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STONEFISH

GENUS SYNANCEIA (LINNAEUS), STONEFISHES: S. VERRUCOSA (BLOCH AND SCHNEIDER) AND S. TRACHYNIS RICHARDSON

Struan Sutherland

> 'Suppose that fella nail go along your foot, you sing out all a same bullocky all night. Leg belonga you swell up and jump about? Bingie (belly) belonga you, sore-fella. Might you die.'

This description of a Stonefish sting by an Aboriginal first appeared in Confessions of a Beachcomber in 1908.[1] There has been little change! Stonefish are still the most dangerous venomous fish in the world and have a justifiably evil reputation in tropical and subtropical waters.

The names given to Stonefish in general are self-explanatory: the Devil Fish, the Warty Ghoul and, best of all, Nohu or the Waiting One. Since ancient times the Australian Aboriginal has been aware of the danger of these creatures. Roughley[2] described how certain north Queensland tribes taught the young to beware of Stonefish as part of the initiation ceremonies, by using a beeswax model of a Stonefish and mimicking the illness produced by its sting.

DISTRIBUTION

Species of Stonefish are found throughout the whole Indo-Pacific region and, in Australia, on the northern coasts from near Brisbane to 500 km north of Perth. Of the two Australian species, the most important is S. trachynis, the Estuarine Stonefish, which inhabits shallow water from Moreton Bay in southern Queensland to as far west as Houtman's Abrolhos Islands in Western Australia. S. verrucosa, the Reef Stonefish, is equally dangerous, but appears to be relatively uncommon.[3] S. horrida (Linnaeus), the Indian Stonefish, was at one time incorrectly believed to occur in Australian waters.

DESCRIPTION

Stonefish are so called, because in their natural habitat they are easily mistaken for a piece of rock or dead coral which has become encrusted with marine growth. This camouflage which allows them to snap up any passing prey, makes it almost impossible for swimmers to see and avoid them.

S. trachynis and S. verrucosa, both heavily built fish, are particularly unattractive. Endean[3] found the mean standard length of fifty-two specimens of S. trachynis was 20.3 cm and Grant[4] records a maximum length of 47 cm. Stonefish do not have scales and their soft skin is covered with a variety of tubercles. Cameron et al[5] reported that the copious secretions from these tubercles contained two fractions (crinotoxins) which were toxic to small marine creatures. These secretions which are bitter to taste, possibly to discourage potential predators, are however, unlikely to cause any injury to man. The small eyes are difficult to see and are carried on bony protrusions. The two species can be distinguished by the fact that the eyes of S. verrucosa are on quite separate protrusions, but those of S. trachynis share the same boss. Moreover, the latter has a yellowish green mouth cavity, while that of S. verrucosa is whitish flecked with red.[4] Apart from these minor differences, the two species are very similar and, since practically all research work and clinical cases to date involve the commoner S. trachynis, little further reference will be made to S. verrucosa.

Habits

The Stonefish has very large pectoral fins, which allow it rapidly to dredge sand or mud from beneath itself, so that it can settle deeply with only its mouth and eyes fully exposed. From this position, motionless, it can suck a passing fish into its very large mouth, After it has caught a victim, it will rescoop its rest place and settle down again in the sand or mud. The large pectoral fins give the fish a strange, floppy swimming motion, which is best seen in an aquarium tank. In a tank, a captured Stonefish tends to lose a lot of its camouflage due to the activity of the aquarium filter system. Stonefish may tolerate brackish water and, in fact, can survive at least twenty-four hours out of the water, provided they are kept cool and moist.

Venom Apparatus

The venom apparatus of this fish is purely defensive, and plays no role in the capture of its prey.

S. trachynis has thirteen dorsal spines, each of which carries two venom glands. The spines are stoutly built and taper to fine points. Each spine has two lateral grooves, into which the spindle-shaped venom glands are firmly attached by connective tissue, especially proximally. Histologically the glands, which are surrounded by thick fibrous tissue, contain discrete granules of proteinaceous venom. The granules are found both in the tortuous ducts of the gland and secretory cells. The distal portion of the gland becomes modified to form the slender duct, which is actually blocked by fine fibrous tissue stretching across the groove it occupies. Thus, in the normal course of events, there is no apparent way in which the Stonefish can voluntarily expel its venom.

When disturbed, the spines become erect, the anterior and longer first three spines are then nearly vertical while the other spines point backwards at lower angles. The tip of each spine is clearly visible, projecting as it does through a small hole in the sheath of skin covering the spine. Should an object, for example, a human foot, be pressed down on the spine, then, as the spine penetrates the object, the small but tight aperture in the duct is jammed down over the venom gland. As the venom gland is firmly surrounded by fibrous or rigid tissue, liquid venom and venom gland tissue are forced up the duct which has become unblocked. When Endean[3] forced rubber sheeting down over the dorsal spines, he described the escape of material from the venom gland as 'a violent

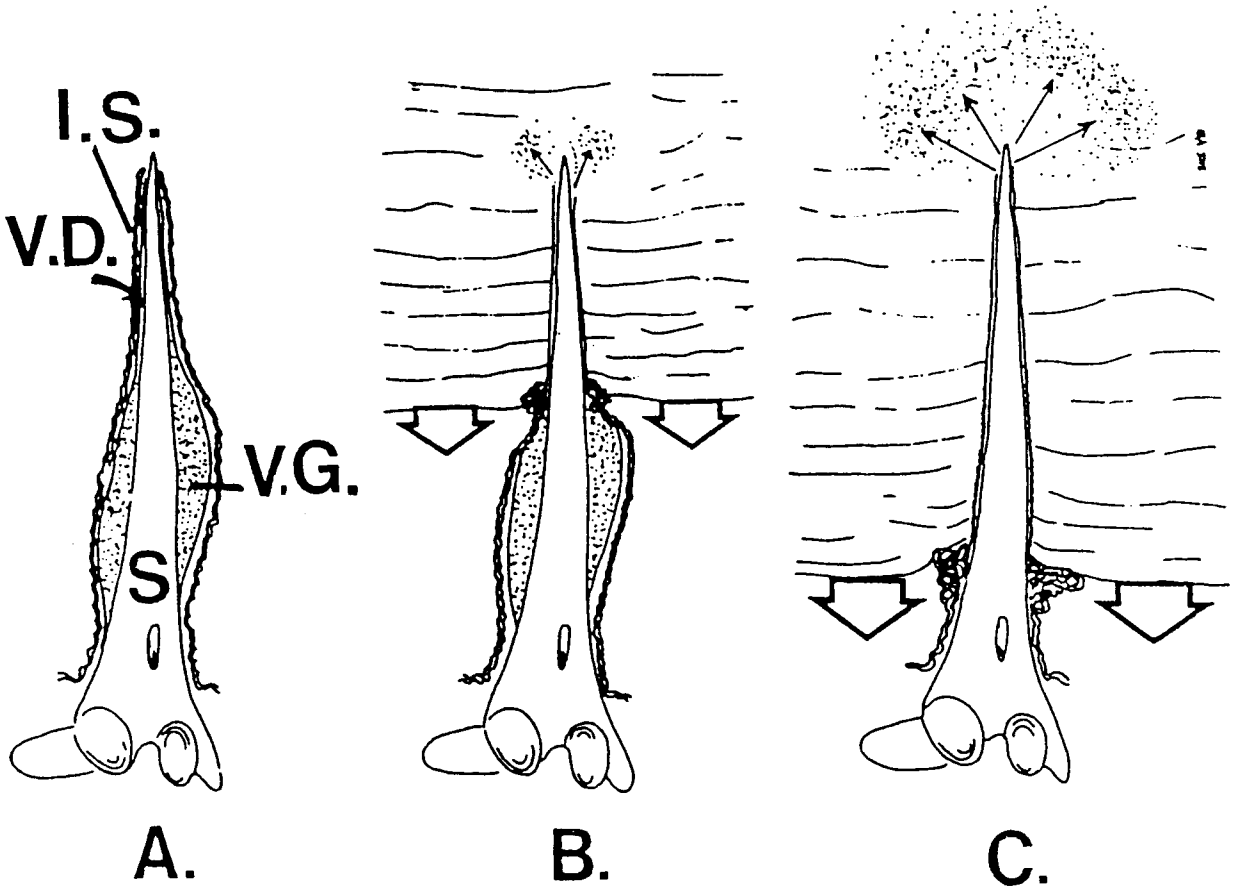

Figure 1. Venom apparatus of a Stonefish:

A. Dorsal spine viewed from behind, showing paired lateral venom glands (VG). Note tip of spine (S) and narrow venom ducts (VD), both passing through a narrow opening in the tough integumentary sheath (IS).

B & C A human has trodden upon the fish, the skin is pushed down the spine (large arrows) and the venom gland is compressed by the crumpled sheath. This pressure forces the venom gland to empty up the narrow duct, and venom and glandular tissue spurt (small arrows) into the deep tissue of the foot.

expulsion'. He demonstrated that pressure directed upon the venom glands did not release venom. Wiener[6] on the other hand, considered great care had to be taken to avoid pressure on the glands when they were being dissected out, otherwise venom was easily lost. Venom liberated by the sheath being forced over the gland contained a mixture of granules and venom gland cells, either singly or in groups. Endean considered that regeneration of both venom gland and venom stores took some three weeks.

Endean recorded that, in an average sized fish (20 cm in length), .the venom glands are 1 cm in length and 2 mm in diameter. The venom duct is some 7 mm in length. He estimated that the depth of penetration of the spine required to cause venom release would be 0.6 cm in the case of a small fish to 1.0 cm with a mature fish. Endean stressed that even a small Stonefish 7.8 cm long had well developed spines and venom glands.

Venom

Duhig and Jones,[7] who pioneered studies on Stonefish venom, showed the venom remained toxic in the venom glands for several days after the death of the fish. They found that the venom was haemolytic in vitro to a variety of erythrocytes, but only in the absence of serum. This effect is not considered of clinical significance.

Wiener[6] dissected out the venom glands from living specimens of S. trachynis and then freeze dried the venom he collected. He found that as much as 0.03 ml could be aspirated from a single venom gland. Dissection of seven specimens, of average length 26 cm and average weight 0.7 kg, yielded an average of 6 mg of venom per spine. The total yield of venom per fish ranged from 49 mg to 88 mg. Wiener noted that often the venom glands were not full and sometimes the gland had been replaced, possibly with strands of connective tissue.

Wiener also studied the toxicity of S. trachynis in a number of animals. He resuspended the venom in saline buffered at 7.4 and then centrifuged it to remove cellular material and debris. He estimated that 25 to 30 per cent of the harvested material was lost in this way, but decided to ignore this loss when estimating toxicities which related directly to the starting material. Thus, the quantities of venom injected are 25 to 30 per cent lower than stated. In

mice of 15 g weight the LD50 of venom was 0.004 mg intracerebrally, 0.007 mg intravenously, 0.03 mg intraperitoneally and 0.05 mg subcutaneously. The figure for the intravenous LD50 compares favourably with that of 0.004 mg obtained by Saunders[8] using venom freshly extracted from S. verrucosa.

In guinea pigs of 250g body weight, a subcutaneous dose of 4 mg of venom caused death within three hours. Death was preceded by weakness and coma. A guinea pig dosed with 2 mg died on the third day. Oedema and necrosis had developed at the site of the injection. Guinea pigs receiving 1 mg or 0.6 mg died on the sixth and eleventh day respectively. A dose of 0.2 mg caused a skin lesion only. Post mortem studies on these animals was not instructive.

Wiener determined that the minimum ischaemic dose of venom (intradermal) in a rabbit was 0.002 mg in a volume of 0.05 ml. When he gave an intravenous injection of venom (0.17 mg/kg) into an anaesthetized, monitored dog, transient hypertension and apnoea occurred. Larger doses produced cardiac and respiratory arrest within two minutes. Saunders[8] found that, when S. verrucosa was injected intravenously into monitored rabbits, hypotension developed which was associated with electrocardiographic changes such as atrioventricular block and ventricular fibrillation. Saunders[9] found .also that the actions and toxicity of S. horrida venom are similar to S. verrucosa venom.

Austin et al[10] presumably using S. trachynis venom rather than S. horrida, found that the venom when injected intravenously in rabbits caused hypotension, respiration distress and muscular paralysis. These studies suggested that Stonefish venom has powerful myotoxic properties which can cause loss of function of all muscle types. The paralysis appeared to be caused by a conduction block in the muscle due to slow polarisation. They considered that the death of the rabbits was due to paralysis of the diaphragm, rather than myotoxic effects on the heart muscle.

Austin et al[10] undertook some biochemical studies upon the venoms of S. trachynis and S. verrucosa. They found that the major toxin of both is a labile protein with a molecular weight of the order of 150 000. The venom was shown to contain a potent hyaluronidase and a capillary permeability factor. No protease or phospholipase A activity could be found, the venom had no effect on blood clotting, nor did it cause haemolysis. Deakins and Saunders[11] investigated methods of isolating the toxin and found starch/gel electrophoresis to be a suitable procedure. They also found the toxin to be labile, but it could be stabilized for some months if lyophilized with 5 per cent sucrose.

HUMAN ENVENOMATION

Stonefish stings are extremely painful. The pain is simultaneous to penetration by the spine and rapidly worsens. Swelling of the envenomed area develops at the same time and may soon extend. The victim may be irrational because of the pain. The severity of the signs and symptoms is usually in direct proportion to the depth of penetration of the spine(s) and the number of spines involved.

As well as the local effects, muscle weakness and paralysis develop in the affected limb and varying degrees of shock may occur. Human fatalities have been caused by Stonefish stings in the Indo-Pacific regions, but no known deaths have occurred near Australian waters.

Smith[12] described two deaths due to S. verrucosa. One occurred in the Seychelles in March 1956, when a healthy 15-year-old boy collapsed became cyanotic and died within a short time(not defined) of receiving the punctures to his foot. He suffered terrible agony before he became unconscious. The other fatality involved an adult man who died within an hour of standing on a Stonefish at Pinda, Mozambique. He collapsed and was 'almost delirious' prior to lapsing into unconsciousness.

CHOICE OF ANTIVENOM

Stonefish antivenom will neutralize the venoms of S. trachynis, S. verrucosa and S. horrida. It is prepared by immunizing horses with the venom of S. trachynis and is available as a pure equine $F(ab)_2$ preparation. Ampoules contain approximately 2 ml, which will in vitro neutralise 20 mg of venom.

Miss Wendy Cowling kindly prepared the following brief survey of reports of Stonefish antivenom usage over the period 1965 to 1981 (inclusive).

A total of 267 reports were received at the Commonwealth Serum Laboratories from the following sources: Queensland 129, Northern Territory 35, Western Australia 54 and overseas 49. Males were stung in 83 per cent of cases and, apart from three persons injured on their arms or wrists, all stings occurred either on the hands (36 per cent) or the feet (63 per cent). The victim's average age was 27 years and the median age was 25 years. Four children under the age of 5 years received antivenom for stings. No deaths were reported.

Sutherland and Trinca[13] published details of Stonefish stings which occurred specifically in Queensland from 1967 to 1978. In the period considered, ninety-five Queensland cases received antivenom and eighty-seven of the cases involved males. Injuries were restricted to the hands or feet twenty-three of the former and seventy-two of the latter. The seasonal incidence was as follows: January (7), February (3), March (12), April (12), May (8), June (6), July (7), August (10), September (10), October (2), November (7), December (11).

The higher rate of stings amongst males compared with females probably reflects the men's professional or amateur fishing activities. The high incidence in March/April and August/September may be related to Easter and school holidays. The limitation of injuries to either the hands or the feet suggests that many of these stings could have been avoided with appropriate care.

FIRST AID

No attempt to should be made to retard the movement of venom from the stung area. To delay the escape of venom will only enhance local pain and tissue damage. Relief of pain is the most urgent requirement. In minor cases this may be achieved by bathing the stung area in warm to hot water (but not scalding the skin). However, hot water is not often available. In severe cases. pain relief may only be obtained by the combined use of antivenom and potent opiate drugs, such as morphine or pethidine.

CLINICAL MANAGEMENT

Local Anaesthetic As Additional First Aid

It is often recommended that a local anaesthetic, such as lignocaine or the longer acting bupivacaine, be injected into the track of the sting and the surrounding area. Such a procedure may afford pain relief for sixty minutes or so, but should only be considered as an adjunct to the administration of antivenom. Mixtures of adrenaline and lignocaine should never be used to provide local pain relief, since adrenaline will decrease the blood flow in the region.

Very rarely, when there are multiple stings and the agonized victim is almost irrational and uncontrollable, the use of a regional block, which is easily performed by an anaesthetist, should be considered. A general anaesthetic is not recommended. A regional block, if bupivacaine is used, has the advantage of lasting eight to twelve hours and so will not enhance the systemic effects of the venom and be much safer for the patient than a general anaesthetic.

Other Local Measures

Incision of the stung area. Quite often incisions are made into the stung area to promote bleeding and, hopefully, the escape of venom. The writer does not usually advise such measures, but accepts the fact that, in very severe cases, if they are performed professionally, the clinical outcome may be improved.

Emetine. Injection of emetine hydrochloride (65 m/ml) into and around the wound has produced some relief of pain, probably due to the acidity of the solution inactivating the venom. However, there are now few commercial sources of emetine hydrochloride.

Potassium permanganate (Condy's Crystals). This solution should not be used, as it will merely cause local tissue damage and aggravate the wound. It is probable that, as was shown with trypsin in snake bite cases,[14] the potassium permanganate cannot effectively pursue the injected venom through the tissues.

Indications For Antivenom

Antivenom is recommended for all cases, except those involving only a puncture wound with moderate discomfort. Other exceptions, which may not need antivenom, are those cases reaching medical care (and antivenom) a number of hours after the sting, which are not severe stingings and which are clinically improving.

The initial dose of antivenom will depend upon how many spines have deeply punctured the skin, for example:

1 or 2 punctures, contents of one ampoule (2,000 units)
3 or 4 punctures, contents of two ampoules (4,000 units)
5 or 6 punctures, contents of three ampoules (6,000 units)

Each ampoule contains 2 ml of antivenom. The antivenom is usually given intramuscularly. It is becoming acceptable practice to administer anti-histamine, adrenaline and sometimes steroids prior to the injection or infusion of antivenom. Once the decision to give antivenom has been made, proceed as follows:

1. Give the patient an appropriate dose of anti-histamine parenterally.

2. Draw up in a syringe 1 ml of adrenaline (1 mg/ml). If the patient has a known allergy to equine protein or has had antivenom before, give a small dose of adrenaline subcutaneously (for example, 0.5 mg for an adult). It may be wise to give all other patients 0.25 mg of adrenaline subcutaneously as a prophylactic measure. A child should be given a smaller dose.

3. If the patient has a history of allergy, especially to equine protein, or has received equine protein before, a steroid (eg. 100 mg methylprednisolone) should be given intravenously.

4. Antivenom should only be given if full and tested facilities are available for treating an anaphylactic reaction.

5. Fifteen minutes after the parenteral administration of the antihistamine (with or without adrenaline at another site), give the antivenom intramuscularly.

In a very severe case, three or more ampoules may be required and the use of the intravenous route should be considered, particularly if the pain is widespread or the patient is shocked.

Antivenom should not be injected in or around the area of the sting. Such an injection may worsen the local effects of the venom.

Hospital Management

Bed rest is usually essential. The injured limb should be comfortably immobilized and the envenomed region lightly covered with sterile dressings. Like other penetrating marine stings, Stonefish stings are potentially contaminated with bacteria. The likelihood of tissue necrosis caused by the venom renders the injury more prone to clostridial infections than simpler wounds. Therefore, tetanus prophylaxis should be carried out in accordance with the victim's immune status, while .antibiotics may be used to control wound infection. The more severe injuries can require early surgical debridement of dead tissue and drainage. When antivenom has been delayed and considerable ulceration exists, then skin grafting may be necessary.

SEQUELAE

Sometimes a Stonefish sting remains painful for months and/or a recurrent inflammation or discharge occurs at the site of the sting. This is usually due to a foreign body in the wound, in particular a portion of a Stonefish spine. Being semi-transparent and quite deeply embedded, it is easy to overlook a broken-off spine and, if any doubt exists, it is preferable to explore the wound surgically during the initial admission to hospital.

Sometimes no foreign body is found. In one such case a middle-aged Melbourne man suffered a possible Stonefish sting to his skin on a remote island in New Caledonia in 1975. His envenomed leg became grossly swollen and for several days he was delirious and his companions thought he was dying. When he

reached civilization several weeks later, the skin was still swollen and discoloured. Over the next three years he suffered episodes of tender reddening of the area, associated with high fever sweating and confusion. These attacks occurred every two or three months and lasted several days. In 1978, he was diagnosed as suffering from recurrent haemolytic streptococcal cellulitis. He responded rapidly to a course of intravenous crystalline penicillin, followed by procain penicillin, and in 1981 he was still free of any recurrence. Because of his response to penicillin and negative x-ray, it was decided not to explore the region of the sting.

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The above is an edited extract from Australian Animal Toxins by Dr SK Sutherland, published by Oxford University Press, covering one of the topics discussed by Dr Sutherland as guest lecturer at the 1985 Annual Scientific Meeting of SPUMS.

Dr Sutherland has copies of Australian Animal Toxins available for $45.00, which is a large reduction on the bookshop price.

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THE ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT

Des Gorman

INTRODUCTION

The Hyperbaric Medicine Unit created within Royal Adelaide Hospital (RAH HMU) is a joint venture of the Institute of Medical and Veterinary Science, Royal Adelaide Hospital, and the Victorian Division of the National Safety Council of Australia. The RAH HMU is part of the Department of Anaesthesia and Intensive Care, answerable through the Senior Director of that department to the hospital Medical Superintendent and Chief Executive Officer. It is envisaged that the position of Senior Director will become a professorial appointment to Adelaide University in the immediate future.

ROLES AND FUNCTIONS

The RAH HMU has clinical and education roles, considerable research commitment and the task of co-ordinating the Diving Emergency Service. The Royal Adelaide Hospital was chosen as the ideal site for the HMU because the hospital is co-located with the Institute of Medical and Veterinary Science and Adelaide University, an excellent retrieval system was already based on the RAH Intensive Care Unit, and because the local abalone fishermen constitute a dysbaric problem of considerable magnitude.

The RAH HMU will provide emergency and non-emergency clinical services to both South Australia and the Northern Territory, and will encompass all aspects of hyperbaric medicine. Clinical services will be based on the Intensive Care Unit, involving Intensive Care Unit registrars and intensive care trained nurses also trained in hyperbaric medicine to act as recompression chamber attendants.

The static recompression chamber installed at RAH was built by DRAGER Pty Ltd, is compatible with NATO bayonet transportable recompression chambers, and