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

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THE death adder was one of the first venomous reptiles to be described in Australia (Shaw, 1801). 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 Kairuku sub-district of Papua call this snake *afi*, 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, 1966).

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 tread 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 defæcating 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

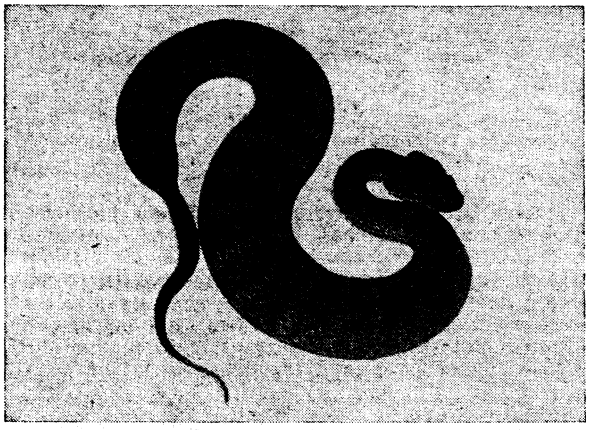

FIGURE 1: The death adder (*Acanthophis antarcticus*) (Photograph, H. G. Cogger).

(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 lævus*, 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, 1929*b*). Its fangs were larger and more mobile than any other Australian venomous snake studied by Fairley (1929*b*). Tidswell (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, 1929*a*). Kellaway (1929*a*) estimated the certain lethal dose in man to be 0·025 to 0·15 mg. per kilogram. Fairley (1929*a*) concluded that the death adder had the most efficient biting apparatus and highest venom yield of any

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TABLE 1

Summary of Clinical Details of 15 Patients with Death Adder Bites

Patient's Number	Sex	Approximate Age (Years)	Time of Bite	Month of Bite	Circumstances Bite[1]	Site of Bite	Symptoms: Headache	Symptoms: Vomiting	Symptoms: Pain Reg. Glands	Signs: Visual Disturbance	Signs: Glands—Tender, Enlarged	Signs: Muscle Paralysis	Result of Urine Testing	Progress after Antivenene[1]
3548	M.	35	6.30 p.m.	October	Running behind horse; trod on S.; S. hung on; killed	Toe	+	–	–	–	+	–	–	N.G.; no progression
3774	M.	40	10 a.m.	November	Trod on S.; S. hung on; killed	Ankle	–	–	–	–	–	–	–	No progression
3813	M.	25	2 p.m.	November	Sitting under house; S. hung on; killed	Toe	–	–	–	–	+	–	–	N.G.; no progression
4229	M.	35	11 a.m.	December	Trod on S.; S. let go; killed	Toe	–	–	–	–	–	–	–	N.G.; no progression
11294	M.	40	11.30 a.m.	August	Walking; S. " came at him "; kicked off; killed	Ankle	–	–	+	–	+	–	–	No progression
15125	M.	25	3 p.m.	December	Not recorded; S. killed	Ankle	–	–	–	–	–	–	–	No progression
16722	M.	20	11 a.m.	March	Not recorded; S. killed	Foot	–	–	–	–	–	–	–	No progression
17896	M.	25	5.30 p.m.	May	Not recorded	Heel	–	–	–	–	+	–	–	No progression
25948	M.	35	1 p.m.	June	Lay on leaves; S. under leaves; S. killed	Leg	–	–	–	–	–	–	–	N.G.; no progression
27853	M.	30	9 a.m.	October	Kicked off; S. killed	Toe	–	–	–	–	+	–	–	No progression
28040	M.	25	7.30 p.m.	October	On road through grass; bitten; did not see S.; dark	Foot	–	+	–	+	+	Ptosis	Albumin one-eighth	P.; reversed
28488	M.	20	8.30 a.m.	November	Picked up grass; S. hung on; pulled off; thrown away	Finger	–	–	–	+	+	Severe, generalized	–	P.; reversed
32029	F.	35	10 a.m.	August	Trod on S.; S. let go; killed	Toe	–	–	–	+	–	Severe, generalized	Albumin, one-quarter	P.; reversed
35266	M.	25	3 a.m.	September	Trod on S.; kicked S. free	Heel	+	+	+	–	+	–	–	No progression
35645	M.	35	7.30 p.m.	September	Walked on some leaves; bitten; shone torch; killed S.	Foot	–	–	+	–	+	–	–	No progression

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

Australian snake (the taipan was not studied). Fairley and Splatt (1929) considered the death adder to be, for its size, the world's most deadly terrestrial reptile.

Experiments by Fairley (1929*a*) on sheep, and by Kellaway (1929*a*) 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, 1932). In the monkey the venom produced ptosis, dyspnœa, 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 hæmorrhages were present in the lungs of the rabbit, horse, guinea-pig and rat, and from this Kellaway (1929*a*) concluded that the venom had some cytolytic action. The venom was found to be hæmolytic only in the horse and to be mildly hæmolytic in the rat. Unlike many other Australian venoms (tiger snake, black snake, brown snake and taipan), death adder venom contained no thrombase and did not produce intravascular coagulation. The venom was considered to be strongly neurotoxic and mildly hæmolytic, with a possible weak anticoagulant action (Kellaway, 1929*b*).

Earlier Clinical Work

Isolated case reports of death adder bites were reported by Campbell (1891), Clatworthy (1891), Parry (1891), Hunt (1891), Croll (1912), Fairley (1929*a*) 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 fom 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 œdema 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.

TABLE 2

Results of Blood Tests Performed on Patients with Death Adder Bites

Patient's Number	White Cell Count (per Cubic) Millimetre)	Percentage of Neutrophils	Platelets (on Smear)	Erythrocyte Sedimentation Rate (Wintrobe) (Millimetres in One Hour)	Bleeding Time (Duke) (Minutes)	Coagulation Time (Lee and White) (Minutes)	Clot Quality	Result of Rabbit Antifibrin Test	Fibrinogen Titre
27853	6400	58	Normal	6	—	4·5	Good	Negative	1/32
28040	12,000	82	Normal	20	4	5·5	Good	Negative	1/16
28488	9400	84	Normal	2	4·25	4·5	Good	Negative	1/32
32029	15,200	88	Normal	7	3	4	Good	Negative	1/32
35266	16,700	91	Normal	2	—	4	Good	Negative	1/32
35645	9000	78	Normal	5	2·5	6	Good	Negative	—

Ptosis was the earliest sign of involvement of the nervous system by the venom. The most severely paralysed patients had a generalized 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 defibrination syndromes, whatever their ætiology. A fibrinogen titre (Sharp *et alii*, 1958) 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 euglobin 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 Splatt, 1929). If the snake bite occurs during the day, the death adder should be seen and, because of its characteristic appearance, should be recognized. The snake can usually 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, the taipan and Papuan black snake, which strike quickly and disappear very rapidly, often not being seen by the victim.

The local wounds or incisions do not continue to bleed. If the puncture wounds are bleeding some hours after a snake bite, a death adder bite is unlikely.

The early symptoms and signs of death adder envenomation are similar to those which occur with other Australian and New Guinea venomous snakes, except that abdominal pain and symptoms caused by the thrombase and hæmorrhagin in the venom, such as the spitting or vomiting of blood, or the results of the hæmolysin, such as passing red or black urine, do not occur.

There is no clinically demonstrable disturbance of blood coagulation after death adder bites. The blood clots well and the bleeding and coagulation times are normal. No breakdown products of fibrin occur in the blood, and the rabbit anti-fibrin test gives a negative result. There is no evidence of any fibrinolytic activity. The absence of a clinically demonstrable disturbance of blood coagulation distinguishes death adder bites from the bites of the other common Australian and Papuan venomous snakes. The venoms of the tiger snake (*Notechis scutatus*), the common black snake (*Pseudechis porphyriacus*), the common brown snake (*Demansia textilis*), the taipan (*Oxyuranus scutellatus*), and the Papuan black snake (*Pseudechis papuanus*), all contain thrombase.

The erythrocyte sedimentation rate was usually normal. A neutrophil leucocytosis was a variable phenomenon. The urine was usually free of albumin, and examination of the centrifuged deposit of urine did not reveal any abnormality. Therefore, laboratory tests do not help the physician to decide whether death adder venom has been injected. This must be a clinical assessment. If symptoms and signs of envenomation are present, laboratory test results (normal bleeding and coagulation times, and particularly a negative result to the rabbit anti-fibrin test) do help to differentiate death adder bites from other venomous snake bites.

Another extremely important difference between death adder venom and the thrombase containing venoms is that the paralysis produced by death adder venom is rapidly and completely reversible by antivenene (three to six ampoules of New Guinea polyvalent antivenene plus 3,000 to 12,000 units of death adder antivenene given by intravenous injection) at least up to seven hours after the bite in my experience. I have never seen (in over 40 cases) the paralysis produced by taipan and Papuan black snake venom reversed, despite the use of large doses of antivenene. Kellaway (1932) also noted that the paralysis produced in animals by tiger snake venom was very difficult to reverse with antivenene. It is possible that all Australasian thrombase-containing venoms behave in this fashion.

The reversal of the paralysis after antivenene administration is a most dramatic phenomenon. Tracheotomies were performed on the two most severely paralysed patients, and one of them also required artificial respiration. Yet by the time the patients returned from the operating theatre one hour after the antivenene was given, the muscle power was almost normal and the need for a tracheotomy had passed. Within two hours the muscular power was normal. After Papuan black snake 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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