5.6 Australian jellyfish – epidemiology of envenomation

see Map 1 – The Indo-Pacific and Australia, showing human deaths and chirodropid distribution.

5.6.1 Chironex fleckeri

Chironex are often referred to in Australia as Stingers or the northern Australian box jellyfish. Through the Surf Life Saving Association, and though his contact with many organisations, the author is trying to get the general public to realise that there are many varieties of "box-jellyfish" with symptoms varying from mild skin pain (*Carybdea sivickisi*) to death from respiratory and/or cardiac failure. Thus, it is important to be specific and the name *Chironex* box-jellyfish is being suggested.

Distribution

Australia

Until recently, this genus and species has been thought to be present in Australian waters only. Stings to humans extend from south of Broome in Western Australia (east Indian Ocean, latitude 18^{o} south) (Marsh *et al* 1986) around the northern Australian coastline down to Gladstone, Queensland, on the eastern Australian coastline (west Pacific Ocean, latitude 24^{o} south) (Lumley *et al* 1988). *Chironex* specimens have been reported caught or sighted in the Exmouth Gulf in Western Australia (latitude 22^{o} south) (L Marsh, 1988, personal communication) to Bustard Bay, south of Gladstone, Queensland, in the east (latitude 25^{o} south) (G Mackenzie, 1991, personal communication).

World

Until recently the distribution of *Chironex* was stated to be Australia only (Southcott 1956; Barnes 1960, 1966; Hartwick 1987;1990). The author believes this distribution can now be widened to include nearby areas of the Indo-West-Pacific Ocean (see Map 1): -

In 1990 the author examined and identified 6 chirodropid specimens in the Smithsonian Institute, caught by Mayer in the Philippines (1910). Although Mayer had identified them all as *Chiropsalmus quadrigatus*, in the opinion of the author, who has studied many hundreds of specimens, 2 were actually *Chironex fleckeri*, 2 fitted the somewhat "vague" description of *Chiropsalmus quadrigatus* as described by Haeckel (1880), and the other two were too badly damaged, or preserved, for the author to be able to suggest an identification. These observations agree with

those of Barnes who stated to the Royal Society in Cairns in 1964 (Kinsey 1986) that he had identified specimens of *Chironex* in the Smithsonian that had been caught by Mayer (1910). The author feels that this is the reason that the description of *Chiropsalmus quadrigatus* in Mayer (1910) is so confusing – he seems to be actually describing a combination of the features of *Chiropsalmus quadrigatus* and *Chironex fleckeri*, even though the latter was only identified as a news genus and species many years later (Southcott 1956).

In 1993 the author also identified as *Chironex fleckeri,* the specimens sent by Major T Hooper from Borneo to Phil Alderslade, even though it had never been identified in this area before. These were two of several specimens caught very close to where Major Hooper's son had sustained an almost fatal jellyfish sting several months previously (see Case History below)(Fenner & Williamson 1996).

Also in 1993 the author examined a specimen in the South Australian Museum that came from Vietnam, which he also would identify as *Chironex*.

These specimens were also reviewed by Rifkin, who also agrees on this identification and that the distribution of *Chironex* is much wider than previously believed (Rifkin, in Williamson *et al* 1996, p 259).

Habitat

The work of Hartwick (1987, 1990) and Barnes (1960, 1966) show *Chironex* to be a coastal jellyfish. When the air is hot and still (frequently with a gentle northerly wind, common in tropical Australia in the summer months), they will come into very shallow waters at gently-sloping sandy beaches close to creek and river outlets where mangroves are common (Hartwick 1987;1990). Hartwick developed the life-cycle for *Chironex* after first breeding specimens in the laboratory (Yamaguchi & Hartwick 1980), and then discovering their polyps in a natural habitat under rocks and mangrove roots, up to 5 kilometres from the sea in mangrove-lined rivers and streams (Hartwick 1987).

For these reasons *Chironex* are often prevalent around the mouth of these creeks, especially as these are also areas where their prey of prawns and fish occur. Similarly they like the gently sloping, sandy beaches near these creeks. Here they can swim with the tentacles trailing behind them with little fear of them `snagging' on rocks or other obstacles fishing for their prey, including the prawns and small fish that inhabit these areas. Unfortunately, these are the same areas that humans like and

frequent. *Chironex fleckeri* have been seen and caught over rocks, but this is less common (Fenner *et al* 1995).

Although *Chironex* are common in very shallow, calm water they have also been reported out to sea in rougher waters. A surf life saver was stung in deep, rough water near Cairns in 1991 when a *Chironex* was washed on to the deck of the victim's surf ski while he was paddling some 200--300 m offshore (Fenner *et al* 1995). Mulcahy (1999, personal communication) also reports that they have been trawled off the bottom by trawlers a couple of kilometres offshore. This observation remains unconfirmed.

Season

Chirodropid fatalities throughout the Indo-Pacific have been documented in every month of the year (Cleland & Southcott 1965, p.114-116); some of these may be from *Chironex* (see above). In the Northern Territory, reports of *Chironex* stings have occurred in every month of the year except July, but fatal stings have only been registered from September to May (Fenner *et al* 1995)(see also, database results below).

Appearance and behaviour

Although the bell may grow to 30cm, previously specimens of this size have only been caught in far north Queensland, however, on 4 April 1997 a specimen 25cm diameter was washed up on a beach near Mackay (Figure 7). At the same time, up to 50-60 other specimens were also washed up on to local Mackay beaches. This finding is extremely unusual and cannot be explained. *Chironex* are very strong swimmers and rarely beached, with just the odd report of its occurrence (Fenner *et al* 1995). Specimens of sea water have been taken and are to be analysed for pollution and other laboratory tests to see if any explanation can be given.

The maximum size of specimens reported from the Northern Territory is 14cm across the bell (Alderslade, in Williamson *et al* 1996, p.266); the reason for this is unknown.

The bell is usually transparent, and often very difficult to see, even in very shallow water. It has a box-shape with four corners (pedalia), from which up to 15 thick, flat tentacles may arise. These tentacles may contract to a few cms or extend up to 3 meters or more in length – thus giving a theoretical tentacle length of up to 180 meters on a single large specimen. Hartwick once estimated that up to 4000 million nematocysts may be present in the mature adult (Williamson 1985a). Assuming there may be 180 meters (maximum) of tentacle length, this acquaints to

approximately 22 million per meter extended tentacle. Death has occurred from just 1.2 meters of tentacle length (B Currie, 1996, personal communication) – in which case roughly 25 million nematocysts would have caused death, although the approximate number actually penetrating the skin, envenoming and causing death is not yet known.

Chironex fleckeri are the most evolved species of jellyfish having 4 rhopalia, or sense organs that lie mid-way between the pedalia, and at the base of the bell. These rhopalia are organs that: -

- differentiate light and dark using a rudimentary lens
- detect vibration – such as impending danger such as rough water from storms or predators (including humans)
- maintain balance and orientate the jellyfish position in the water.

They feed on small fish and prawns that are quickly killed when they swim into the tentacles. The reason *Chironex* venom has such tremendous “killing power” is not, as many people believe, to attack and kill humans, but to be able to kill its prey quickly, before the prey struggles too much, breaking off tentacles. Jellyfish, like humans, need to eat to live and breed. If *Chironex* lose their tentacles then they lose `killing power', they cannot feed and they die.

Their tentacles are `elasticised' and may extend out many meters when they are gently swimming along `fishing' for prey. They can be contracted up to only a few centimetres when they are actively swimming. *Chironex* are active swimmers, unlike many jellyfish, swimming by a muscular contraction of the bell, which shoots out a jet of water propelling the jellyfish forwards. They can swim at the pace of a walking man, although the energy expended is too great to maintain this for any great length of time (R Hartwick, 1988, personal communication).

Chironex can recognise objects in its path and avoid them. Such obstacles may be objects in the water (ie. rocks and humans), and will often retract their tentacles and swim away from this perceived obstruction or danger.

Through media statements, many people believe that jellyfish `attack’ humans; it is actually the humans `attacking’ the jellyfish, usually by running into the water too quickly, not allowing the innocent jellyfish to be able to retract its tentacles and swim away. Tentacles that are torn off the jellyfish by the victim struggling to get away

mean that the jellyfish loses its `killing power' and consequently, as it cannot catch its prey, it dies.

Clinical effects

The tentacles cause instant severe skin pain like being branded by a red-hot iron. Tentacle material is usually avulsed from the animal by the struggling victim, and cling firmly to the victim's skin which is often diagnostic in chirodropid envenomations and fatalities. These remaining tentacles will contain many thousands of unfired nematocysts. The longer these tentacles remain in contact with the skin, the greater the risk of increasing the envenomation – thus making vinegar such an important immediate, first-aid treatment (see 5.8.3).

Victims often scream with the pain and children will often stand in the shallow water where they have been stung, pulling at the tentacles, and consequently receiving more stings to their upper limbs. As the keratin of the palms is often too thick for the nematocyst thread tube to penetrate, stings marks will often be confined to the backs of the hands only. Conversely, adults will usually jump back out of the water and run for help, the increased muscular effort increasing heart rate and consequent venom absorption. Thus the first treatment of *Chironex* stinging may be to `retrieve and restrain', to prevent further envenomation (First aid and medical treatments are considered below in 5.8.3 and 5.8.4).

Severe, whip-like marks are visible on the skin with an intense acute inflammatory response developing rapidly. Usually, a `frosted-ladder' pattern matching the appearance of the nematocyst `batteries' on the tentacle will be visible, and is of diagnostic value (Barnes 1960; Williamson *et al* 1980; Williamson *et al* 1996). This appearance is due to millions of discharged and undischarged nematocysts remaining on the skin of the victim, in the same battery lines as are present on the tentacle.

Linear erythematous wheals then rapidly develop, often with a white ischaemic centre. Serious envenomations develop surrounding oedema and darkening of the skin with vesiculation and partial or full thickness skin death, usually resulting in permanent scarring.

Often confusing, victims who die rapidly do not have sufficient time to develop this severe inflammatory response and their skin marks may appear quite insignificant.

Treatment discussion

(See treatment below)

Compression bandaging as suggested by Sutherland *et al* (1979) for snake bite is suggested (see First aid 5.8.3). Although it has not been scientifically proven to be effective in the acute onset of *Chironex* envenomation, using the principle of holding any remaining venom in the skin (see also, intra-vascular venom injection, 3.3.3), compression bandaging is recommended for all major *Chironex* stings.

A major sting is defined as one causing: -

- An impaired conscious state, or unconsciousness
- Shallow, slow breathing with central cyanosis, or respiratory arrest
- Reduced pulse strength, irregularities, or asystole
- Covering the equivalent of more than 50% of one limb
- unrelieved pain

There are many other factors that are used to define a major sting, but they will always result in one, or all, of the above signs. An important point stressed to all surf life savers and others treating sting victims is that it is important to realise the significance of a sting victim who was loudly distraught and physically active who suddenly becomes quiet - usually indicating the onset of impaired cerebral perfusion, with a reduced conscious level and possible cardio-respiratory arrest.

Case History 1

Case history written by Fenner (Williamson *et al* 1984)

At 1000 on 10 March 1984 a healthy 5-year-old girl weighing 2Okg was wading waist-deep in the sea at a beach near Mackay. She received a box jellyfish sting, mainly to her legs, which measured a total of about eight metres of tentacle contact. She was immediately pulled screaming from the water by her grandmother who restrained her hands which had already sustained some additional stings. The victim's mother doused the adherent tentacles with vinegar within three minutes. The girl who was in great pain, but fully conscious and not cyanosed, was brought to a local GP surgery within 15 minutes of the envenomation. She immediately received an intramuscular injection of pethidine (40 mg), and an intravenous injection of 20 000 units (one ampoule) of box-jellyfish antivenom, diluted 1:2 with water. Within seconds, the pain, and the size and 'anger' of the wheals on her limbs, had diminished. On admission to hospital 25 minutes later she was again in some pain, and had obvious tentacle marks. Her vital signs were normal. No compressive bandages were used at any stage.

In hospital critical care unit, she received IV hydrocortisone (30 mg at once, then every four hours) by the intravenous route; IM pethidine (20 mg) and vinegar 'soaks' to the sting area.' Within six hours, and while breathing inspired oxygen, she developed central cyanosis with tachypnoea, but no audible moist lung sounds.

Eight hours after the envenomation, she received further IV injections of hydrocortisone (100 mg) and of box-jellyfish antivenom (20 000 units), with no improvement in her condition. She had oliguria and her chest x-ray showed interstitial pulmonary oedema. The oxygen was increased but central cyanosis persisted. Over the next three hours the patient began to pass urine through the indwelling catheter, and a rapid improvement in her clinical condition was noted. Twelve hours after envenomation she was no longer cyanosed, had passed 800 ml of urine, and no analgesia was required. The next morning, while breathing room air, bilateral bronchial breathing was heard on auscultation, and the sting areas were swollen and tender. All other signs and symptoms had resolved. and her condition was obviously much improved. She was discharged from hospital well on the fifth day after admission.

Unfortunately, secondary infection with *Staph. aureus* developed in her right popliteal fossa over the next two weeks treated by the author with co-trimoxazole, 7.5 ml twice a day and topical applications of an antibiotic preparation [Neosporin) and hydrocortisone [1%1 cream) with a seemingly good result. However, scarring developed which remains obvious 12 years later.

Case history 2

Case history written by Williamson and Fenner (Lumley *et al* 1988)

On January 20, 1987, a healthy five-year-old boy who weighed 20 kg was bathing waist-deep in the sea at Barney Point beach near Gladstone, central Queensland. The weather was hot and still, the sea calm, and the water temperature was 29.5 'C. At about 1000 he ran from the water, shrieking and attempting to pull at adherent, translucent tentacles on his legs. His grandmother, who had accompanied him to the beach, assisted in this process with a towel, and in so doing sustained minor stings to her own forearms.

The child's screams attracted attention. This person came to the patient's assistance and doused vinegar over the sting area on the child's legs, which were already showing red wheals. The estimated elapsed time between the initial envenomation and this first-aid manoeuvre was four to five minutes.

The grandmother ran for her car, and the child, who was described as "pale and sobbing", was placed, sitting up in the back scat, with the helper. The elapsed post-envenomation time now was approximately 10 min. The grandmother drove to the ambulance centre in less than three minutes by ignoring all traffic signals. The condition of the child, who still sat upright, was "gradually deteriorating". Just before reaching the ambulance centre the

householder observed that the child had ' 'blue lips" even though "respiratory sounds" were still apparent.

On arrival at the ambulance centre at 10. 15 a.m., the patient was described by the ambulanceman as unconscious, with a ghostlike pallor ' pulseless and with "fixed, dilated pupils". The child was transferred to an ambulance vehicle where single-operator cardiopulmonary resuscitation with oxygen was commenced. The victim was then rushed to the casualty department of the hospital, where it arrived at 1018.

An initial assessment showed an unconscious apnoeic child with peripheral and central cyanosis, no palpable pulse, and urticarial wheals on his legs, hands and lower abdomen. The child was intubated, IV access obtained and 100% oxygen and ECC given. An ECG showed pulseless idioventricular rhythm which failed to respond to any therapy. After 20 min of continuous resuscitation, and approximately 40 min after the initial sting, further resuscitation was abandoned.

Post-mortem examination and investigations
Conducted 25hrs after death a post-mortem showed urticarial lesions with tentacle imprints on both wrists, legs, thighs, and the lower abdomen, width 4-5mm and total length 4 m. Skin biopsy showed many nematocysts with a concentration from zero to clusters of more than 4000/mm^2, presumably corresponding to the "batteries" of nematocysts that are grouped on the epithelium; most had discharged, and their threads had penetrated the mid-dermis, with trajectories that ranged from 0. 1 mm to 0.7 mm. A cellular inflammatory reaction was not present, which reflected the lack of time for such a response to occur

5.6.1.1 *Chironex* treatments:

See section 5.8.1 for awareness, prevention and treatment of *Chironex* stings, sections 5.8.4 and 5.8.4 for *Chironex* first aid and medical treatments and section 5.6.1 for data on *Chironex* stings.

5.6.2 Carukia barnesi -the Irukandji

Ecology

The `Irukandji' is a small `box' jellyfish that occurs in tropical Australian waters. Information from the database (below) suggests that the occurrence is greater than previously believed (Williamson 1985a, p 15). Stings have now been reported from waters south of Gladstone in central Queensland, 200 kilometres south of the Tropic

of Capricorn, northwards around the north Australian coast and as far south as Broome in Western Australia (Fenner, in Williamson *et al* 1996, p 247).

Although it is a deep-water jellyfish, swarms may often be brought to the surface at swimming beaches in these areas, where multiple stings may occur in summer months.

Appearance

The bell is transparent and only 2.5-3.5cm in diameter, making it almost impossible to see in the water. There are just 4 tentacles, one in each corner. When contracted these tentacles may be just 5-7cm but they may extend to some 60-70cm when the Irukandji is `fishing' for its prey of small fish (Southcott 1967)

Initial envenomation

The mark on the skin is usually the imprint of the jellyfish bell, making it very difficult, if not almost impossible to see; less often, tentacle marks may be seen (Fenner *et al* 1988). A couple of moments after the usual sting from the bell, the skin may develop a mild redness and a `goose-pimple' effect which may last from 30 minutes or so; localised sweating sometimes occurs. In some cases this initial mild skin mark may be totally missed. The reddish imprint, if visible, may last several days.

After the initial sting there is a characteristic time delay before the onset of the severe systemic symptoms which comprise the Irukandji syndrome (Flecker 1952b). The delay varies between 5 and 50 minutes, but is characteristically 30 minutes after the initial envenomation.

Irukandji syndrome

After this initial time delay a bizarre set of distressing systemic symptoms occurs Flecker 1952b; Barnes 1964).

The syndrome now has three recognised clinical sequelae - in some cases the third is also present (Fenner *et al* 1986b, 1988; Carney & Fenner 1997): -

1. Pain
2. Catecholamine effects
3. Global cardiotoxicity leading to pulmonary oedema.

A victim may have any combination of the following signs and symptoms, but always has the severe muscle cramps:

Pain

- Low back pain - a continuous severe `boring' pain in the sacral area. The victim has trouble walking – the first diagnostic sign to the author when he was on patrol one day when a victim hobbled towards him, holding his back! (Fenner *et al* 1986b)
- Muscle pain or `cramps' – these move rapidly into all 4 limbs and the abdominal and chest wall muscles. The pain is described as severe, unbearable and coming on in `waves' (similar to labour pains) - although never fading completely (Fenner *et al* 1986b).
- Chest pain or `tightness' – this is a difficult diagnostic dilemma. It was firstly suggested that it was due to muscle cramps in the intercostal muscles, although cardiac muscle pain could not be excluded (Fenner *et al* 1988). However, recent work by Carney and Fenner (1997) suggests that there may be significant cardiac pain as some victims get a significant rise in their creatinine kinase (CK) and CK-MB. There has been no bronchospasm demonstrated to date.

Catecholamine excess

Many of the signs and symptoms associated with the Irukandji syndrome resemble those of an adrenal medullary tumour (phaeochromocytoma), or a Funnel-web spider or scorpion envenomation, with excessive release of catecholamines into the bloodstream (Fenner *et al* 1988). As the syndrome starts to take effect, usually some 30 minutes after the initial mild envenomation, victims usually experience many, if not all, of the following symptoms: -

- Sweating – this may be localised to the actual stung area, or even be localised in a totally unrelated part of the body. In severe envenomation the sweating is usually generalised, profuse and drenching;
- Pilo-erection – similar to the sweating, piloerection may be localised at the original site of envenomation, or generalised. Again, it may occur in an area totally unrelated to the site of envenomation – no explanation can be given for this although it has been observed in a number of cases treated by the author;
- Anxiety and `wretchedness' - The victim is over-anxious. They feel “absolutely dreadful” and often have “a feeling of impending doom” (a thought that is often shared by the treating first aider!);

- Restlessness - Victims are restless and move continuously, trying unsuccessfully to get comfortable. This is characteristic of the syndrome, but is also probably aggravated by the severe muscle pains;
- Headache – This may be severe and incapacitating; it may be a frontal headache, or global; it does not occur in every case;
- Nausea and vomiting – the victim is usually nauseated and intractable vomiting may occur in some cases;
- Increased respiratory rate, often with a `sighing respiration';
- Tremor - A fine tremor usually occurs, or fasciculation of the small muscles of the limbs. In some cases this may be severe;
- Pallor, or peripheral cyanosis - peripheral vasoconstriction usually occurs, which may be intense in some cases;
- Oliguria – this may be due to the fluid loss from the sweating and/or vomiting, and may be aggravated by reduced renal perfusion. No literature has been published on this but it is usually an incidental finding whilst treating severe cases;
- Tachycardia – This occurs invariably. Cases that are monitored early (eg helicopter evacuations) often show a tachycardia which may be irregular with ventricular extra-systoles, however, this has only been observed on rare occasions in hospital cases and may just occur in the early stages of envenomation: -

  Further work is currently being arranged in this area by the author. It is hoped that lifeguards may be supplied with monitors to take rhythm strips as soon as possible after envenomation, before the Ambulance reaches the victim (which often takes up to 30 minutes because of the remoteness of most envenomations;
- Hypertension - The blood pressure may reach levels as high as 280/150mm Hg in some cases in previously-normotensive victims. To date this has not caused any documented cerebrovascular accidents. It was believed to be a contributing factor to the heart failure (Fenner *et al* 1988), but recent work by Carney & Fenner (1997) suggests the heart failure may be caused by direct myocardial toxicity (see below);

Later complications: cardiopulmonary decompensation

Sudden breathlessness may develop in victims with the Irukandji syndrome, usually some 15-18 hours post-envenomation (occasionally less). This has proved to be acute pulmonary oedema (Fenner *et al* 1988; Carney & Fenner 1997).

Fenner *et al* (1988) demonstrated a (toxic) global cardiac dilation, but could not exclude a pulmonary membranous contribution, whereas Martin & Audley (1990) felt the oedema may be due to massive alpha-adrenergic stimulation. Recently a number of severe cases were treated where echocardiography showed marked global dilatation and left ventricular dysfunction (see Case History 1 below)(Carney & Fenner 1997). Further studies using a Swan-Ganz catheter to measure right (and left atrial), pulmonary artery and pulmonary wedge pressures, together with comparison of the pulmonary oedema fluid and plasma were suggested (Fenner *et al* 1988) and recently confirmed as essential for further study on the cause and effect of the global dilatation and pulmonary oedema (Carney & Fenner 1997).

Case History 1 (from Carney & Fenner 1997)

A 19-year-old previously-well woman was stung on the left arm by a jellyfish while swimming in tropical waters near Mackay on the 26/03/96. Within 15 minutes she developed severe back pain and muscle pains needing pethidine 50mg IV and 50 mg IM of pethidine during helicopter evacuation. She was breathless and was given oxygen 8l/min by face mask; her SaO_2 during transfer was 96 % on oxygen.

On arrival in a local hospital 15 mg of intravenous phentolamine was infused for mild hypertension (140/110). She was very agitated and was given 5 mg of intravenous midazolam and 10 mg of intravenous diazepam, and a pethidine infusion was commenced at 30 mg/hour for severe muscle pains.

Later that night she woke with cough and a wheeze; shortly after she started to cough up pink, frothy sputum. She was tachycardic (heart rate 130bpm) and breathless. Her SaO_2 was 75 % on room air, which was increased to 85 % on 2 litres of oxygen/minute via the nasal prongs. She had bilateral crepitations and a chest x-ray showed bilateral hazy infiltrates suggestive of pulmonary oedema. Nebulised salbutamol was unhelpful and two 40 mg IV boluses of frusemide were somewhat ineffective. She was commenced on 15 litres/minute of oxygen via a non-rebreathing mask which kept her SaO_2 at 85 % overnight. Her blood pressure remained about 140/90.

The next morning she was transferred to the Mackay Base Hospital and commenced on an intravenous glyceryl trinitrate drip and a CPAP by mask at 7.5 cm H20 – increasing her SaO_2 to 90 %. Blood tests showed her CK was elevated to 330 (Table 15 below) and 24 hours later her LDH rose to 255. A FBC showed a neutrophil leukocytosis (PMN 21.7, WCC 24.1), other blood tests were normal except a mil raise of her AST at 63 (NR <60). Her ECG showed sinus rhythm, right axis deviation, non-specific lateral t-wave changes, and poor R-wave progression anteriorly.

Her blood pressure started to drop to 100/61, her pulse remained at 130 and her SaO_2 was 90 %; the intravenous GTN was reduced and pressure support ventilation of 10 cm H2O and CPAP 7.5 cm was commenced, improving her SaO_2 to 94 %. She still had bilateral crepitations with a gallop rhythm, but remained alert and orientated.

An echocardiogram was performed 24 hours after initial envenomation. It showed severe global left ventricular dysfunction (Table 16) with slight mitral regurgitation. A left radial intra-arterial line and a left subclavian central venous line were inserted; the CVP being 11 mm Hg.

The next morning (28/3) Lisinopril 2.5 mg mane was commenced and frusemide 40 mg bd orally continued. She remained hypotensive (97/75mm Hg) and tachycardic (pulse 130) but her SaO2 had improved to 95 % on FiO^2 0.7 and PS 10/CPAP 7.5. The ventilation was ceased and she was given oxygen via a non-rebreathing mask.

The following day (29/3) her BP dropped to 82/54; bilateral crepitations remained in both lung fields. She was commenced on a dopamine infusion at 4 mcg/kg/min but as this produced little effect this was changed to dobutamine, slowly increasing to 12 mcg/kg/min; her BP improved to 95/50. The urine output fell off during the day, and with a CVP recording of 2-mm Hg a total of 500 ml of Haemaccell was given in boluses and Dopamine recommenced at 5 mcg/kg/min, with a substantial improvement in urine output and blood pressure to 112/60. Hypokalaemia of 3.4 was treated with oral potassium supplements.

The following day (30/3) the SaO^2 had improved to 94 % on room air, BP 109/50, pulse 117 and the urine output was good. The Dobutamine was weaned and the Lisinopril increased to 5 mg mane. Her ECG showed right axis deviation, t wave inversion in leads V1 - V4, diminished R wave in V2, but she remained in sinus rhythm.

The next day (31/3) the Dopamine was ceased and a repeat echocardiogram was performed, which showed an improvement in left ventricular function (Table 16). She was discharged two days later on Lisinopril 5-mg mane. A final echocardiogram ten days after envenomation showed normal LV function (Table 16). This is the first time that this has been shown (Case from Carney & Fenner 1997).

Table 15 - Irukandji envenomation – clinical and laboratory readings

Clinical and Laboratory readings	Case 1
Creatine Kinase (CK) (20-200 iu/l)	330
CK:MB ratio (NR 0-4 %)	4.9
AST (1-42 u/l)	63
LDH (110-250 u/l)	255
WCC (3.5-12.0 x10^9/l)	24.1
Initial ECG	Right axis deviation; T wave inversion V1-3
Late ECG	Right axis deviation; T wave inversion V1-4 Diminished R wave V2
CXR	Normal heart size; Bilateral hazy infiltrates
Highest BP (mm Hg)	140/110
Lowest BP (mm Hg)	82/54
CVP (cm H2O)	+ 12
Highest FiO2	0.7
CPAP via face mask	10 cm H2O pressure support + 7.5 cm H2O CPAP
Inotropes	Dobutamine 12 mcg/kg/min + Dopamine 5 mcg/kg/min
Complications	Initial hypertension Hypotension Respiratory distress Pain Poor urine output Severe LV dysfunction on echo which improved

Table 16 - Irukandji envenomation – echocardiographic dimensions

Echocardiographic Dimensions	Case 1		
	Day 2	**Day 6**	**Day 10**
LV Diastole (normal range 35-56 mm)	55	52	50
LV Systole (normal range 20-40 mm)	50	45	37
Fractional Shortening (normal range 25-45 %)	9	14	26

Cerebral oedema

A case has recently come to light where cerebral oedema occurred. This is the only time this symptom has been reported in the Irukandji syndrome (Fenner & Heazlewood 1997), although it was recently reported in a major chirodropid envenomation (Fenner & Williamson 1996) – see also, Case History in 5.6.1 above.

Case History - Cerebral oedema

A 7-year-old male was stung by an Irukandji in northern Australian waters at 1100 on 20 December 1981. At 2100 he was found to be confused, disorientated, and with tachycardia and tachypnoea. He had several episodes of profound sweating, cyanosis and agitation. Chest auscultation revealed bilateral course crepitations, more marked on the left and a

chest X-ray demonstrated increased interstitial markings consistent with pulmonary oedema. He had 4+ glycosuria and dextrostix of 130mg/100ml (N.R. 80-120mg/100ml) and had periodic verbal and visual hallucinations.

On physical examination he was pale and in obvious respiratory distress needing 6 litres of oxygen/min via facemask to maintain his central colour. Examination of his fundi showed blurring of the disc margins. Initial management consisted of intravenous (IV) dexamethasone and intermittent frusemide (lasix)(exact doses not known). His symptoms persisted for the first 24hrs of admission and on the second evening he became more disorientated and unresponsive to spoken word. He was given 100ml of 20% mannitol intravenously. Two hours later he had a huge diuresis with a wet bed, 3 hours later was answering questions with a grunt, and 4 hours later was obeying commands and was easily roused. Ten hours later he was talking freely and answering questions and subsequently made a complete recovery with no neurological sequelae.

This case of cerebral oedema has only recently come to light. Other cases have probably occurred, but may not have been so obvious. It is suggested that the conscious state is carefully monitored in all serious Irukandji envenomations, and appropriate investigation is made of any
deterioration.

Discussion

Pulmonary oedema is a proven complication of Irukandji envenomation (Fenner *et al* 1988; Martin & Audley 1990). It has now been proven that direct myocardial toxicity can occur, causing hypotension and pulmonary oedema (Carney & Fenner 1997). Mechanisms include excessive catecholamine release which has previously been reported as causing myocarditis associated with phaeochromocytoma and exogenous catecholamines (Rona 1985; Imperato-McGinley J 1987; Scott *et al* 1988), and direct myocardial toxic effects similar to *Chironex fleckeri* venom and pulmonary oedema may result from alterations in the endothelial permeability of the pulmonary vasculature due to a venom effect (Burnett in Williamson *et al* 1996, p255). A right heart catheter is probably necessary to differentiate these possibilities.

The measurement of cardiac enzymes, particularly the cardiac specific CK-MB fraction confirms that cardiac damage also occurs. The long-term result of this cardiac damage has not yet been confirmed in all cases, although present evidence suggests that the damage may be only temporary.

It would seem obvious that therapeutic trials are needed to devise an effective treatment regime; one such study with the author and Dr Ian Carney is under way at

the Mackay Base Hospital (See Medical treatment 5.8.4). However, there are many small hospitals in tropical Australia, in which medical staff change regularly and often come from other Countries. Trying to put such a therapeutic trial into place is difficult.

Treatment

The treatment is presented below in 5.8.4 but a recent pitfall in treatment which occurred in which a beta-blocking agent was inadvertently used, and which the author was consulted to assist in finding a solution, is presented:-

Beta-Blockers

In a recent case propranolol was given for the tremor associated with a victim of an Irukandji sting. As she also had hypertension it was thought that it might also help this. However, the victim became very hypotensive, and then developed acute renal failure which was later followed by cardiac failure:-

Case Study

Eighty milligrams of propranolol was given orally in an attempt to alleviate the tremor. At 0200 the patient was noted to be cold and clammy with severe peripheral shutdown and a relative bradycardia of 80, (compared to the previous 120bpm), with femoral and carotid pulses only just palpable, and a blood pressure of 60/25mm Hg. Despite the severe hypotension, the patient was noted to be alert and communicative. A bolus of 500ml of haemaccel (plasma expander) was given with moderate effect (BP 63/47mm Hg). Blood gases revealed pH 7.28 PaO_2 119 $PaCO_2$ 35 HCO_3 16 Base Excess -10 and oxygen saturation 98% on 4 litres per minute via nasal prongs. A femoral artery catheter was inserted which revealed a dampened waveform and a mean blood pressure of 60 mm Hg. The morphine infusion was ceased temporarily at that point. An electrocardiograph was grossly abnormal with a sinus rhythm of 78 bpm, a first degree heart block, broad complex QRS morphology, tall T waves and poor R wave progression. It was noted at 0300 that the patients serum potassium had risen to 8.5 mmol/litre (NR <5.5) with Na^+ 134 mmol/litre, Urea 9.3, Creatinine 116 and CK 331 IU. The severe hyperkalaemia was treated with 50 ml of 50% dextrose iv and short acting insulin subcutaneously. A cental venous line was inserted, and the CVP was noted to be 20 cm H_2O. A chest xray showed good line placement, normal heart size (allowing for the projection) and clear lung fields. An isoprenaline infusion was commenced at 0350. Electrolytes at this point showed K^+ of 8.0 mmol/litre, HCO_3 19 and Creatinine of 122. Five ml of 10% Calcium chloride was given intravenously and an ECG was repeated, which was showed normal sinus rhythm. At 0430 the K^+ was still 8.5, with HCO_3 16, AST 37, LDH 154, CK 331, and the white cell count was elevated at 17.9. By 0500 the isoprenaline infusion had been increased to 5 mcg/hour and the blood pressure was 82/51 with a heart rate of 84 bpm. The K^+ had then fallen to 6.5 with a pH of 7.28. A further 5 ml of 10% Calcium chloride was infused. The isoprenaline infusion was titrated against the blood pressure until 0630 when the BP was 112/79 with an

infusion rate of 12 mcg/hour. The blood sugar at this point was noted to be low at 1. 8 mmol/litre and 10 ml of 50% dextrose was infused. A repeat ECG was again felt to be in sinus rhythm with normal QRS morphology. At 0900 there were crepitations noted at the right lung base, and the oxygen saturation was 93 % on with an FiO2 of 28%. At 1000 blood revealed Na+ 141, K+ 5.7, Cl- 110, HCO3 21 Creatinine 85, Glucose 3.3, LDH 328, CK 392, CK:MB 21 and CK:MB/CK ratio elevated at 5.4%.

At 1300 the oxygen saturation was 92% on 12 litre per minute of oxygen via a facial mask. The blood pressure at this point was 121/62, CVP 13 cm H2O and heart rate 92 bpm while the isoprenaline infusion had been weaned to 4 mcg/hour. The isoprenaline infusion was ceased at 1430 with a BP of 112/59 and heart rate 97 bpm. At 1530 5 cm H2O of continuous positive airway pressure (CPAP) was commenced via a face mask with an FiO2 of 60% yielding an oxygen saturation of 92% and an intravenous infusion of glyceryl trinitrate was commenced. At 1700 the patient complained of severe pain in the lower limbs and profuse sweating and an intravenous infusion of morphine was again commenced.

The CPAP was increased to 10 cm H2O with an FiO2 of 60 % giving an oxygen saturation of 95 %. At 2300 the FiO2 was increased to 70%, and a fever of 38.1oC was noted. Blood cultures and a urine specimen were obtained which revealed no growth. The FiO2 and CPAP was gradually weaned over the night and at 0900 on the second day after admission oxygen via a face mask at 35 % was instituted with an oxygen saturation of 98 %. The K+ was then noted to be 4.0, CK 285, and CK:MB 11 (ratio 4.7%). An echocardiogram was performed on the third morning after admission which showed a mildly dilated left ventricle with mild global hypokinesis, LVD 54mm (NR 35-56), LVS 41mm (NR 20-40), fractional shortening 24% (NR 25-45), trivial mitral regurgitation and E point-septal separation which was mildly increased at 15mm (NR <8mm). Enalapril 2.5mg per day was commenced and the patient gradually mobilised. On the fourth day after admission the Enalapril was increased to 5 mg bd and the patient discharged on day five. Also a German-speaking Swiss, she elected to continue her world tour and was unfortunately lost to follow-up.

Antivenom research

As mentioned in 4.1 above, antivenom may prove helpful for Irukandji envenomation - as it has for *Chironex* envenomations.

In April that year (1996), right at the very end of the Irukandji season (usually October-April) the author was fortunate to have his `catcher', a local net fisherman, see some very small carybdeids swimming in a sheltered area behind a rock wall at the mouth of the Pioneer River in Mackay. He caught nine of them, using a bucket, but then carried on with his own fishing for a while. Unfortunately this meant that the specimens were in poor shape when they were received, as they were in very warm water which had not been changed for several hours. They were quickly snap-frozen and stored in a freezer at minus 70^{o} Celsius until they were ready for shipment to

Melbourne where they were to be analysed. Six of them proved to be Irukandji (the others were immature *Tamoya* sp – C Wiltshire, 1996, personal communication) and preliminary studies of the Irukandji venom have proved interesting (see below).

The new method using the net towed by an IRB proved to be very effective when over 3 days in December 1996, conveniently whilst the author was there to assists, there was an infestation of Irukandji at the beach where the net and the IRB were stored. This was the very beach where Jack Barnes had laid on the bottom for many hours in 1966 before catching the first Irukandji (Barnes 1966), named *Carukia barnesi* in honour of his efforts (Southcott 1967). In this 3 days 29 Irukandji specimens were caught, and then stored in the local Cairns Surf Life Saving Club's freezer.

These specimens were then sent to Melbourne by air courier. Unfortunately they were lost on journey, and when they were found 5 days later only 3 very poor specimens were able to be salvaged. These are now being studied (C Wiltshire, 1997, personal communication). Since then the weather conditions have been unfavourable and only 3 further specimens have been caught this way.

A further specimen was caught when it was seen lying on the sand at Mackay Beach, by the lifeguard. It caught his eye by its fine flapping movements, reflected in the sunlight. It was transferred to a container and delivered to the author, whereupon it was promptly frozen. This is the first reported occurrence of an Irukandji being beached.

There is insufficient venom in these four specimens to be able to inject into rabbits (S Sutherland, 1997, personal communication) to develop a preliminary antivenom. There will, however, be further venom recovered for continued investigations. As the season is rapidly drawing to an end, it is unlikely that any more will be caught until the next season (October 1997 to April 1998).

Irukandji venom

The Irukandji specimens caught last year have had some preliminary work. Several of the largest molecular weight fractions of the Irukandji venom appeared to cause tachycardia in rat heart preparations, and some of the smaller factions had "different effects" (C Wiltshire, 1997, personal communication).

Unfortunately, further work has not been published, nor even analysed further at the time of this report. It is however, being prepared for a new toxicological paper on the Irukandji venom of which the author is a contributor.

Differential diagnosis of Irukandji syndrome

One of the problems that often occurs is phone calls are often received from either hospitals, doctors, or even divers, requesting information on Irukandji stings which may easily be confused with two other, more-common, medical problems: -

Myocardial infarction

Cases with the initial chest pain of the Irukandji syndrome, especially if pulmonary oedema develops, have in the past been misdiagnosed as an acute myocardial infarction with developing heart failure (Fenner *et al* 1988). This may be reinforced by a history of swimming (exertion) especially if the history of a mild sting is not elicited, or is forgotten by the victim. Generalised muscle pain, however, is usually a clearly elicited fact.

The situation is further confused if blood is taken for cardiac enzymes as the Creatinine phosphokinase (CPK) and the CK-MB fraction may be raised well above the normal levels. In some severe cases, often with obvious pulmonary oedema, possibly with myocardial toxicity and hypokinesis, the CK-MB may be well above the normal range (<8), with an abnormal, and significant, ratio (NR <1.6)

Decompression sickness

The Irukandji syndrome in a diver also resembles decompression sickness, and may present a difficult differential diagnostic problem (Williamson 1985b). There have now been a number of cases around the Great Barrier Reef who have phoned the Divers Emergency Service Australia (DES) number when, a short time after surfacing, a diver suddenly develops severe low back pain, chest pain (`trouble' breathing) and is distressed and restless (Fenner *et al* 1995).

Careful history taking is essential. A history of a minor sting on the back of the neck when surfacing, a small mark, often difficult to see, and/or careful differentiation of the symptoms is necessary. All this is conducted over a radio telephone from a dive boat on the reef to the DES phone Base in Adelaide - no easy task.

For information and data on Irukandji stings, refer to Sting results 5.6.2.

Compression immobilisation trial

The author has just arranged a trial that will start next year. Surf Lifeguards who patrol the beaches in north Queensland have become very adept at recognising the faint pink patch, often with local pilo-erection or sweating, as that of an Irukandji – even well before the delayed onset of systemic symptoms. The author believes that this provides a window of opportunity to trial the first aid treatment of compression immobilisation.

As from next season (approximately September 1997), lifeguards recognising fresh Irukandji stings will treat patients into two groups, assigned randomly. One group will have the area washed with vinegar, a vinegar-soaked pad applied over the sting and covered with a compression bandage, and then the whole limb immobilised. The other will have no treatment.

The lifeguard will then fill out the top form of three. In it will be detailed times of onset of symptoms, and if, in the thoughts of the assessor, they were as severe as usual. The same form would then be completed by the transporting Ambulance Officer, and the third copy completed by the doctor (treating nurse). Collation of the forms would then be made and the results stored in a database. The results will be then be statistically analysed to see if this would be a reasonable first aid method to apply.

5.6.3 Morbakka

Taxonomy and Distribution in Australia

The information on this large carybdeid is mentioned in 5.1.1.

Envenomation

Victims are usually stung whilst swimming (Fenner *et al* 1985). The pain is not severe, but varies from "pin-pricks" to a marked burning sensation (a local name is `fire jelly'). Tentacles may remain on the skin, but are usually not easily detached – unlike chirodropids. Initially, the skin has raised white wheals with a surrounding red flare – looking, as well as feeling, that the skin is on fire. This burning pain may last for almost 24 hours.

Often within half-an-hour, minor `Irukandji'-like symptoms appear, although much more mild. Victims may develop a cough, backache and a feeling of a lump in the throat, symptoms that often last for the next 24 hours. The skin lesions are basically

unchanged 24 hours later, but usually stop burning and become itchy and tender to touch. The lesions became paler by the third day after envenomation and develop a papulovesicular appearance that may persist for 10 days. Rarely, in some severe stings, similar to chirodropid stings, the skin may darken and become blistered and necrotic.

Clinical record 1

At 11.00 a.m. on January 20, 1984, while swimming with her father at Margate Beach, Moreton Bay, in murky water one metre deep, a 12-year-old girl was stung by a "Moreton Bay Stinger". At the time there was a strong on-shore wind.

She dived under the water and on surfacing she immediately complained of "pin-pricks all over her shoulder". Putting her hand to her neck and shoulder she pulled off a long tentacle that disappeared under the water. She described the tentacle as slightly white and translucent, as long as her arm, and as thick as a telephone cord. The jellyfish was seen by her father who immediately identified it visually as *Tamoya* from photographs that he had seen beforehand and confirmed this by checking afterwards.

Initially, the skin had raised white wheals with a surrounding red flare. The predominant symptom at this time was a severe "burning" pain, which lasted for almost 24 hours. Within half-an-hour, the girl had developed a cough, backache and a feeling of a lump in the throat - symptoms that lasted for the next 24 hours. Paracetamol (500 mg) and dexchlorpheniramine maleate (2 mg) were given by mouth. They had little effect apart from that of sedation.

The skin lesions were basically unchanged 24 hours later, but had stopped burning and had become somewhat itchy. They were also tender to touch. The lesions became paler by the third day after envenomation and had a papulovesicular appearance that persisted to the 10th day.

Clinical record 2

On 21 November 1996, while swimming in a small bay in Sydney Harbour, a 44-year-old woman felt a sudden burning pain on her right arm and her back. Unable to move the arm properly because of local severe pain and paresthesiae, she struggled to shore. Here she noticed she had several long raised white wheals down her right arm and on her back. These were surrounded by a bright red flare. She also had some low back pain, although there were no marks in this area, and she felt somewhat anxious and sweaty.

She received no treatment and over the next few hours the sting marks became darker and over the next few days were dry, but necrotic. When the scabs fell off 7-10 days later she was left with open sores which took a further 2 weeks to heal, but left scarring. A US resident she returned home, but has been lost to follow up.

The sting marks were too big for the smaller *Carybdea rastoni* (see below). As specimens of *Tamoya virulenta* had been caught in a Sydney sea water pool on the beach in October that year (J Bartlett, 1996, personal communication), this was probably the stinging jellyfish in this case, especially as the sting marks and systemic symptoms match *Tamoya virulenta* ("Morbakka") symptoms fairly closely.

5.6.4 Carybdea rastoni – the Jimble

This is a small carybdeid that usually has a minor sting that may not even be felt (Fenner & Williamson 1987). However Williamson (1996, personal communication) states that at certain times of the year, pearl divers in Broome give a clear berth to the Jimble as it can cause symptoms as severe as an Irukandji syndrome. At other times of the year (winter – June to November, when the water is cold) their sting is very mild. No formal research has been undertaken in this area to date.*

- The observation that the potency of the toxins of different cnidaria may vary with environmental water temperature/seasons is another promising research direction.

Jimble stings are a regular summertime encounter in Gulf St Vincent, South Australia every summer and cause numerous hospital casualty appearances for pain relief. The animal was first identified in this coastal locality in 1886 (Haake 1887). The stings almost invariably occur in the late afternoon, and result from surface swarms of the animal. Ocular stings are also seen (J Williamson, 1996, personal communication). Jimble venom toxinology research awaits attention, with possibly potentially far-reaching results for carybdeid sting management.

Another unusual feature with this particular jellyfish is, that at times it appears to have `warts' on its bell, which contain stinging nematocysts. At other times the bell is clear. Other than this species, just the Irukandji and the Morbakka have similar nematocyst warts.

The author treated a small sting (7cm tentacle length) in 1983 when an 11-year-old boy was stung whilst surfing off Mullaloo, Perth, Western Australia. The boy was acutely distressed with severe localised pain. However he had no systemic symptoms. The author himself suffered extensive stings to his face in 1987 (Fenner & Williamson 1987) but had no pain and no itching – in fact no sensation whatsoever.

Because of these gross differences in sting severity, and the different appearance of the bell of the jellyfish, perhaps again we are talking of two species that look alike, but have not yet been described – similar to the Morbakka.

5.6.5 Carybdea sivickisi

This is a the smallest cubozoan known, has been described in a number of Countries in the temperate waters of the Indo-Pacific; Hartwick (1991b) first described on the Great Barrier Reef in 1991. Very little information has been published on its sting, although Hartwick (1991b) described it as "minor skin pain". However, recently a 26-year-old-woman phoned the author describing a dive trip on the outer Barrier Reef off Townsville when after jumping in the water she and her diving companions felt multiple small stings on every exposed area of the body. They could all see thousands of tiny jellyfish swimming actively around, which the dive master said were fairly common and were called "sea lice". Although there are no actual creatures called sea lice, this is a very common term applied by swimmers to the multiple small stings that may occur in the temperate waters of Australia (sting reports from Surf Life Saving – see 5.5). In view of Hartwick's (1991b) exact description of the appearance of the jellyfish in the water off the Great Barrier Reef, along with their stings, the most likely cause of these "sea lice" stings often reported on the reef, and by swimmers off temperate Australian beaches.

Despite many of her diving companions being stung and suffering no long-term effects, this lady who contacted the author, and who lives in Western Australia, has continued to get skin itching since her original envenomation three months prior. There are no marks visible and no explanation can be offered, however, antihistamines relieve the itch. The author is attempting to get some serum from her so that it can be lyophilised and sent to Burnett in the United States for serology (Burnett *et al* 1988). This may help if there is any likelihood of delayed hypersensitivity, although with the lack of stings marks and the longevity, the author feels it may be unlikely.

5.6.6 Physalia physalis – the Pacific man-o'-war

Victims of the Australian *Physalia physalis* experience severe skin pain, with only slow relief from cold packs. Up to 30 minutes later mild Irukandji-like syndrome with nausea, vomiting, abdominal colic, limb muscle cramps, anxiety, restlessness and chest pain may occur in some 30% of cases of envenomation. The chest pain is due to intercostal and back muscle myalgia resulting in inspiratory pain cutting off the breath with a "grunting" noise (Fenner *et al* 1995). These symptoms have are very similar, but much more mild, than the syndrome after envenomation by *Carukia barnesi* (the Irukandji) described above.

Case study

After a sting from a *Physalia* sp. in Mackay a 15-year-old girl suffered intense skin pain, worse than the usual envenomation from *Physalia utriculus* (the `bluebottle').

Thirty minutes later this was uncharacteristically followed by severe low back pain, muscle cramps and symptoms of excessive catecholamine release, including nausea, sweating, anxiety and restlessness (Fenner *et al* 1993a). The specimen responsible for stinging the girl was captured and examined by the author. It was noted that this *Physalia* sp. was macroscopically different from the common Australian *Physalia*, known as the `bluebottle' (Fenner *et al* 1993b).

Fortunately *Physalia physalis* are much less common that *Physalia utriculus* and consequently less of a problem in Australian waters.

5.6.7 Other Australian jellyfish

These all cause skin pain of varying intensity, although not severe. The symptoms of pain are usually easily controlled with cold packs applied to the skin (Exton *et al* 1989)(see Treatment regimes below).

Physalia utriculus

Physalia utriculus ("bluebottle") stings cause immediate skin pain and whealing, which is quickly and efficiently relieved with cold packs or ice. Some pain may be felt in the regional lymph glands and occasionally systemic symptoms including nausea, lethargy and dyspnoea have been reported (Williamson 1985a).

Cyanea capillata

The “hair” jellyfish causes skin pain and zigzag whealing; it is not a great problem to Australian bathers although large numbers of victims may be stung some years (see 5.5.3). The symptoms of skin pain are usually settled with cold packs.

Cyanea may occasional be a nuisance by the sheer mass of their numbers in the water. In 1960 a Burmese naval vessel was delayed sailing from Cairns Harbour, North Queensland when its water intakes became blocked with *Cyanea*, causing engine overheating (Cleland & Southcott 1965, p.153).

Rhizostomes

These are jellyfish with no tentacles, but bearing eight thick `mouth arms’. *Rhopilema* species in Australia is a problem. However, large numbers may be present on the beaches of eastern Australia which are usually *Catostylus mosaicus,* a species that was first described in 1824 from Australia (Quoy and Gaimard 1833).