THE DEATH ADDER (ACANTHOPHIS ANANTARCICUS): THE EFFECT OF THE BITE AND ITS TREATMENT


Port Moresby General Hospital, Territory of Papua and New Guinea

The death adder was one of the first venomous reptiles to be described in Australia (Shaw, 1861). Because of its viper-like appearance, the snake was thought for a long time to be a true viper (McCoy, 1861), but Krefft (1869), although doubtful about its classification because of its viperine appearance, correctly classified the death adder as an elapine snake.

Characteristics of the Snake

The early Australian settlers called the snake "the death adder" because of "its hideous aspect" and "its deadly effect" (Bennett, 1834). The "deadly effect" of the snake seems to have been based on the effects of its bite on domestic animals and its bad reputation among the Aborigines (Cunningham, 1827; Bennett, 1834; Govett, 1836), for fatal snake bites were very uncommon among the early settlers in Australia (Tench, 1793; Bland, 1861). Another common name for the snake was "the deaf adder" (Cunningham, 1827; Berncastle, 1862-1863). This name characterized its sluggish, torpid nature and as it did not move out of the way like other snakes it was thought not to hear (Cunningham, 1827). However, the Mekeo natives of the Kaliru sub-district of Papua call this snake a/f, which means "sharp-eyed", indicating that perhaps it is not always sluggish and torpid.

The viper-like appearance of the death adder readily identifies it from all other Australian and New Guinea venomous snakes (Figure 1). It has a broad, triangular head, a narrow neck and a short, stout body which terminates abruptly in a very short, thin tail. The colour varies, according to its environment, from a light brown to a reddish brown or black. Lighter or darker transverse bands mark the body. Most specimens are less than two feet in length. Krefft (1869) recorded one specimen measuring two feet eight inches in length and with a nine-inch girth. The largest specimens seen by Waite (1898) and Kellaway and Eades (1929) were two feet five and one half inches long. The largest specimen ever recorded appears to be one at the Australian Museum measuring three feet and one half an inch (H. G. Cogger, personal communication).

The death adder mostly sleeps by day and moves about after-dark. During the day the death adder may use its broad, shovel-like head and a side-ways swinging motion of its body to burrow beneath leaves, sand or gravel. It may lie partially concealed so that only part of its back may be visible. The snake frequently lies in an S-shaped curve and the twitching of its tail is used to attract prey (Worrell, 1963).

It is sluggish in the daytime, and when discovered makes no attempt to escape (Kellaway and Eades, 1929). It often does not move even if people tread near it, and does not strike until touched. Bancroft (1894) said he had seen a native and a horse treading on a death adder, and neither was bitten. Waite (1929) related the story of a patrol of 30 to 40 New Guinea natives passing over a death adder until it was noticed by the last member of the patrol. The patrol leader's boot imprint was two inches from the head of the snake. G. A. V. Stanley (personal communication, 1965), on a geological expedition in the Gulf and western districts of Papua, noticed that one of his patrol group was defaecating beside a death adder; the man was told to move. The snake did not strike. When touched, the body of the snake flattens out and it strikes at a low level over a short distance with amazing speed (Kellaway and Eades, 1929).

Distribution

The snake is widely distributed throughout Australia and is most numerous in the warmer parts. It does not occur in Tasmania or the southern part of Victoria.

Figure 1: The death adder (Acanthophis antarcticus) (Photograph, H. G. Cogger).

(Kellaway and Eades, 1929). A sub-species, Acanthophis antarcticus pyrrhus, occurs in central Australia (Kinghorn, 1956).

The death adder occurs not only in Australia but also is widely distributed throughout the mainland of New Guinea, including West New Guinea. It has been reported from all mainland districts of the Territory of Papua and New Guinea except the Southern Highlands. It is found in coastal areas and in the highlands up to an elevation of 5,300 feet. In New Guinea the snake is a forest dweller and two sub-species, of doubtful validity, Acanthophis antarcticus rugosus and Acanthophis antarcticus lavus, are described from Papua.

The death adder also occurs in the Celebes and Ceram.

Biting Apparatus and Venom Studies

The fang length varies from 5 mm. to 8-3 mm. and averages 6-2 mm. (Fairley, 1929b). Its fangs were larger and more mobile than any other Australian venomous snake studied by Fairley (1929b). Tidwell (1906) reported two estimations of the venom yield of the death adder as 42.4 mg. and 71-0 mg. Fairley and Splatt (1929), in a study of 42 death adders, obtained an average maximum venom yield of 84-7 mg. and a maximum venom yield of 235-6 mg. This is a large venom yield for an Australian snake. Milligram for milligram, when tested on sheep, death adder venom was considered to be 10 times as toxic as cobra venom, and half as potent as tiger snake venom (Fairley, 1929a). Kellaway (1929a) estimated the certain lethal dose in man to be 0-025 to 0-15 mg. per kilogram. Fairley (1929a) concluded that the death adder had the most efficient biting apparatus and highest venom yield of any

1 Physician.
2 Present address: School of Public Health and Tropical Medicine, University of Sydney.
TABLE 1
Summary of Clinical Details of 15 Patients with Death Adder Bites

<table>
<thead>
<tr>
<th>Patient's Number</th>
<th>Sex</th>
<th>Approximate Age (Years)</th>
<th>Time of Bite</th>
<th>Month, Bite</th>
<th>Circumstances Bites1</th>
<th>Site; Bite</th>
<th>Symptoms</th>
<th>Signs</th>
<th>Result of Urine Testing</th>
<th>Progress after Antivenene1</th>
</tr>
</thead>
<tbody>
<tr>
<td>3548</td>
<td>M</td>
<td>35</td>
<td>6.30 p.m.</td>
<td>October</td>
<td>Running behind horse; struck on S.; leg; killed</td>
<td>Toe</td>
<td>+</td>
<td></td>
<td>N.G.; no progression</td>
<td>No progression</td>
</tr>
<tr>
<td>3774</td>
<td>M</td>
<td>40</td>
<td>10 a.m.</td>
<td>November</td>
<td>Trod on S.; S. hung on; killed</td>
<td>Ankle</td>
<td></td>
<td></td>
<td>No progression</td>
<td>N.G.; no progression</td>
</tr>
<tr>
<td>3613</td>
<td>M</td>
<td>25</td>
<td>2 p.m.</td>
<td>November</td>
<td>Sitting under house; S. hung</td>
<td>Toe</td>
<td></td>
<td></td>
<td>N.G.; no progression</td>
<td>N.G.; no progression</td>
</tr>
<tr>
<td>4229</td>
<td>M</td>
<td>35</td>
<td>11 a.m.</td>
<td>December</td>
<td>Trod on S.; let go; killed</td>
<td>Toe</td>
<td></td>
<td></td>
<td>N.G.; no progression</td>
<td>N.G.; no progression</td>
</tr>
<tr>
<td>11294</td>
<td>M</td>
<td>40</td>
<td>11.30 a.m.</td>
<td>August</td>
<td>Walking; S. &quot;came at him&quot;; kicked off; killed</td>
<td>Ankle</td>
<td></td>
<td></td>
<td>N.G.; no progression</td>
<td>N.G.; no progression</td>
</tr>
<tr>
<td>15125</td>
<td>M</td>
<td>25</td>
<td>3 p.m.</td>
<td>December</td>
<td>Not recorded; S. killed</td>
<td>Ankle</td>
<td></td>
<td></td>
<td>N.G.; no progression</td>
<td>N.G.; no progression</td>
</tr>
<tr>
<td>16722</td>
<td>M</td>
<td>20</td>
<td>11 a.m.</td>
<td>March</td>
<td>Not recorded; S. killed</td>
<td>Foot</td>
<td></td>
<td></td>
<td>N.G.; no progression</td>
<td>N.G.; no progression</td>
</tr>
<tr>
<td>17896</td>
<td>M</td>
<td>25</td>
<td>5.30 p.m.</td>
<td>May</td>
<td>Not recorded</td>
<td>Heel</td>
<td></td>
<td></td>
<td>N.G.; no progression</td>
<td>N.G.; no progression</td>
</tr>
<tr>
<td>23548</td>
<td>M</td>
<td>35</td>
<td>1 p.m.</td>
<td>June</td>
<td>Lay on leaves; S. under leg</td>
<td>Leg</td>
<td></td>
<td></td>
<td>N.G.; no progression</td>
<td>N.G.; no progression</td>
</tr>
<tr>
<td>27853</td>
<td>M</td>
<td>20</td>
<td>9 a.m.</td>
<td>October</td>
<td>Kicked; S. killed</td>
<td>Toe</td>
<td></td>
<td></td>
<td>N.G.; no progression</td>
<td>N.G.; no progression</td>
</tr>
<tr>
<td>28040</td>
<td>M</td>
<td>20</td>
<td>7.30 p.m.</td>
<td>October</td>
<td>On road through grass; bitten; did not see S.; dark</td>
<td>Foot</td>
<td></td>
<td></td>
<td>N.G.; no progression</td>
<td>N.G.; no progression</td>
</tr>
<tr>
<td>28458</td>
<td>M</td>
<td>20</td>
<td>8.30 a.m.</td>
<td>November</td>
<td>Picked up grass; S. hung on; pulled off; thrown away</td>
<td>Finger</td>
<td></td>
<td></td>
<td>Potosis</td>
<td>Albumin F.; reversed</td>
</tr>
<tr>
<td>52029</td>
<td>F</td>
<td>35</td>
<td>10 a.m.</td>
<td>August</td>
<td>Trod on S.; S. leg; killed</td>
<td>Toe</td>
<td></td>
<td></td>
<td>Severe; generalized</td>
<td>P.; reversed</td>
</tr>
<tr>
<td>35263</td>
<td>M</td>
<td>25</td>
<td>3 a.m.</td>
<td>September</td>
<td>Trod on S.; kicked S. free</td>
<td>Heel</td>
<td></td>
<td></td>
<td>Severe; generalized</td>
<td>Albumin, P.; reversed</td>
</tr>
<tr>
<td>35645</td>
<td>M</td>
<td>35</td>
<td>7.30 p.m.</td>
<td>September</td>
<td>Walked on some leaves; bitten; shone torch; killed</td>
<td>Foot</td>
<td></td>
<td></td>
<td>Severe; generalized</td>
<td>No progression of quarter</td>
</tr>
</tbody>
</table>

1 Symbols: S., snake; N.G., none given; P., paralysis.

Australian snake (the taipan was not studied). Fairley and Spath (1928) considered the death adder to be, for its size, the world's most deadly terrestrial reptile.

Experiments by Fairley (1928) on sheep, and by Kellaway (1929a) on monkeys, horses, cats, rabbits, guinea-pigs, rats, and mice, showed the venom to be strongly neurotoxic. The neurotoxic action was due to a peripheral curare-like neuro-muscular block (Kellaway, Cherry and Williams, 1922). In the monkey the venom produced ptosis, dyspnea, ataxia and paralysis. There was no local lesion at the site of injection of the venom. The heart was found to be still beating when the animal was examined at autopsy. No significant lesion was seen at this examination.

Some hemorrhages were present in the lungs of the rabbit, horse, guinea-pig and rat, and from this Kellaway (1929a) concluded that the venom had a hemolytic action. The venom was found to be hemolytic only in the horse and to be mildly hemolytic in the rat. Unlike many other Australian venoms (tiger snake, black snake, brown snake and taipan), death adder venom contained no thrombolytic activity and did not produce intravascular coagulation. The venom was considered to be strongly neurotoxic and mildly hemolytic, with a possible weak anticoagulant action (Kellaway, 1929b).

Earlier Clinical Work
Isolated case reports of death adder bites were reported by Campbell (1891), Clatworthy (1891), Parry (1891), Hunt (1901), Croll (1912), Fairley (1929a) and Flecker (1940). The symptoms recorded were vomiting and those due to the effects of the paralysis of the bulbar and ocular muscles. Tidswell (1906), in reviewing police reports of snake bite cases from New South Wales and published case reports from other States from 1891 to 1905, found 10 death adder bites with five deaths. From these figures the 50% mortality rate of death adder bites is quoted. Ferguson (1926) reviewed police reports of snake bite cases in New South Wales from 1906 to 1924, and found no cases of death adder bite.

PRESENT STUDY
Over a six-year period (1960 to 1965), 15 patients who were considered to have been bitten by death adders were admitted to the Port Moresby General Hospital. Nine of these patients were studied personally by the author. Details of five of the 15 cases were included in a previous paper on snake bite in Papua (Campbell, 1964). The dead snake was brought to the hospital by eight patients, identified by the patient or employer as a death adder (five cases), or the description of the snake and the findings on examination of the blood were compatible with a death adder bite (two cases).

Some of the clinical details are summarized in Table 1, and the results of blood tests carried out on six patients are summarized in Table 2.

All the patients were bitten while in forested country in Papua; 10 in the Brown River-Vanapa River rain forest behind Port Moresby. Three patients were bitten at night time. Only one patient saw the snake before it bit him, while at least five people trod on the snake. After biting, the snake often hung on until killed. Eleven of the patients killed the snakes. The snakes brought to the hospital were small and immature.

If the patient was standing when bitten, the site of the bite was always at or below the level of the malleoli, most commonly on the toes or the dorsum of the foot immediately behind the toes. Five patients complained of slight pain in the bitten part. The wounds had been incised in 11 cases. Two puncture wounds were seen only in one patient. Three or more indefinite marks were present in the region of the bite in two patients (local wound was not described in one patient). Slight edema surrounded the bitten area in four patients.

The symptoms were mild. The patient complained of a slight brief headache and/or vomited once or twice, and/or complained of pain, which was sometimes severe, in the lymph glands draining the bitten area. The visual disturbance was difficult to define, being manifest as a blurring of vision or difficulty in seeing clearly. One or more or all of these symptoms were sometimes absent, and the first indication that, venom had been injected was the onset of paralysis of the bulbar or ocular muscles.

The earliest signs of envenomation, which sometimes appeared within one hour of the bite, were tenderness and enlargement of the regional lymph nodes. These signs were not constant, and were absent in one of the most severely poisoned patients.
Ptosis was the earliest sign of involvement of the nervous system by the venom. The most severely paralysed patients had a generalised paralysis of the voluntary muscles, which threatened life by producing respiratory obstruction or respiratory insufficiency.

The rabbit anti-fibrin test (Ferreira and Murat, 1963) is a flocculation test for human fibrin degradation products and gives a positive result in debridement syndromes, whatever their aetiology. A fibrinogen titre (Sharp et alii, 1968) of 1/32 which was found in four cases, is considered normal for Papuans (Booth and MacGregor, 1965). The clot quality which was observed by allowing the blood to clot in a centrifuge tube was good in the six patients tested.

The euglobulin lysis time, Fearnley fibrinolysis time and the fibrin polymerization test also gave normal results in one patient with serious envenomation.

**DISCUSSION**

A venomous snake bite occurring during the night in Australia and New Guinea is more likely to be caused by a death adder than any other snake (Fairley and Spiatt, 1929). If the snake bite occurs during the day, the death adder should be seen and, because of its characteristic appearance, should be killed. The snake can usually not be killed, because it tends to hang on after biting or tends not to move away quickly if it lets go.

As the death adder is a short snake, it strikes low. A snake bite above the level of the ankle in an adult is unlikely to have been caused by a death adder. The snake bites tenaciously and is more likely to inject venom than the larger venomous snakes. After Papuan bites, more and taipan bites, the paralysis slowly recovers over a period of two to seven or more days after the bite, and even at the end of a week, although all movements are present, muscle power is still not normal and may not be normal for another week.

Probably only two of the 15 patients would have died without treatment. The condition of some of the patients with symptoms and early signs of envenomation might have progressed to a fatal issue without antivenene, but this is indefinite as the condition of two patients with similar symptoms and early signs, who were not given antivenene, did not progress. A possible case fatality rate of 15% may be because of the fact that all the snakes brought in were small. Nevertheless, the overall mortality rate of death adder bites probably approximates more to this figure than the commonly quoted 50%.

**SUMMARY**

1. The characteristics of the death adder and earlier experimental work on its biting apparatus and venom are summarized.

2. The clinical details of 15 patients who were bitten by death adders are summarized.

3. The effects of the death adder bite differ in two important respects from those of the bites of the other common Australasian venomous snakes: the death adder venom has no clinical effect on blood coagulation; and the paralysis produced by the venom is reversed by antivenene.
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