastro-intestinal tract or pituitary gland. Russell's Viper venom contains several haemotoxic components which act at various steps in the clotting mechanism. In the snake's natural prey, this causes rapid and fatal intravascular coagulation (clotting in blood vessels) but in man, the venom dose is insufficient to have such a devastating effect and results in disseminated intravascular coagulation (widespread minute clots) with subsequent depletions of clotting factors and platelets. This in turn leads to non-clotting blood.

In about 35% of victims there is hypotension (low blood pressure) and shock resulting from a generalised increase in capillary permeabillity. Occasionally, abdominal pains and vomiting

occur. The most serious feature of Russell's Viper envenoming is kidney failure. This occurs in up to 40% of cases with systemic poisoning and may be preceded by loin pain or tenderness. Oliguria (small volumes of urine) and haematuria (blood in the urine) can develop in 24-72 hours of the bite and death is likely in severe cases of renal failure if dialysis is not available. Unfortunately, kidney machines are not generally available in Burma, particularly outside Rangoon. It was initially thought that the damage to the kidneys was a consequence of the disseminated intravascular coagulation but studies at the Department of Medical Research, Rangoon, suggest that there may also be a directly acting nephrotoxin (kidney poison). As previously stated, the mortality from these bites is about 10% with acute renal failure being the prime cause of death, although disseminated intravascular coagulation and haemorrhage into the lungs or pituitary may also be found at autopsy.

Victims usually bring the corpse of the offending snake with them to hospital. This enables accurate identification of the species. Hourly blood samples show any change in clotting ability and if non-clotting blood is found antivenom treatment is initiated. Pryogenic reactions (fever, chills and rigors) occur in almost 100% of patients given BPI (Burma Pharmaceutical Industries) monospecific antivenom but anaphylactic (allergic) reactions are uncommon. This is due to the presence of foreign proteins despite the antivenom having undergone a purification process. Antivenom will effectively restore blood coagulation in 4-48 hours. However it does not appear to prevent the development of renal failure even if adequate treatment is given within one hour of the bite. In non-fatal cases of severe envenoming recovery can take many weeks, although pituitary haemorrhage may lead to a syndrome of hormonal imbalance and subsequent ill health. This has been shown in patients bitten up to 24 years previously.

An interesting feature of Russell's Viper bites is the variability of symptoms and signs in the victims. For example, non-clotting blood may be found in conjunction with limited or absent local pain and swelling. Alternatively, local features may be marked whilst systemic features are absent! This may be related to genetic variations or perhaps differing rates of production of venom components. Genetic variation is more likely to explain regional differences, such as the frequency of conjunctival oedema (swelling of the outer layer of the eye), seen only in Burma or rhabdomyolysis and massive haemolysis (destruction of muscle and red blood cells) as described in Russell's Viper bites from Sri Lanka.

GREEN PIT VIPER *(Trimeresurus albolabris)*

There are several species of *Trimeresurus* in Thailand. *Trimeresurus albolabris* is responsible for most bites, primarily due to its abundance, followed by *T. macrops* and occasionally *T. wagleri* or *T. purpureo maculatus.*

It is common around Bangkok and up to 20 cases of bites may present per week at Chulalongkorn Hospital, especially during the rainy season. Relying on its camouflage rather than fleeing, it tends to bite people who come too close, albeit unsuspectingly, to the bushes or small trees in which it lives.

The bite of the green Pit Viper is rarely, if ever, fatal. Victims present with severe pain in the bitten limb, together with gross swelling and discolouration of that limb. Viper venom is composed of much larger protein molecules than cobra venom and therefore tends to be absorbed slowly through the lymphatic system rather than by venous drainage. Thus it may take several hours before the severity of envenoming can be gauged. The extent upwards of swelling of the affected limb is proportional to the dose of venom injected.

The venom contains haemorragin which damages blood vessel walls, causing extravasation of plasma and red blood cells into the limb accounting for much of the characteristic swelling and bruising. Non-clotting blood is a feature of severe envenoming resulting from both difibrination and platelet (requirements for clotting) deficiency from excessive repair of the damaged vessels. A direct antiplatelet toxin component in the venom may also be present.

Victims are observed and the severity of envenoming is graded by the extent of swelling, blood clotting time and platelet count. Only cases of severe poisoning are admitted to hospital and are treated with specific antivenom. There is no local necrosis and the affected limb slowly resolves to its normal size and colour.

MALAYAN PIT VIPER (*Colloselasma rhodostoma*)

The Malayan Pit Viper is found in many parts of Thailand and is the commonest cause of snake bite in the Malayan peninsular. It is often trodden on as it is well camouflaged and characteristically does not move away when disturbed.

The bite is rarely fatal and has some similarities with *Trimeresurus* bites. Local pain and swelling start within a few minutes but may continue to increase for 24-72 hours depending on the dose of venom injected. This is accompanied by bruising discolouration. Local necrosis also occurs in about 30% of cases and may be preceded by gross blistering of the skin. Necrosis is usually only superficial but, as with cobra bites, secondary bacterial infection is a common complication.

The systemic features of severe envenoming include non-clotting blood and a general haemorragic syndrome characterised by spontaneous bleeding from the gums into the skin, continuous oozing from the bite and occasionally haemoptosis (coughing up blood) or cerebral haemorrhage (bleeding into the brain). Untreated, this syndrome lasts 3-4 days, although the blood remains incoagulable for 5-11 days. Hypotension and shock may occur in some patients probably due to the loss of circulating fluid into the bitten limb and possibly also to the effects of intravascular coagulation.

Specific antivenom will reduce or prevent development of shock or haemorrhagic syndrome. Prior to its availability, mortality in hospital-treated cases was only about 1%. An occasional death may still occur due to cerebral haemorrhage, anaphylactic antivenom reactions or secondary infection. Swelling resolves in 5-20 days but, in the presence of local necrosis complete recovery may take 1-10 months.

THAI COBRA *(Naja kaouthia)*

Cobras represent about 5-10% of bites in Thailand and Burma, the Monocellate Cobra *(Naja kaouthia)* being the most common, although *N.N. sputatrix* is commoner in some parts of Central and Northern Thailand. Cobra bites are less frequent than might be expected as these snakes are generally active and escape when disturbed, striking usually only when cornered or threatened.

The bite of the Thai Cobra normally results in local pain and swelling within a few minutes. Systemic envenoming is characterised by ptosis (sagging of the eyelids) followed by glososopharyngeal palsy (difficulty in speaking and swallowing) and finally paralysis of the respiratory muscles. These features may take several hours to develop; the toxins responsible acting specifically on receptors at the junction of the nerve and muscle, thereby blocking the transmission of nerve impulses. In the absence of treatment, severe envenoming will lead to death from respiratory failure (ie suffocation). This poisoning is, however, reversible and these effects wear off in 2-5 days. Untreated, the natural mortality is about 10%.

Monospecific Antivenom is effective and available in Thailand, yet its use is currently controversial at Chulalongkorn Hospital, where supportive measures only are employed. All victims are admitted for observation and placed on an artificial ventilator in the intensive care unit if necessary. Once the patient has recovered from this initial life-threatening condition, he may have to spend several more weeks in hospital as severe necrosis and gangrene at the site of the bite often follows. This is a direct venom effect and the dead tissue may require surgical excision, followed later by skin grafting if the area is extensive. The wound must be kept clean as the risk of secondary infection is high. Some improvement in the clinical state can be produced by anticholinesterase drugs which increase the concentration of the natural neurotransmitter substance acetylcholine at its receptor site, thereby reducing the blocking effect of the venom at the same site.

MALAYAN KRAIT *(Bungarus candidus)*

Information relating to the incidence of *Bungarus candidus* bites is limited, but it is generally considered to be a rarer cause of snake bite in Thailand. However, since Krait bites in general are associated with a higher mortality (77%, reported in 35 cases of *Bungarus caeruleus* in India), they represent the commonest cause of fatal snake bite. Bites often occur at night in houses while the victims are sleeping, causing difficulty in identifying the species responsible.

There are normally no local effects from the bite except some numbness. Pain and swelling are not features of Krait bites, although there may be abdominal discomfort. The systemic features indicate that the venom is a powerful neurotoxin and 'Bungarotoxin' is well known to cause neuro-muscular blockade. Paralysis starts 1-12 hours following the bite with ptosis, difficulty in swallowing and talking, proceeding to difficulty in breathing and finally total paralysis affecting all limbs, face, eyes and respiratory muscles.

Supportive artifical ventilation may rapidly become necessary to prevent suffocation. There is no specific antivenom currently produced and it has been found that Thai *Bungarus fasciatus* antivenomm is ineffective, but a response to Haffkine poly-specific antivenom *(Bungarus caeruleus, Echis carinatus, Vipera russelli, Naja naja)* has been reported. Experiments on mice have found that Australian Tiger snake antivenom has a greater protective effect against *Bungarus candidus* venom than the Haffkine antivenom. As with Cobra bites, anticholinesterase drugs may be useful in treatment.

CONCLUSIONS

There are many misconceptions concerning snakebite. I hope in this article I have removed some of them. Current and past research has uncovered much interesting and useful information, although snakebite related problems have never been high priority and further work is undoubtedly required. In addition, treatment will continue to be difficult in Third World countries or where transport and medical facilities are limited.

ACKNOWLEDGEMENTS

I am grateful to Professor D.A. Warrell, John Radcliffe Hospital, Oxford, and Dr. R.D.G. Theakston, WHO Collaborative Centre for the Control of Antivenoms, Liverpool School of Tropical Medicine, for advice, supplying me with information, and helping to arrange my elective to Thailand and Burma.

REFERENCES

Aung Than Batu (1986) Review Article: Recent studies of Russell's Viper Bites in Burma. *DMR Bulletin Apr 1986.*

Chasin Viravan, Udom Veeravat, et al. (1985). ELISA Confirmations of Acute & Past Envenoming by the Monocellate Thai Cobra *(Naja kaouthia) am j trop med hyg* **35** (1), 173-181.

May Ho, A. Warrell, et al. (1986). Clinical Significance of Venom Antigen Levels in Patients Envenomed by Malayan Pit Viper *(Calloselasma rhodostoma). am j trop med hyg* **35** (3), 579-587.

Kamolrat Silamut, May Ho, et al: Detection of venom by Enzyme Linked Immunosorbant Assay (ELISA) in Patients Bitten by Snakes in Thailand. *British Medical Journal* **294** 1987, 402-404.

Myint-Lwin, D. A. Warrell, et al: Bites by Russell's Viper *(Vipera russelli siamensis)* in Burma: Haemostatic, Vascular & Renal Disturbances and Response to Treatment.

Phelps, T. (1981). *Poisonous Snakes* Blandford Press.

H.A. Reid, P.C. Thean, et al (1963). Clinical Effects of Bites by Malayan Viper *(Ancistrodon rhodostoma) Lancet* 23 March, 617-626.

H.A. Reid, R.D.G. Thearston (1983). The Management of Snake Bite. Bull WHO **61** (6), 885-895.

D. A. Warrell, Sornchai Looareesuwan, et al. (1986). Randomised Comparative Trial of Three Monospecific Antivenoms for Bites by the Malayan Pit Viper *(Calloselasma rhodostoma)* in Southern Thailand: clinical & Laboratory Corelations *am j trop m d hyg* **35** (16), 1235-1247

D. A. Warrell, Sornchai Looareesuwan, et al. (1983). Severe Neurotoxic Envenoming by the Malayan Krait Bungarus candidus (Linaeus): Response to antivenom & Anticholinesterase. *British Medical Journal* **286**, 678-680.

Watt, et al. (1986). Positive response to edrophonum in patients with neurotoxic envenoming by cobras *(Naja naja philippinensis). New Eng J Med* **315**, 1444-1448

Tun-Pe, et al. (1987). Acute & chronic pituitary failure resembling Sheehans syndrome following bites by Russell's Viper in Burma. *Lancet* Oct 3, 763-767.

Plate 1. *Vipera russelli siamensis* (photo Prof. D.A. Warrell)

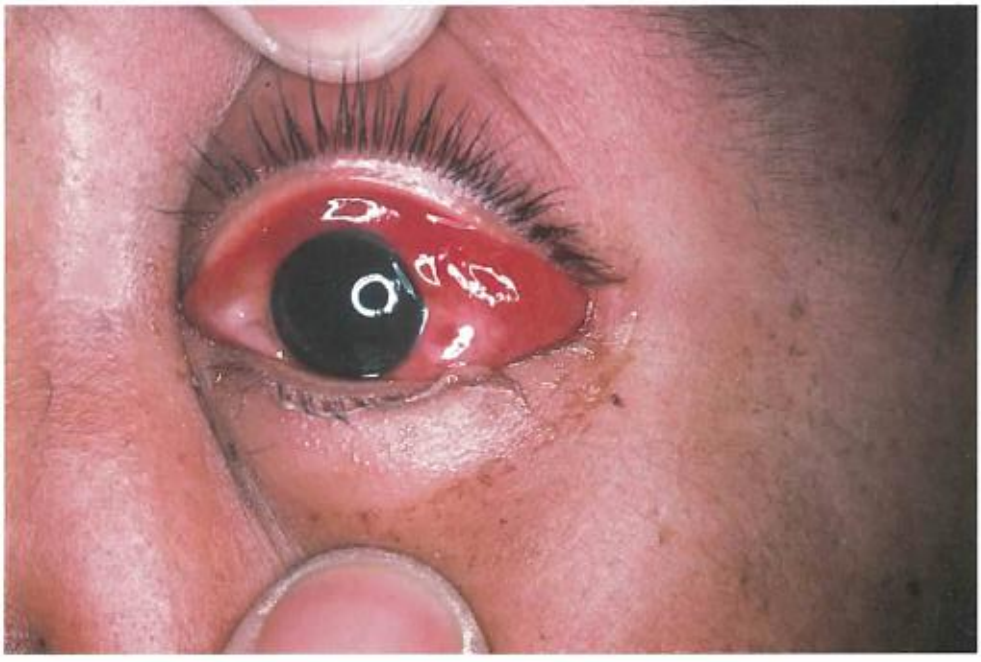

Plate 2. Conjunctival oedema, a feature of systematic envenoming by Burmese *Vipera russelli siamensis* (photo Prof. D.A. Warrell)

Plate 3. *Trimeresurus albolabris* (photo Prof. D.A. Warrell)

Plate 4. *Calloselasma rhodostoma* (photo by Prof. D.A. Warrell)

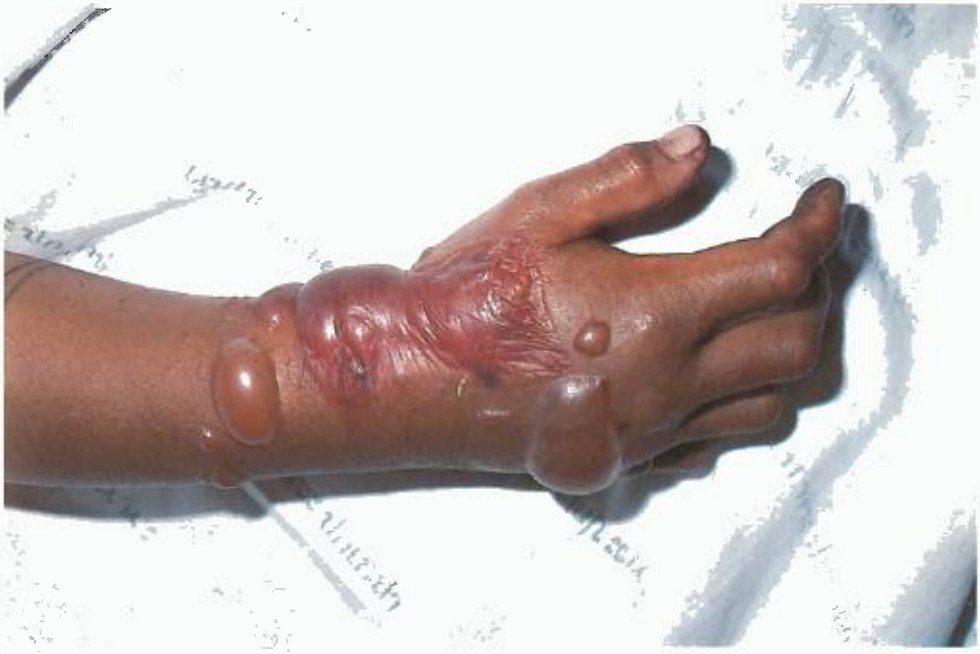

Plate 5. *Calloselasma rhodostoma* bite to hand, showing blistering of skin (photo Prof D A. Warrell)

Plate 6. *Naja kaouthia* (photo Prof D A. Warrell)

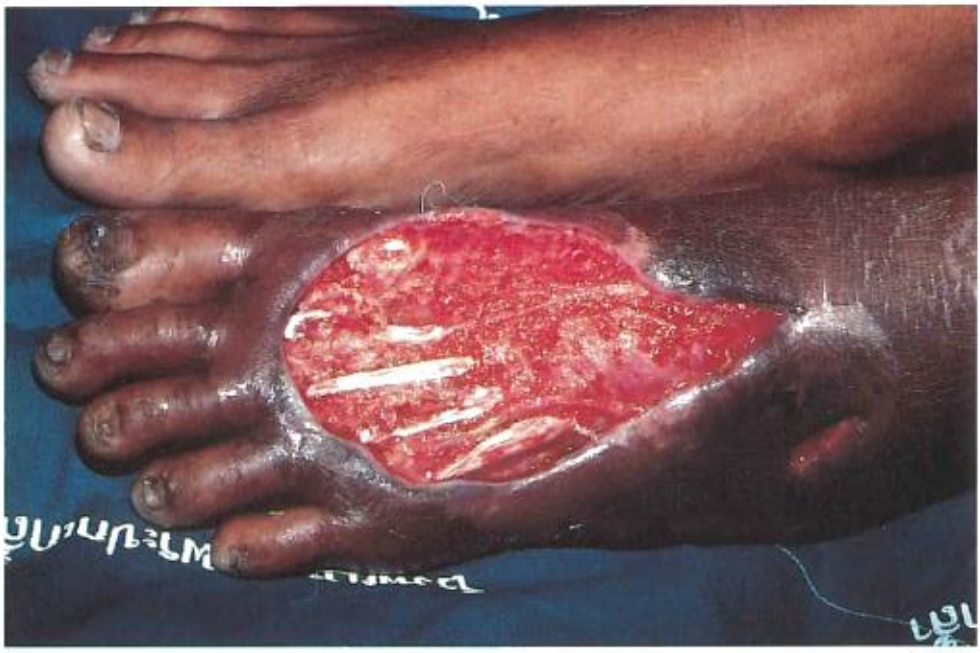

Plate 7. *Naja kaouthia* local uecrosis following bite to foot (photo Prof. D.A. Warrell)

Plate 8. *Bungarus candidus* (photo Prof. D.A. Warrell)