

Reports of Cases.

A FURTHER CASE OF SNAKE-BITE BY A TAIPAN ENDING FATALLY.

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KELLAWAY AND WILLIAMS (1929) published experimental evidence that the venom of the taipan (*Oxyuranus scutellatus*) was predominantly neurotoxic. This point is again referred to by Kellaway (1942), who made this further statement: "In animals poisoned by neurotoxic venoms 'starting movements' are observed in the late stages of poisoning. These convulsive movements are probably central and may be caused by low-grade asphyxia or directly by the action of the venom."

The similarity of the neurotoxin of the taipan to that of the tiger snake (*Notechis scutatus*) was suggested by Kellaway (1929) when he demonstrated cross-immunization to taipan venom from tiger snake antivenine.

Reference to previous cases of taipan bite, described in the literature, shows much unreliability, as in many of the cases neither was the snake clearly seen and identified nor were the clinical notes always complete enough to allow conclusions to be drawn as to the effect of the venom.

In one of the cases recorded by Flecker (1940) a permanent loss of smell and taste resulted—a feature not unusual with other neurotoxins. In the same account, two fatal cases are described (the snake not having been positively identified); one case is characterized by respiratory failure, the other by immediate onset of convulsions.

Flecker (1944) described a further two fatal cases, one of which was not identified as a taipan bite. Both cases, again, were characterized by convulsions.

Further reference to a case described by Reid and Flecker (1950), in which the snake was positively identified as a taipan, shows again all the clinical signs of a neurotoxin. In this case there was the picture of peripheral circulatory failure—a feature which, although common with the cytolytic toxins of black snake and copperhead snake venoms (Kellaway, 1942), is not expected with the taipan. Reference to the clinical details of this case shows that the patient lost a considerable quantity of blood with the scarification, his hæmoglobin value being 40% seven days after the bite.

The present account deals with a case of snake-bite; the snake was captured alive and was identified positively as a taipan.

Clinical Record.

The victim was K.B., aged twenty years, of slight physique, who described himself as "an amateur snake collector". It is stated by relatives that he had been bitten by several snakes previously.

The patient arrived at Cairns during the early part of July, 1950, fully determined to secure a taipan alive. After several unsuccessful journeys inland, in which he collected many other snakes, including non-venomous varieties, he returned to Cairns. On July 26 he received word that a taipan had been killed under a home at Anderson Street, on the outskirts of the city. He immediately made known his determination to seek out the mate of this snake the next morning. In spite of warnings from local residents, he set off, searching an area of rubbish dump off Anderson Street. From details related by the victim to Mr. S. E. Stephens, of Edgehill, it is possible to reconstruct the circumstances of the capture. Approaching a pile of rubbish, he came upon the taipan coiled up in the sun. Quickly putting out his foot in order to immobilize the reptile, he took hold of it by the neck with the left hand. Allowing the reptile to coil around his body and holding the tail in his right hand he proceeded along the bush

track a distance of about one-half to one mile, until he reached the main road to Edgehill. Here he hailed a truck, and still holding the reptile in this manner, climbed into the cabin beside the driver, asking to be driven to Mr. Stephens to have the snake identified.

Upon arrival at the residence the reptile was immediately identified as a taipan. As he loosened his grip for a moment to secure a better hold, the reptile broke loose, dropped to the ground, and in quick succession struck at his boot, his trouser cuff and then successfully at his left hand. The snake was then secured, and a tourniquet was applied to his left arm. This was at 10.30 a.m.

Upon his arrival at Cairns Base Hospital at 11 a.m., the patient gave the general impression of bravado and excitement, showing greater interest in the welfare and comfort of the reptile than himself. When asked why he had not scarified the wound, he answered that in his opinion scarification "wasn't worth the trouble". He stated that he was not worried about himself, as he believed that snake victims died from fright more than from the effects of the poison.

Examination of the patient's left hand revealed two puncture wounds on the left thenar eminence, with a double row of puncture wounds running laterally. The arm had a double tourniquet applied above the elbow. No radial pulse was detected. General examination revealed no abnormality. The pulse rate was 96 per minute.

Scarification of the wound was not attempted, owing to the half-hour delay; 4500 units of tiger snake antivenine were given by the intravenous route. As the patient said that he was a hay fever subject, "Anthisan" (0.1 gramme) was given three times per day. The tourniquet was removed every ten minutes for a period of ten seconds until 12.15 p.m., when it was dispensed with completely.

At 3 p.m. the patient complained of blurred vision. He had vomited yellowish fluid three times and developed a severe headache straight after. Examination revealed a slight ptosis and weakness of both masseter muscles. His pulse rate was 120 per minute, the pulse being of good volume, and his body temperature, taken by mouth, was 98.6° F. The skin felt clammy. The affected hand was now red and swollen.

A hypodermic injection of five minims of 1:1000 adrenaline solution and five minims of "Necysthaphalin" was given. "Anacardone" (two millilitres) was also given by intramuscular injection. At 7 p.m. the patient had vomited yellowish fluid twice. Examination revealed extension of the paralytic process; slight internal strabismus was now present and ptosis was extreme. He was unable to move his tongue appreciably; his mouth gaped and its floor sagged under the effects of gravity. As he was unable to swallow, it was necessary to aspirate saliva continuously. The patient was unable to phonate, and had to resort to pencil and paper. The sterno-mastoid muscles were weak upon both sides and some upper intercostal paralysis was noted. The pulse was full and the rate was 120 per minute. The body temperature was 97.2° F. A further 3000 units of tiger snake antivenine were given intramuscularly. *Mistura Potassii Citratis*, half an ounce, was given four-hourly in addition to five milligrammes of picrotoxin intramuscularly.

Examination of the patient at 8 p.m. revealed almost complete loss of intercostal breathing, complete facial paralysis and some weakening of upper and lower limb musculature with corresponding loss of tendon reflexes.

Shortly before the patient was transferred to the respirator room a further 4500 units of tiger snake antivenine and one millilitre of "Anacardone" were given intramuscularly.

At 8.25 p.m. respiratory distress was apparent; yet when the patient was placed in the artificial respirator he fought strenuously against the artificial rhythm. This necessitated his removal, and dependence upon the administration of oxygen and posturing into Fowler's position. When this was done the patient showed little respiratory distress.

When he was examined at 9 p.m., the patient's condition was much the same. A hypodermic injection of atropine (1/100 grain) was successful in reducing salivary secretion.

During the night the patient slept normally and appeared to be capable of sufficient respiratory exchange.

Slight cyanosis was reported at 9 a.m. the next day. The patient was restless and his temperature was 96.0° F. The oxygen flow was increased to four litres per minute with a good response. The administration of dextrose solution (5%) was started by the intravenous route at the rate of 80 drops per minute. However, at 9.30 a.m. less than 500 millilitres had been given when the patient became restless and dragged the needle out. Immediately after this (10.50 a.m.) he had a rigor, and his pulse was rapid and only just perceptible. One millilitre of pitressin and five milligrammes of "Neosynephrin" were given immediately. Some improvement was noted by 11.25 a.m.; ventilation was deeper, and the patient was conscious. The radial pulse was slower and fuller.

At 12.30 p.m. the patient became restless, respiratory movements became shallow and moderate cyanosis developed. The oxygen administration from a standard "B.L.M." mask was increased, and two millilitres of "Anacardone" were given intramuscularly. However, cyanosis became intense and respiratory movements disappeared; but the pulse rate did not increase and the pulse was full. Manual artificial respiration was instituted preparatory to insertion of the patient into the respirator. No ventilation occurred when he was put into the respirator. As it was thought that a mucous clot might have blocked the airway, the patient was then subjected to direct laryngoscopy. The chords were seen to be clear and only very little mucus was present. A McGill's tube was inserted to ensure an airway. Artificial respiration had continued in the meantime.

At 1.20 p.m. one millilitre of "Anacardone" was given intramuscularly. The patient remained cyanotic and cold, although respiratory exchange was adequate.

At 1.30 the pulse failed and no signs of life were detected.

At the post-mortem examination it was noted that around the two puncture wounds was a quarter-inch area of dry, black tissue resembling dry gangrene.

Conclusion.

A case of bite from *Oxyuranus scutellatus* ending fatally is described.

The patient probably received a small dose of venom, as is suggested by the late onset of symptoms and the absence of convulsions.

One was unable to detect any amelioration of symptoms with the use of 12,000 units of tiger snake antivenine. This is surprising in view of the findings of Kellaway (1929) and of Flecker and Reid (1949).

This case illustrates the fact that preconceived ideas on the part of the patient in respect to first aid may seriously affect his progress.

Indicative of the effect of a neurotoxin, the symptoms in this case are predominantly those of slowly developing flaccid paralysis and bulbar palsy.

The use of drugs designed to stimulate the respiratory centre had no particular effect on the paralysis of this patient. This is significant, perhaps, in face of Kellaway's statement in respect to the neurotoxins of Australian snakes. Kellaway states that apart from central effects, "... the venoms have a curari-like action on motor endings, and a further direct action upon muscle itself. To this curari-like action the phrenic end plates in the diaphragm are particularly sensitive; and partial curarization of the diaphragm plays an important part in the failure of respiration which is the commonest cause of death after the bites of these snakes".

References.

- Flecker, H. (1940), "Snake Bite in Practice", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 8.
— (1944), "More Fatal Cases of Bites of the Talpan", *ibidem*, Volume II, page 383.
Kellaway, C. H. (1929), "Snake Venoms", *ibidem*, Volume II, page 536.
— (1942), "The Symptomatology and Treatment of the Bites of Australian Snakes", *ibidem*, Volume II, page 171.