AUSTRALIAN PARALYSIS TICK

Just for completeness since we are talking about spiders and arachnids, mention should be made of the Australian Paralysis tick which is found in a wide area from Mallacoota to Cairns. The adult form is particularly dangerous because as it is engorging and burying itself temporarily in the skin of a human it may release a very potent neurotoxin. It is an unusual neurotoxin because it works extremely slowly. One can inject it into a dog and nothing will happen for 18 hours and then the dog will start getting paralysed. This toxin acts presynaptically and reverses quite slowly. First-aid is to gently remove the intact tick. There is an antitoxin but it is one type of paralysis which recovers, in most cases, with just standard intensive care. Once the tick is removed, the victim generally improves quite rapidly.

THE BOX JELLY FISH STING

John Williamson

By any accounts *Chironex Fleckeri* is a significant animal and the number of fatalities that it has produced will support that statement. The Northern Australian and Western Indo-Pacific box jellyfish has now been responsible for 68 documented human deaths in this country, 70% of whom were women and children, due to their generally smaller body mass and hairless skin. The whole crux of the problem of the box jelly fish is related to the speed with which this animal can produce envenomation in human beings and of course in its natural prey. It has already been pointed out today by Dr Sutherland in his opening address that animals including this one have no interest whatever in human beings. All envenomations by this animal are due to accidental encounters and it is the human's fault. The animal does not attack. It is pertinent to say that none of these fatalities have received any effective form of resuscitation to date.

When a snake envenomates, or a spider or a blue-ringed octopus or a cone shell, the venom is all deposited in one place so there is a limited surface area, although obviously an effective one, between the venom parcels and the blood bearing tissues and the lymph bearing tissues where the absorption occurs. However, with the box jelly fish, the venom is divided into many thousands of millions of tiny parcels spread over the architecture of the tentacles. The nematocysts occur on transverse bar-like patterns on the tentacles. Consequently, when envenomation occurs the venom bearing nematocysts or microbasic mastigophores, as our colleague Bob Hartwick likes to call them, discharge their venom in multiple million tiny doses and inject them into the victim in many different sites at the same time. The trajectory of most of these nematocysts is something just under a millimetre which will carry the venom into the subepidermal, richly vascularised tissues. This arrangement of envenomation offers an enormous area for absorption so the speed at which high blood levels of toxin are achieved following a serious envenomation by one of these animals is extremely rapid. It is measured in minutes.

Small victims die very rapidly on the beach. A large number of the fatalities and the non-fatal stings involve the Aboriginal population in this country.

In a survivor of a serious sting, there is a much more vigorous inflammatory response. One can see the actual cross hatched or ladder pattern which is diagnostic of the sting of this jelly fish. The only other jelly fish that may produce a pattern like this, and it will not be as dramatic, is the fire jelly.

Three to ten per cent acetic acid in water (vinegar), as far as our studies have shown, renders the nematocysts of the box jelly fish irreversibly inactive within a period of about 30 seconds. Nothing we have been able to do can provoke them to fire following exposure to that concentration of acetic acid. Methylated spirits causes a massive discharge of nematocysts from the tentacle of the box jelly fish. If that tentacle happens to be applied to a human victim, then there will be increased envenomation. When the tentacle has been treated with vinegar (4-6% acetic acid), it does not matter whether it is brown vinegar or white vinegar, and then methylated spirits is applied there is no response. Time prevents me from discussing the role of vinegar in the treatment of other jelly fish stings.

I must emphasize that it is important to understand that vinegar does nothing for the pain. It inactivates the unfired nematocysts but it has no effective role to play whatever in the treatment of the pain of the sting of the venom that has already been injected. Vinegar does work for other jelly fish but certainly not for all. Vinegar certainly renders the nematocysts quite harmless and useless.

We advocate, purely by extrapolation and without any experimental proof at this stage, the use of compressive immobilisation. We would advocate in any serious sting, and certainly any sting where resuscitation becomes necessary, the immediate application of vinegar followed by the application of a compressive immobilisation bandage over as much of a sting area as possible. Remember compressive bandages are not tourniquets. This treatment appears reasonable to us at this stage while we await experimental confirmation that this will trap the venom in the skin. However, remember this has to be done immediately, because the speed of absorption of this venom is such that unless it is done immediately it is unlikely to have any greatly beneficial effect.

Critics of this approach, in the absence of experimental verifications could well say that one is not doing the best for the patient because this will trap the venom in the skin and will produce increased pain. The answer is, I feel, that priorities are important. If the victim is unconscious increased pain will not matter. If he or she has been seriously stung, it is better to have severe pain than to run the risk of losing ones life. The dermato-necrotic, or skin killing, effect will have, theoretically at least, been made worse by this action. There is no doubt about the powerful skin killing effects of the venom. However where life is threatened, this may be the lesser of two evils. The circumstantial evidence that we now have, which is extensive and strong, shows that the administration of antivenom will reduce both the pain and the skin killing effects. Obviously resuscitation takes absolute priority. Our recommended emergency treatment on the beach now consists of vinegar dousing, "pressure-immobilisation" of the sting area, and application of ice-water through the compressive bandages for partial pain relief in the conscious

victim and the early intravenous or intramuscular administration of specific antivenom (concentrated Chiron*ex Fleckeri* venom-specific immunoglobulins, isolated from the serum of hyperimmunised sheep) with appropriate precautions. That is the sequence of events that we advocate, particularly when we are teaching the surf life savers who patrol beaches in the risk area and other members of the public.

When talking to a group of intensivists, this problem is a little academic because everything happens so quickly that it all happens on the beach and by the time the victim, if he is alive, gets to the intensive care unit, it is largely all over bar the shouting. Unless the intensivist happens to be on the beach or in the ambulance he is unlikely to see any of the actual shooting. That is not to say that a knowledge of this is not important for intensivists. When the patients reach intensive care follow up treatment makes a big difference. But nothing takes the place of immediate and effective treatment on the spot. Before I leave this subject of resuscitation, let me re-emphasize what has already been emphasized today, that when a person is being resuscitated from a massive envenomation, when resuscitation is effective absorption of venom will occur again and unless it is trapped in the skin or neutralised by antivenom one can expect a further collapse from systemic effects. So one has to keep on working and one does not leave the patient.

The characterisation of the venom is incomplete at this stage. It has two broad molecular weight groups. The study of the lethal factor which includes the so-called cardiotoxic factor has been done on guinea pigs and rabbits, mice, rats and toads. It worries me a little bit to extrapolate of some of these conclusions to the live human clinical situation on the beach.

There are a number of interesting new developments in this area. For example, recently in the Medical Journal of Australia some workers from Maryland published encouraging results in mice where verapramil reversed the arrhythmic potential of *Chironex Fleckeri* venom. They suggested that this may be an approach to the first-aid situation. We have only two well documented severe envenomations that have been survived. They both received effective resuscitation on the beach. But in both those victims, one a child and the other a pregnant adult female, only expired air resuscitation was necessary. There is no doubt that they received a potentially lethal dose of venom. The question I ask is whether the danger to the adult human or the child human is a cardiotoxic one from this venom or is it something that is acting centrally neurologically which produces respiratory arrest. I am sure that the venom does not do the myocardium any good at all, but it may not be the critical factor. It would appear on the evidence so far that expired air resuscitation alone, if effective and sustained, will be all that is required in the absence of some other complicating factor. The haemolytic component appears to be clinically unimportant. Why does apnoea occur when the venom is extremely thermolabile? When it is raised from the temperature of the sea to the temperature of the human body its longevity may not be very great. Effective resuscitation for those two cases was only really necessary for a period of about 30 minutes to three quarters of an hour before spontaneous ventilation recurred. This fact has important implications in teaching first aid and in the approach to the problem.

The administration of the antivenom is quite another problem. There is no question that it is effective, at least on the basis of the cases that we have recorded so far. Vic Callanan, Max McDonald and I, among others, are teaching the life savers in the risk part of the world, to give antivenom intramuscularly to people who obviously require it. It is not difficult to decide when a case does require antivenom. The antivenom is a concentrated mixture of immunoglobulins from hyperimmunised sheep so it carries the risk of serum reaction and all appropriate precautions should be taken which have been discussed this morning. When the choice is between antihistamines, adrenalin and steroids it is not hard to understand why we have recommended that the life savers give steroids and not one of the others on the beach. The whole subject of box jelly fish envenomation is heavily influenced by the need to be practical. It is no good telling life savers to give adrenalin and it is not much better to ask them to give antihistamines to someone whose conscious state may already be impaired. So it seems to us that steroids and antivenom is the best choice on the beach until the patient gets to an area where more expert medical treatment is available. The antivenom is supplied in ampoules, from the CSL. We recommend three ampoules intramuscularly on the beach assuming that the people there cannot be expected to give an intravenous injection. If intravenous access can be obtained that is excellent. Obviously that is the route of choice but it is most unlikely to occur on the beach. If you have ever tried to do a venipuncture on a wet, sand covered limb in a shocked or struggling patient, you will know that it is just about impossible. As for one in ten dilution on the beach, that is impossible too. Intravenous antivenom is certainly desirable in the casualty or the intensive care unit.

A lot of claims have been made about pain relief. We have looked at a lot of substances applied to box jellyfish stings and *physalia* (blue-bottle) stings. None of them seem to do anything whatever for pain relief, or for anything else such as nematocyst inhibition. The only substance which offered any sort of pain relief was Skefron which is a volatile complicated hydrocarbon which appeared to achieve its effect by simple cooling. It seems to us that from a practical point of view on the beach in the risk period in the Northern part of Australia and for that matter, elsewhere in the world where the risk occurs, if the person is conscious and in a lot of pain one might try reducing the pain by applying ice-water or ice directly over the sting area through the bandages. Ice is likely to be available on the beach in summer at a barbecue. Certainly they will not have a complicated hydrocarbon handy and Skefron costs a certain amount of money. It is a very small can and I have no idea of the potential toxic effects of that agent. The message is, at our present meagre level of knowledge, to be extremely optimistic and extremely aggressive and teach the lay public, because they are the people who are going to be on the spot when this problem occurs, the proper first aid and the need for protective clothing.

There are three jelly fish stings and one other toxic marine animal sting that have either been published or come to my notice, which resulted in detectable antibodies and produced a subsequent reaction some weeks later in the absence of

further tentacle contact. One is the sea nettle which in America is a Chr*ystosora* animal, the closest relative we have in Australia is *Pelagia* or the mauve stinger. Another is their Man O' War which is very similar to, if not identical with, our Portuguese Man O'War or blue-bottle *(physalia)*. Published work shows that antibodies, particularly IGG but also IGE antibodies, have been measured in people stung by these animals and the antibody titre is correlated with the severity of the sting and of the symptoms. The other two animals are the box jelly fish, and a toxic sponge which came into contact with the hand of a diving Adelaide surgeon. These two are cases I have seen or been consulted about. The antibodies of at least the first two appear immediately. They may persist for years. In the case of the girl who was stung by a box jellyfish at Yeppoon about 16 days later the whole thing blew up again. It was clear that this was not infection. In fact this secondary reaction caused more problems for her than did the primary sting. She did have a history of allergy. When I was contacted on the phone I advised that she be treated with steroids, as I had done with the chap in Adelaide, and she got better.

It looks very much as if allergic reactions in jelly fish envenomation may be important. This applies to the immediate reaction as well as to delayed reactions. Elevated specific immunoglobulins, particularly IGG and IGE, have been demonstrated particularly with the sea nettle and *physalia,* and these can persist for years. Recurrence of clinical cutaneous reaction to jellyfish stings may occur within a few weeks without additional contact with the tentacles. As far as sea nettle and blue-bottle are concerned serelogical cross reactivity occurs.

SEA SNAKE ENVENOMATION

Hilary Mercer

My presentation concerns a case of a sea snake bite which is apparently the first case which has been reported in the Australian literature, although there have been many cases reported from Malaysian waters.

A couple of years ago a two year old child was paddling on Lamamoor beach which is quite a picturesque spot between Emu Park and Yeppoon. She started screaming and the mother ran down and saw a rather loathsome creature attached to the child's ankle. As she approached the creature swam away towards two teenage boys who killed it and brought it along for identification. The mother had great presence of mind and grabbed her daughter around the calf with both her hands and did not let go. The pair of them were taken to the Yeppoon Ambulance station where the wound was washed and inspected by the ambulancemen. No tourniquet or compression bandage was applied.

The mother removed her hands. Up to that point the child was speaking coherently and quite bright. But within 30 seconds of the mother taking off her hands, the child became very weak, developed ptosis and some respiratory distress. They were rushed to the Yeppoon Hospital, which was close by, where about 20 minutes after envenomation the child became cyanosed and needed intubation. From there they went to Rockhampton Hospital. They arrived there about an hour after envenomation. By this time the child tolerated reintubation without any resistance whatsoever.

Then about one and a half hours after envenomation, we gave the first dose of sea-snake antivenom. By this time the snake had been brought along and identified by one of our local herpetologists, and this was later confirmed by the Queensland Museum, as being *Astocius Stoksii*. Incidentally, no antihistamine was given because of a previous reaction to promethazine and for some obscure reason adrenalin was not given either.

Over the next two hours there was no real improvement and we gave two further ampoules of antivenom. After the third ampoule there was some apparent clinical improvement. The child opened her eyes and started looking about. However over the next 10 hours or so the child seemed to regress and 14 hours after envenomation the child had odd clonic movements and we thought the conscious state was deteriorating again. We treated her with phenytoin and gave a fourth ampoule of antivenom.

I then spoke to Struan Sutherland on the phone and he suggested that we were probably not giving enough antivenom. So we gave another three ampoules. The only thing that stopped us giving more was that the child developed a rash which responded rapidly to antihistamines. After those further three ampoules the child became a lot better. About 22 hours after the bite we were able to extubate her. Two hours after that she was sitting up and attempting to speak.

The following day she was sent to the children's ward. Over the next few days she had very odd movements of her limbs and hallucinated but she was able to be discharged six days after the envenomation. Subsequently there were no real problems. However she must have had sadistic brothers because they kept creeping up to her with bits of grass and things and saying "Ah, the snake's got you!" and she would go all 'funny'.

There was nothing dramatic about the investigations. The coagulation status was normal, muscle enzymes were up, but cardiac enzymes were normal. The white blood cell count was raised, as expected, to about 27,000. Renal function tests were quite normal. Myoglobinuria was only found on one occasion about 48 hours after the envenomation.

The snake itself was about one and a half metres long. It was an *Astocius Stoksii* which is a snake that is not seen very often around here. It inhabits the waters of the Indo-Malayan coast more than here. We see many sea-snakes in central Queensland. They are regarded as potentially dangerous by divers and fishermen but we do not see many bites. They seem to be timid creatures. However when they are mating they conglomerate in large number and may come towards people which is very unnerving apparently.

The way this child's foot was mauled may have been responsible for the massive envenomation in this case. This snake is the largest of the sea snakes. It has the largest mouth with the largest fangs, as far as I know, of the sea snakes. Its fangs can penetrate wet suits.