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#### **EDITORIAL**

The increasing interest now being shown in the interaction between man and that seemingly humble and helpless group of marine creatures, the iellyfish. has as a probable basis the increasing numbers of people who enter the water off the Queensland beaches. The economic impact on the tourist industry of stories about it being necessary to wear all-over protective material such as pantyhose before entering the water cannot be anything other than adverse, and the advice that one should pack a large bottle of vinegar with the ultraviolet light barrier chemicals is both accurate and discouraging. It is against this background of increased awareness of the importance of research into marine stingers that the Australian Institute of Marine Science (AIMS) hosted a symposium in April 1986. Some of the papers presented there form the core of this issue of the Journal.

It is abundantly clear that these simple creatures are able to produce complex chemicals and a highly efficient delivery system with what the Pentagon would accept as a massive overkill capacity, as the creatures' usual prey is only a minute proportion of the mass of even a child. It is humbling to realise that our technology is far outstripped by nature's manufacturing expertise and that we are struggling to identify chemicals that nature has been efficiently manufacturing for thousands of years. As humans are affected by the toxins once they enter the body, the most promising lines of work are based on increasing protection, reducing or destroying the power of the weapon to fire, and the last ditch resort to antivenoms to reduce or nullify the systemic effects of the toxins. Victims may find some comfort in the knowledge that if vinegar aggravates their trouble this indicates that the stinger was not one of the dangerous ones. Description of the symptoms resulting from contact with marine stingers indicate that the problem they create is serious and worth the research effort presently being expended.

Case reports are the raw ore from which can be

extracted information vital to making improvements in our recognition of what problems occur and how they are actually managed. Full and honest reporting of events, which includes mention of imperfections in diagnosis and management, is of great importance and the resulting reports have a value far greater than those where the author is presented as a perfect clinician. There are certainly some dangers in honesty but it is hoped that an educated medical profession will fight the good fight and succeed in educating the legal profession to the facts that diagnosis is a skill and not a scientific certainty and that improvements in management flow from sharing information of diagnostic and management difficulties. There is great gain to the community from learning through the experiences of others, which reduces the frequency of reinventing the wheel, or perpetuating some incorrect belief or management.

Safety is multifactorial and this is now well recognised in diving. There is an ongoing interest in defining standards for both training and medical fitness. The latter have made more progress in Australia and the UK than in the USA where a recent UMS sponsored workshop has noted the absence of any requirement for a medical examination before commencing diving instruction. Indeed there may be no need for the majority of divers to have a diving medical degree of evaluation. This has not been debated, except in the UK by the British Sub-Aqua Club (BS-AC). Information accessibility is increasing with the change from reliance on written to computer modes of storage. This may be an opportune time for an impartial review of the occasions where health factors have been noted in a diving misadventure. The UMS Workshop on Emergency Ascent Training (1977) illustrates some problems which can arise when highly emotive matters are being discussed, where the beliefs of the participants are presented and there is no apparent initial agreement of the actual facts. It is probable that rules based on facts would fare better it later questioned in a court of law.

# **IBIS HOTEL, MOOREA, FRENCH POLYNESIA**

#### BLINDNESS DUE TO EXTRAOCULAR PRESSURE

#### AN UNUSUAL CASE FOR HYPERBARIC OXYGEN TREATMENT

# Peter McCartney

The patient was a fit, 37 year old man, Mr AS. Towards the end of an informal family game of cricket, a cricket ball struck Mr AS on the left orbit causing severe pain and a feeling of fullness above and below the eye.

The patient felt that if he blew his nose, he would relieve the fullness. He did this within two minutes of the injury, and in his own words "I immediately knew I had done the wrong thing".

The pain above and below the orbit became more severe, and he had double vision. The double vision cleared within 5 minutes and he could see again, but he was still in pain.

His wife drove him home, arriving at about 7.00 pm. The swelling did not seem to be great, and after a meal he felt more comfortable and went to bed. This was about 8.30 pm. His overall state seemed to him to have improved, and although the eye was a little swollen, he felt it was not bulging and he settled and went to sleep peacefully.

He woke around midnight, with severe pain and a feeling of sheer panic. He jumped out of bed and ran about. His wife managed to calm him, and they both realised his eye was bulging.

He took two disprin, and this had no effect, so he took two Panadol. This also had no effect, and he started vomiting. It was decided to ask for medical advice. The family doctor was away and they knew this, so they phoned a doctor in Sandy Bay, 24 kilometres away, and asked him if he would come out and help. The doctor agreed to this and made the journey.

On arrival, the doctor made an attempt to wash the eye and examine it, but quickly decided that the swelling was to great to permit any examination, and took the patient to the Royal Hobart Hospital in his car.

On examination at the Royal Hobart Hospital, he had marked exophthalmos on the left with an increased intraocular pressure due to pressure of the surrounding tissues on the globe. There was no perception of light in the left eye which had an unreactive pupil. Ophthalmoscopy showed an exsanguinated fundus.

His management included adequate analgesia. A lateral canthotomy was performed and he was given diamox and mannitol. Massage of the globe of the eye was also performed.

CAT scans were performed. An axial scan through the orbits showed (L) proptosis and a considerable amount of air inside and outside the muscle core. A more

caudal scan again showed extensive air within the orbits as well as fractures involving the orbital wall posteriorly and the adjacent ethmoid. The optic nerve appeared uninterrupted. A scan through the maxillary sinuses showed opacification of the left maxillary sinus. There was subcutaneous air over the anterior wall of the left maxillary sinus. Finally a coronal scan showed a fractured left orbital floor, air in the left orbit and an opacified left maxillary sinus.

This man had an apical fracture of his left orbit with no evidence of direct bony compression of the optic nerve. There was air in the orbit and he had a blind eye due to external pressure on the eye.

The retinal artery is narrow, and is an end artery system. It supplies only 30 per cent of the retina's oxygen requirements. It has intrinsic autoregulation of flow,  $PO_2$  and  $PCO_2$  being the two regulators. High  $PO_2$  causes vasoconstriction while a high  $PCO_2$  causes vasodilation.

In contrast, the choroidal system is a wide bore network. Venous oxygen saturation is an astonishing 95 per cent. Flow is regulated by the sympathetic nervous system and accounts for a 70 per cent share of the retina's oxygen supply. These features of the blood supply to the eye are shown in Figure 1.

Once the intraocular pressure rises above arterial pressure, blood no longer perfuses the eye. The same time constraints for anoxia apply to the eye as for brain tissue.

It was considered that orbital decompression would be of benefit, but in view of the long delay, possibly unsuccessful. An optic canal decompression procedure was offered to the patient by the neurosurgeon. The patient declined this. One factor in this decision was that his father had died following neurosurgery.

Therapeutic compression breathing oxygen was ultimately decided upon as the air mass was thought to be the major factor in producing the raised intraorbital pressure.

He was compressed to a pressure equivalent of 9 metres of seawater (MSW) (30 feet of seawater (FSW)) and put on oxygen. The compression profile was

	MINUTES	MSW
12.51	zero	surface
12.56	+5	9
13.56	+65	9
14.26	+95	surface

After treatment there was decreased proptosis, his discomfort diminished. The blood flow in the eye returned to normal however his vision and pupillary responses were unchanged.



If compression and hyperbaric oxygen had not been used he would have been a candidate for enucleation. At the time of writing, both his eyes appear normal, and move normally. There is only a small superotemporal area of visual field left in the left eye. There is no pain or discomfort. The patient has made a good adjustment to his injury, and is grateful for the treatment as he realises that the injury could have

Thanks are expressed to Drs E Ryman, Deputy Director, Royal Hobart Hospital; Michael Treplin, Ophthalmic Consultant to the Royal Hobart Hospital, David Griffiths and Michael Martyn, Department of Anaesthesia, Royal Hobart Hospital; and G Brian, Department of Ophthalmology, Royal Hobart Hospital.

resulted in him losing his eye.

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#### PULMONARY BAROTRAUMA OR SPONTANEOUS PNEUMOTHORAX A CASE PRESENTATION

#### Beris Ford

Let me introduce you to my area. I live in Whangarei which is 160 km north of Auckland, New Zealand, and is in the middle of a long isthmus, the East Coast of which is popular for scuba diving. The Poor Knights Group of Islands lie 30 km off the coast. They are New Zealand's Mecca for scuba divers and about 20,000 dives per year are done here and perhaps 250,000 in all of Northland.

J, a part Maori, was 20 years old when she came in to see me for a scuba diving medical. There was not much in her history, a couple of bouts of cough with sputum, and occasional nasal obstruction. There was nothing much in the examination except she had larger lungs than predicted, Vital Capacity (VC) 4.3 1, predicted, 3.8 1, Forced Expiratory Volume in 1 second, 3.45 1, predicted, 3.25 1, and a slight reduction in percentage FEV/VC, actual 80 per cent, predicted 88 per cent. Despite my urging, she refused to have a chest film because she would not take time off work. She worked in the Government note printing factory where security is naturally very strict. To attend for an x-ray would mean taking a whole day off work.

Because of pressure from her and her diving instructor, I passed her, but explained carefully my reasons for requesting a chest x-ray.

I next saw her 4 months later and had forgotten about her medical and her diving, and she did not bring it up. She attended with a chest discomfort, and a cracking noise in her chest. The diagnosis is so obvious now but since she gave no history of chest injury or diving at the time I did not suspect it. She was tender over the sternum and the left 2nd rib. Again she refused to have a chest x-ray and I asked her to come back if it did not resolve rapidly,

She did not do anything for another 2 months when she attended the doctor at her place of work. Then her symptoms had gradually progressed and she was short of breath and had a tachycardia. By that time she had definite signs and he was more successful in getting her to have an x-ray. This showed a complete left pneumothorax and some shifting of the mediastinum.

Taking a more detailed history disclosed that she had been diving on most weekends since her qualification. She was therefore no longer a novice but on her own admission, not very confident.

She had a dive several weeks before her first presentation. This was to 10 metres, and she just did not feel right. She had difficulty staying down and she felt she was floating up all the time. She felt uptight and panicky. After the dive she felt abnormally tired and exhausted.

It was about two to three days later that she first noticed what one described as the cracking noises and slight retro-sternal discomfort. It was a week or so later that she first sought medical attention.

You will now be aware that it took 3 months from the dive to the diagnosis of her pneumothorax. Once diagnosed, she was admitted to hospital.

A chest drain was placed and despite the use of negative pressure she lung did not expand fully. It was almost fully expanded after 8 days but by about 13 days she developed an effusion with infection. The fluid grew an enterobacteria.

A few days later, because of the failure of conservative management, a surgical operation was sought. Here, there was no delay and she was taken to theatre the next day.

At operation, she had a normal bronchoscopy. About 700 ml of blood stained fluid was present in the chest. There was a fibrin coating over the lung and it seemed that this might have been the reason why the lung would not expand. There was no evidence of any site of leaking when the lung was inflated. However, a 5 cm x 3 cm cyst was found in the apex. The cyst was removed and a pleurectomy was performed. Ten days later she was sufficiently recovered to be discharged.

She has, of course, been advised to discontinue scuba diving. Her next foray on the sporting scene was to take up parachuting. This can also have hazards for individuals with lung problems. Parachuting did not last long as she developed back pain and x-rays showed her to have a spondylolisthesis. So parachuting did not seem to be such a good idea.

# DISCUSSION

I found it quite difficult to get much out of the literature on pneumothoraces and could find nothing on pneumothoraces and diving at the Auckland Medical School. I did get some papers on spontaneous pneumothorax. This may well not be too wide of the mark as in the case of J, it is not really possible to say whether this was a spontaneous pneumothorax or whether there was an abnormal pressure change, a true barotrauma aetiology. In this case, the treatment was as for a spontaneous pneumothorax.

A variety of treatments for spontaneous pneumothorax have been used.

- 1. Simple aspiration.
- 2. Silver nitrate pleurodesis and drain.
- 3. Tetracycline pleurodesis and drain.
- 4. Under water drain, with or without negative pressure.
- 5. Surgery.

About 95 per cent resolve satisfactorily with no treatment or with intercostal drainage. These may be about a 20 per cent recurrence rate in the conservatively treated group.

# CAUSE

It is stated that it is much more common in young adult males, and that is nearly always due to rupture of a sub-pleural bleb or bulla. What causes the blebs or bullae is not known for certain. inflammation, ischaemia and congenital causes have been suggested.

# HISTORY

Air in the pleural cavity was described as far back as the mid 17th century. It was first called pneumothorax by Itard in 1803, and in 1826 it was seen to be caused in one case by rupture of an emphysematous bleb. Schminckle, in 1928 described multiple blebs in the apices of both lungs in a young man who died from bilateral spontaneous pneumothorax. Dolley, in 1929 performed the first known successful surgical treatment for a ruptured bleb in a child aged 3 months. A number of reports of surgical treatments appeared in the thirties and by the early forties pleurodesis in simple forms was reported.

In one review of patients coming to surgery, 94 per cent had blebs, and these were multiple in 60 per cent. The blebs varied in size from  $0.5 \times 0.5$  cm to  $1 \times 2$  cm. In another series 90 per cent had smoked for from two to thirty years before the incident. There was also a bias toward heavy smokers.

Was this really a spontaneous pneumothorax or was it pulmonary barotrauma?

What is the likelihood of a 5 cm x 3 cm bulla being detected on a chest x-ray?

How do we diagnose the small blebs apart for waiting for a pneumothorax?

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EDITOR: Readers may like also to read the case report presented by SYNEK VM and GLASGOW OL. "Recovery from alpha coma after decompression sickness complicated by spinal cord lesions at cervical and midthoracic levels" in <u>Electreoencephalography and Clinical</u> <u>Neurophysiology</u>, 1985; 60: 417-419.

#### CEREBRAL ARTERIAL GAS EMBOLISM A CASE PRESENTATION

#### Peter Chapman-Smith

In mid March 1984, a pleasure dive ended in disaster. G, an experienced, trained scuba diver aged 22 years, ventured to his first dive site in the outer Cavalli Islands, off Matauri Bay, a little north of the Bay of Islands on New Zealand's East Coast. He recalls a brief 15-17 minute descent to 27m (90 feet), but feeling uncomfortable in the prevailing current, he decided to surface with his buddy. At 0930 he reached the surface conscious, inflated his buoyancy compensator, and then fell suddenly unconscious within 2 or 3 minutes. In this brief time he had complained to his buddy of some numbness and paraesthesiae in his arms and legs, particularly the latter. He did not vomit nor cough up any blood.

On the previous day he had done a single scuba dive at 1500 for 25 minutes to a maximum depth of 24m (80 feet). That evening he had consumed approximately 6 cans of beer and a third of a bottle of wine at a party.

The ensuing evacuation was notable for its considerable tardiness. They contacted Whangaroa, a nearby deep sea fishing harbour, by CB radio immediately. A rescue helicopter based in Auckland, 200 miles away was requested by 1015, the nearer helicopter in Whangarei being unavailable. The diver was given oxygen via a mask when they arrived at Te Ngaere Bay at 1000. He was taken by ambulance to nearby Kaeo Hospital and regained consciousness en route. He was apparently confused but could give his name. Within an hour he developed laboured respiration and hypertonicity in all four limbs.

The helicopter requested for 1015, eventually arrived at Kaeo Hospital at 1330. He was stabilised for transport, then the aircraft headed further away from Auckland to Kaitaia to refuel! They flew at 500 feet down the West Coast, stopping approximately every 15 minutes for reassessment by the paramedic on board. G finally arrived at the Devonport Naval Base in Auckland at 1600, some six and half hours after surfacing. The Naval Hospital medical team recompressed him for 6 hours on a 60 foot Oxygen table. In spite of treatment he was a spinal paraplegic when he was transferred to the Critical Care Unit at the Auckland Public Hospital. He required 2 1/2 weeks of intermittent positive pressure ventilation (IPPV) and a tracheotomy. At this point he was conscious, paraplegic and with an executive dysphasia. He was transferred subsequently to the Otara Spinal Unit for 6-7 months, then to Northland Base Hospital for a year.

During his hospitalization, various investigations were done. CSF was normal. CAT scans 2 days and 5 days post accident were normal. Repeated EEG's showed initial Grade III abnormality and a subsequent return to normal. Somato-sensory evoked potentials (SSEPS) showed bilateral high cord lesions with demyelination in sensory pathways. Rapid onset lesions of the spinal cord and cerebrum were reported, which is consistent with multifocal CNS damage caused by gas emboli.

Now he is glad to be flatting again, engaged to be married and commencing work again in a local Whangarei Laboratory.

 ${\it He}\ has\ patchy\ neurological\ recovery\ with\ an\ incomplete$ paraplegia at roughly T5 to T7 levels. It is more like T4 on the right and T10 on the left. He has normal upper limb power, but with poor right sided co-ordination. He still has symmetrical numbness of his first and second finger tips, poor sensation in his feet, but with return of sensation in his legs, this being better on the left. He has no bowel or bladder control. His hearing remains good, as does his speech. Although improving, he has a residual short term memory deficit. A reduction in eye-hand co-ordination and in particular visual co-ordination has made reading difficult. His balance is poor. He feels as though he will fall to the left. He maintains erections, but is unable to ejaculate ("as yet" he tells me). Muscle spasms in his legs are a problem, and are controlled with relaxants. His only other medications are aperients and Ubretid, an anticholinestase. He has frequent urinary tract infections as he self catheterises once or twice daily.

He is a non smoker. He had completed his diving training 5 1/2 years previously. He had been diving three times a week in the 5 months before his accident. These dives included bounce dives to 69m (230 feet). He had several minor undiagnosed (at the time) bends. Elbow pain came on in the boat after the dive on three occasions. He observed arm and leg numbness whilst still in the water after several dives in the last 2 years of his diving. He usually dived with twin 80 cu ft tanks, carrying a DCP, a watch and a single hose regulator with an octopus regulator.

Of particular note, he developed a cough 6 months before the fateful day. This progressed to pleurisy so he consulted his GP. He had several subsequent chest infections in the 3 months before his accident. One week before his accident he felt short of breath while snorkel training in a pool and consulted his GP again. A chest x-ray was ordered and was reported as normal. He sought specific advice about the safety of diving and was evidently reassured.

This is a tragic story, memorable for several features. He was an experienced diver, undoubtedly pushing his luck with regard to the USN tables which are the accepted sports diving tables in New Zealand. He

probably had numerous minor bends, and ignored them before his accident. His temporary unfitness to dive, with multiple chest infections, raises the question of how long divers should be banned from scuba use after a chest infection. He was given questionable medical clearance to dive. The tardy transport to a recompression facility delayed recompression. What was the diagnosis? Was it CAGE or was it rapid onset decompression sickness?

# POSTSCRIPT

This case stimulated discussion and consultation with the emergence of a rapid evacuation network for diving accident victims in the far North of New Zealand.

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#### A CASE OF ALTERNOBARIC VERTIGO

#### John Knight

Alternobaric vertigo, which was first described by Lundgren in 1965, is usually noticed on ascent. Lundgren was interested in the condition as he had suffered "sudden rotational vertigo during ascent from diving as well as in the hyperbaric chamber." His paper was based on the replies to a questionnaire about vertigo sent to 550 members of the Swedish Association of Sports Divers. He received 354 answers of which 92 (26 per cent) had experienced vertigo during scuba diving or when breath-hold diving which could not be explained as due to some known cause.

26 divers had suffered vertigo due to such causes as caloric stimulation, decompression sickness, seasickness, sensory deprivation, food poisoning, overdistention of the gut with air, exertion, hypoxia or hyperventilation and were excluded from the study. In this condition one middle ear pressure is higher than the other. This results in the labyrinth on that side being exposed to a higher pressure, transmitted through the round window, than the other and the result is a mismatch of messages from the two labyrinths and this is misinterpreted by the brain as a rotatory movement. The usual cause is failure of one Eustachian tube to function, so retaining air in the middle ear. Eventually, the pressure in the affected side is sufficient to open the Eustachian tube and air blows out. This equalises the two middle ear pressures and all is well again. Failure to equalise one ear completely during descent is the basic cause. The result of a failure to equalise is at least some degree of swelling of the lining of the middle ear. If this swelling is sufficient to obstruct the Eustachian tube the scene is set. Edmonds classifies alternobaric vertigo as a middle ear barotrauma of ascent in most cases but it can be due to unequal vestibular responses.<sup>2</sup> Edmonds' classification is given in Table 1.

I wish to present a case of alternobaric vertigo which not only did not commence on ascent but also continued long after the dive was over and recurred with every dive.

# TABLE 1

# VERTIGO IN DIVING

Due to unequal vestibular stimulation

- 1. Caloric
  - 1.1 Unilateral external auditory canal obstruction
    - 1.1.1 Cerumen
    - Otitis Externa 1.1.2
    - 1.1.3 **Miscellaneous**
  - 1.2 Tympanic membrane perforation
    - 1.2.1 Shock wave
    - 1.2.2 1.2.3 Middle ear barotrauma of descent
    - Forceful auto inflation
- 2. Barotrauma
  - 2.1 External ear barotrauma of descent
  - 2.2 Middle ear barotrauma of descent
  - 2.3 Middle ear barotrauma of ascent
  - 2.4 Forceful auto inflation
- 3. Inner ear barotrauma
  - 3.1 Fistula of inner ear window
- 4. Decompression sickness
- 5. Miscellaneous

Due to unequal vestibular response

- 1. Caloric
- 2. Barotrauma
- Abnormal gas pressures
- 4. Sensory deprivation

The diver is a man in his 30s who has been snorkelling for 16 years and diving for 8 years. For the first seven years of his snorkelling he was quite unable to equalise his ears, which limited the depth he could dive to when spearfishing. Nine years ago he was spearfishing in a competition and was not doing as well as those who went deeper. So he went down and this time was able to equalise. However, it could not have been proper equalisation, as he was completely deaf in one ear for two weeks! Having learnt to equalise he learnt to scuba dive and had no problems with his ears until 1985.

His work involves many snorkel dives and two shallow tank dives every day that the weather is suitable. He has done this for a few years. However, in about April 1985, he started to develop vertigo and nausea after about half an hour of snorkel dives. At first this did not occur after every occasion but it soon became a regular event. As his livelihood depends on his diving he continued to work. Although he had vertigo with snorkel diving he did not have any when using scuba. After about 6 months his vertigo came on after ten minutes with the snorkel. At this time he decided to take a holiday and was out of the water for some three weeks. During this holiday he went for a trip in a boat and was seasick for the first time in his life.

When he went back to work he used scuba in an attempt to avoid developing vertigo but it started half way through the second tank. He did not let the vertigo stop his dive. He was usually in 10 to 15 feet of water when using scuba. Over the next three days he discovered that the vertigo was related to the size of the swell and that working deeper put off the onset. On the fifth day (Thursday) he consulted me. This time the vertigo had not settled soon after he got out of the water. It had lasted the night through and was still present in the morning. When I saw him it was possible to induce vertigo, but not nystagmus, by putting his head back although he had been out of the water for 24 hours. He said that he was much better than he had been. On questioning he admitted that he had tinnitus. On examination, neither ear drum moved. He had blood behind the right drum (grade 4 barotrauma) and the left drum looked normal except for injection of the malleus handle (grade 1 barotrauma). These appearances suggested that he was failing to equalise his ears. So he was shown how he should do it and advised to keep out of the water for a week to let the damage to his ears settle down. He was advised to clear his ears every foot on the way down and to descend fees first on all future dives. My diagnosis was either alternobaric vertigo or an inner ear window fistula.<sup>3</sup> As an inner ear fistula, which usually presents in divers with deafness and vertigo is a surgically treatable condition I referred him to a diving otolaryngologist who saw him on the Monday. His opinion was that it was alternobaric vertigo and because of the short history he advised the patient to take a week off diving. He considered that decongestants should be held in reserve as a last resort.

I next heard from him over the telephone two weeks later. He had taken a week off and then gone back to work. His vertigo had returned during the second half of the second tank and it was persisting long after he had got out of the water. He was given decongestants and more advice about the need for equalising efficiently.

He came back three weeks later still having problems. He complained of things moving when he looked at them after a dive. From the description it was nystagmus. The symptoms of nystagmus settled sooner than the vertigo which was usually present next day and had just cleared when he started his next dive. His vertigo came on during the second tank although he was clearing his ears all the time. He stated he could feel air bubbling into the back of his nose when he finished equalising. The left drum moved well but the right hardly moved when he auto inflated. Beconase was added to his medication.

He was reviewed a week later when both drums just moved. I had run out of ideas to help him so I referred him back to the ENT surgeon with the suggestion that perhaps one vestibular apparatus was more sensitive than the other and that perhaps some anti-vertigo drugs might help him.

Some months later he rang again. He had been put on Dramamine and he had returned to diving. He had "slowly come good". After two months free of symptoms he had stopped taking his pills and for a week had been symptom free. Then he was diving in a big swell and the symptoms had started again. My advice was to restart Dramamine. This has been successful and he has been able to continue diving without vertigo. Given his history of difficulty clearing his ears in his early days of snorkelling, it is likely that the vertigo that he suffered when snorkel diving in shallow water was due to inadequate equalisation. The vertigo occurring when on scuba is more probably due to him having one vestibular apparatus more sensitive to pressure changes in his ear than the other. This would explain the vertigo coming on with the swells passing over him.

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#### NEW ZEALAND CHAPTER OF SPUMS FIRST AGM AND SCIENTIFIC MEETING

To be held on 13, 14 and 15 November 1986 in Whangamata, New Zealand.

All SPUMS Members, both New Zealand and Australian, are welcome.

The Conference wilt commence on Thursday 13 November with initial Registration, followed by the first AGM of the New Zealand Chapter SPUMS. Scientific Papers will be given in the afternoon.

Conference Delegates will have the option of booking into the Cedarwood Motor Inn which has 26 fully selfcontained units and all holiday resort facilities. On the Thursday evening following cocktails a barbeque and tennis tournament will be conducted on floodlit tennis courts, weather permitting.

On Friday 14, weather permitting, a chartered boat will take divers out to Mayor Island for one of two possible dives at that location. It will be possible for people to go ashore on Mayor Island and spend the afternoon walking the island's various tracks. This locality is a mecca for big game fishing in the North Island. A late afternoon return to Whangamata will be followed by a Seafood Smorgasbord (with luck) dinner with a short scientific meeting to follow.

On Saturday morning the chartered launch departs for further dives in local dive spots, with lunch on board. Alternative activities will be made available for nondiving delegates or spouses who choose not to dive.

A free session will be made available on Saturday afternoon to be followed by further papers between 6 pm and 7.30 pm. Dinner and a Cabaret evening conclude the formal aspect of the Conference on the Saturday night leaving a further half day on the Sunday for those who want to take the opportunity for a further dive.

For further information on the programme or registration please write (airmail) to Dr MR Fraundorfer, PO Box 56, Tauranga, New Zealand.

# HELD AT THE AUSTRALIAN INSTITUTE OF MARINE SCIENCE IN APRIL 1986

# MEDICAL MANAGEMENT OF MARINE STINGS

# Vic Callanan

This paper deals with stings from Chironex fleckeri and Carukia barnesi. While other stings may be important in terms of numbers, these two produce severe consequences for the victim, and require further study. While both animals are jellyfish, the clinical picture produced by each animal is quite different and requires different management strategies. Without in any way wanting to diminish the very important role of prevention, I do not intend to discuss this aspect of management.

# **BOX JELLYFISH - Chironex fleckeri**

# THE PROBLEM

There are several separate problems due to the type of venom and the envenomating apparatus of this animal.

1. Envenomation method

The nematocyst is a very efficient method of venom delivery to the prey of the animal. It injects the venom below the integument of the prey where absorption into the general circulation is swift. If multiple nematocysts are able to inject simultaneously then the effects are even more rapid in onset. Translated to the human victim, the same process can be seen. Millions of minute venom injections are made into the skin giving a large surface area for absorption. This ensures a rapid onset of action for the venom.

2. Venom effects

The high molecular weight mixture of venom components have several effects:

- i. neurotoxic
- ii. dermatonecrotic
- iii. cardiotoxic
- iv. haemolytic
- v. immunologic
- The neurotoxic effect may be produced by i. the effect on calcium channel conductance. The effect is not a neuromuscular blockade. Whether the venom is principally toxic to the central or to the peripheral nervous system is not known. The duration of the effect may not be prolonged. The venom is quite thermolabile, being inactivated after 20 to 30 minutes at body temperature. This is consistent with the observation that neurotoxic effects are not obvious 30 minutes even after very large and potentially lethal envenomations. Expired air resuscitation (EAR) is therefore lifesaving. Return of spontaneous respiration may well occur after a short period of EAR.
- ii. Severe skin damage resulting in skin death and subsequent healing with scarring is a

feature of large stings. Early repeated doses of antivenom may be effective in decreasing the amount of skin death which occurs.

- iii. The effects on the heart of experimental animals has been well documented. Clinical cases of large envenomations who have survived support the theory that this effect is not significant in humans.
- iv. The destruction of red blood cells (haemolytic effect) is not important in the general circulation, but may play a role locally in producing skin death.
- v. The immunologic effects of this complex mixture of proteins are discussed elsewhere in this issue.
- 3. Locality of the stinging

The fact that the stings occur in the water may cause a problem if the victim is in deep water and rescuers are not immediately at hand. No deaths are known to have occurred because of this but the potential is present. Most victims get to the beach unaided, and it is here that treatment must commence if the life is to be saved. A quick response at this site is imperative.

4. Pain

Pain is the immediate symptom of a sting. The pain can be extremely severe and cause sufficient distress to prevent the victim from acting rationally. Communication may be impossible. Some venom effect on the central nervous system may be operative in this extreme case.

# THE PLAN

Management needs to be considered in two stages:

- 1. On the beach
- 2. In hospital

Obviously, not all stings need to go to hospital.

# **ON THE BEACH**

A rapid response is of the utmost importance. the order of priorities is:

- 1. CPR if necessary.
- 2. Inactivate unfired nematocysts to prevent further envenomation. This means liberal application of vinegar to all sting areas.
- 3. Decrease absorption of the venom already injected:
  - i. keep the victim still.
  - ii. apply compressive bandages in life threatening stings.

There is no evidence that compressive bandages

are effective in preventing or delaying the absorption of venom into the circulation. Snake venoms are certainly trapped at the injection site by compressive bandages. I can see no reason why compressive bandages should not work for the jellyfish envenomations. If the venom is as heat labile in vivo as is reported, then trapping it for a period of time would allow inactivation and avoid problems when the bandages are removed. The disadvantage of the bandages is that they may worsen skin death by trapping venom locally.

- 4. Neutralise absorbed venom with antivenom.
- 5. Lessen the acute pain, which is unaffected by vinegar.
- 6. Obtain medical advice and transport the patient to medical aid. The patient should be taken to hospital for all stings greater than one half of one limb in area.

# IN HOSPITAL

Principles of management.

- 1. Restore and support the circulation and respiration as necessary.
- 2. Neutralise any venom in the circulation with antivenom. Multiple doses of antivenom may be necessary.
- 3. Minimise skin death:
  - i. Antivenom appears to be partially effective if given in high doses.
  - ii. All other treatments seem to have little or no effect. Topical and parenteral steroids do not seem to work.
- 4. Treat pain.

Once in hospital, this is usually accomplished by administration of narcotic analgesics by intermittent injection or continuous infusion.

5. Treat immediate or delayed hypersensitivity reactions.

# DIRECTION OF RESEARCH

- 1. Development of a better antivenom. There are several steps in this process:
  - i. Isolate the various components of the venom in pure form.
  - ii. Test each component to ascertain its specific action.
  - iii. Use monoclonal antibody techniques to produce specific antivenoms to the individual actions of the venom.

Eventually we may have several antivenoms, each being specific for a feature of the envenomation. A mixed antivenom may still be the most practical. The volume of the injection and the chance of allergic reactions will each be reduced.

2. Animal model studies to test the effectiveness of compressive bandages is needed. These will need to be done on primates ie. monkeys.

3. A method of pain reduction which can easily be applied on the beach by lay persons is needed. This may not be lifesaving, but would reduce the distress felt by all victims of Chironex stings. We know that the antivenom is highly effective in decreasing the pain and local skin reaction, but there are problems with storage and administration, and it should not be used for minor stings. Even small stings can be very distressing for the victim. A topical application would be ideal, but it is difficult to imagine a topical agent rapidly penetrating the skin barrier to reach the nerve endings stimulated by the venom. We are no further advanced in this area than before.

# **IRUKANDJI -** Carukia barnesi

This small carybdeid jellyfish, described by Ron Southcott and Jack Barnes, and commonly known as the irukandji stinger, causes a very unpleasant syndrome. Other small jellyfish may also cause the same or a similar syndrome. Unfortunately, no study of the venom has been carried out to date.

# THE PROBLEM

1. Delay in the onset of symptoms.

Unlike the Box jellyfish, local pain from initial contact and envenomation is not marked, and the major symptoms occur after a delay of 15 to 30 minutes. This could be due to:

- i. Larger molecules
- ii. Local vasoconstriction at the sting site from venom action
- iii. Lymphatic rather than capillary absorption (do lymph nodes swell or become tender?)
- iv. Possibly more complex pharmacological/ receptor mechanisms
- 2. Pain.

The pain is generalised, prolonged, and severe. How the venom causes pain is unknown. Possibilities seem to be:

- i. Direct effect on neurones causing spontaneous depolarisation
- ii. Painful contraction of skeletal muscle bundles
- iii. Damage to muscle, either direct or ischaemic.
- 3. High blood pressure.

This feature has been documented in several cases now. The following features have also been seen in these cases: pilo-erection, sweating, restlessness, headache, cold extremities, decreased urine output.

These may all be explained if the venom causes stimulation of the sympathetic nervous system. The venom of the funnel web spider (Atrax robustus) causes severe hypertension leading to heart failure and death by an action of initiating spontaneous transmitter release from nerve terminals. In the sympathetic nervous system, such an action could cause the symptoms and signs listed above. A similar action causing acetylcholine release at neuromuscular junctions with resultant inco-ordinate muscle contractions could occur. This may produce pain in a similar fashion to that caused by the muscle relaxant, suxamethonium. No fasciculation has been described in Irukandji stings to my knowledge and so this mechanism is only a remote possibility.

# THE PLAN

# **ON THE BEACH**

- 1. Early recognition of possible Irukandji stings is important. The minor nature of both pain and skin reaction immediately following the sting makes this difficult. Once recognised, it is claimed that resting the skeletal muscle may decrease the severity of the subsequent pain.
- 2. Vinegar application is indicated to inactivate any unfired nematocysts.
- 3. Seek medical aid as soon as generalised pain commences.

# IN HOSPITAL

- 1. Pain relief by narcotic analgesics has usually been necessary. It gives good relief but needs to be maintained for up to 48 hours. A continuous infusion is the most effective mode of administration.
- 2. High blood pressure may need treatment. I would start treatment with an alpha-adrenergic blocking drug. eg. IV chlorpromazine in 5-10 mg increments. If a more powerful effect were required, I would use a direct vasodilating drug such as hydrallizine. Phentolamine has also been used.
- 3. No other effective treatment has been described.

# DIRECTION OF RESEARCH

Research will not be easy because the animals are not common, and large numbers will be needed to obtain sufficient venom for study. It is not even certain which animals to collect if a group of small jellyfish can all cause a similar clinical picture!

Once the venom is available, then all of the research so far done on the venom of Chironex fleckeri will need to be repeated on the new venom. This will take a considerable time unless our methods of catching the animals improve. Clinical documentation of all of the effects observable in victims is important. The stinger phone and associated initiatives are a move in this direction and after a few years we may have a better idea of the range of effects caused by the Irukandji stinger.

We are at the beginning of the jellyfish story as it applies to its effects on victims. The full elucidation of the story will require:

- 1. Hard work by researchers in the field and in the laboratory.
- 2. Funding for that research.
- 3. Some luck or serendipity to shorten what will otherwise be a long haul.

It may take a considerable time, but it will be well worthwhile in the end.

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Advice about jellyfish envenomation and its treatment can always be obtained via the National 24-hour tollfree Stinger Phone 008-015-160.

#### TOXIC REACTIONS TO INJURIES CAUSED BY THE SPINES OF THE CROWN OF THORNS STARFISH (ACANTHASTER PLANCI)

#### PJ Moran and J Williamson

#### SUMMARY

Information relating to the toxicity of the crown of thorns starfish, Acanthaster planci is presented. Examples are given of the different types of reactions which may occur following injury by the spines of this animal. Whilst the severity of the reactions tend to vary widely, they generally include; mild to severe pain (which may last from days to weeks), oedema, erythema enlargement of lymph glands and vomiting. The pattern of symptoms exhibited by patients may depend on the extent of the injury, the sensitivity of the patient to the toxin or toxins and the particular starfish. Responses of an allergic nature, involving extensive oedema and later itchiness, have been reported and these may cause discomfort and may last for several weeks. Present treatments are useful in relieving, over the short term, the pain experienced by some patients and preventing the onset of secondary infections. While the toxin responsible for these sometimes severe reactions has not been identified. One suggestion is that it may contain a neurotoxic component and even possess anticoagulant properties.

# INTRODUCTION

The crown of thorns starfish, Acanthaster planci, which has been recorded from many reefs throughout the Indo-Pacific region, is one of the most well known of tropical marine animals. Unlike animals such as the box jellyfish, Chironex fleckeri its notoriety does not stem from its ability to cause injury and the death of humans but rather its propensity to occur in large aggregations (commonly called outbreaks) which can cause the death of extensive areas of coral.<sup>1</sup> However, it is generally not known (particularly by the public) that the spines of this starfish are capable of producing a toxic reaction(s) which may be worrying to victims.

Acanthaster planci was first reported in the scientific literature by Rumphius in 1705<sup>2</sup> and classified by Linnaeus in 1758.<sup>3</sup> Several papers around that time made reference to the toxic nature of this starfish. Madsen,<sup>4</sup> in reviewing these sometimes conflicting reports, concluded that it was not known whether the animal was itself toxic as such wounds may have become infected. Concurrent with the first documented outbreaks in the late 1950's and early 1960's, further reports of the toxic nature of Acanthaster planci came to hand. In general, these reports reinforced the view that specimens of Acanthaster planci were capable of not only inflicting

a painful wound but of having a toxic effect on victims.  $^{\rm 5,6,7}$ 

The aim of this paper is to draw on more recent information to discuss several aspects of the toxicity of Acanthaster planci. These include descriptions of: its morphology and habitat (directed towards defining the potential danger that this animal poses to people who use the Great Barrier Reef); its spines; its toxicity; the symptoms experienced by victims; treatment of patients. To date these topics have not been extensively discussed elsewhere and a knowledge of them may be helpful in treating both the physical and emotional needs of victims.

# MORPHOLOGY AND HABITAT

Acanthaster planci (Figure 1) is a carnivorous starfish which has been reported on reefs only in the Indo-Pacific region. At the beginnings of outbreaks individuals normally range in size from 250 to 350 mm in diameter,<sup>8</sup> although starfish up to 700 mm in diameter have been reported.<sup>9</sup> This makes it one of the biggest, and potentially most dangerous, starfish in the world.

It is multi-coloured and individuals may vary from purplish blue with red tipped spines through deep red with orange tipped spines, which is the normal colour variation on the Great Barrier Reef, to green with yellow tipped spines.<sup>10</sup> Adults normally possess from 9 to 23 arms, or rays, which may be up to 150 mm in length.

Studies on the distribution of this animal have indicated that adult specimens prefer more sheltered environments such as lagoons and leeward slopes, and deeper water, more than 3 m, on the windward slopes of reefs.<sup>9</sup> During outbreaks they threaten the well-being of both recreational and professional divers. As large numbers of adults, and more recently juveniles, have been reported periodically in shallow water, generally when the weather conditions are calm, they constitute a danger to people, eg. tourists walking on the reef. They may also be a hazard occasionally to fishermen as individual starfish have been dredged up in nets from deep water between reefs.<sup>9</sup>

# SPINES

Acanthaster planci is easily recognised in the field by the plethora of large, sharp spines which cover much of its body (Figure 1). There are about 6 different types of spines on the aboral (upper surface) and oral (lower surface) surfaces of this starfish and these have been described by Caso.<sup>11</sup> Studies using a scanning electron microscope have revealed that each spine is composed of a single crystal of magnesium calcite, of porous structure, which makes it relatively strong but light in weight.<sup>12</sup> Those projecting from the



Figure 1. The crown of thorns starfish on recently killed coral (white area).

upper surface of the starfish, are the largest and may be up to 40 to 50 mm in length. All the spines are articulated at the base (diameter 1 to 2 mm) and are extremely sharp, their tips have three raised edges which assist in "cutting" through tissue.<sup>12</sup> Therefore the spines are able to penetrate wet suits, heavy gloves and other underwater protective clothing. Extreme care should be taken whenever handling, or in close proximity to a specimen of Acanthaster planci. Figures 2, 3 and 4 show a recent injury from a crown of thorns starfish spine.

# TOXICITY

Contrary to general opinion, there is no evidence to suggest that a toxin or venom is actively injected into a victim after being wounded by a spine.<sup>13</sup> A number of potentially toxic compounds have been isolated from the tissue overlying the spines.<sup>14,15</sup> All form part of the group of compounds termed saponins which are derived from steroids and are known to be toxic to a variety of marine organisms.13 At present it is not known whether the symptoms shown by patients are a direct result of these compounds. Studies concerned with isolating and identifying such chemicals have indicated that they may be present in insufficient quantities to cause these sometimes severe reactions.<sup>14</sup> Despite not knowing what causes these reactions, it has been suggested that the toxin or toxins would probably act in a neurotoxic manner,<sup>16</sup> although this has not been confirmed as yet. Results from other studies have indicated that reactions to injuries from Acanthaster planci may be the result of a series of different processes. For example, crude extracts of material isolated from the surface of spines have been demonstrated to have a haemolytic effect on human red blood cells.<sup>17</sup> In addition, other studies have indicated that the inflammation that may occur around a wound may be mediated by the activities of histamine-like compounds.<sup>17</sup> Given the copious flow of blood which may come from the relatively small wounds produced by spines, it would appear that the toxin involved also has anti-coagulant properties.<sup>16</sup>

# SYMPTOMS

There are now a number of reports<sup>4</sup> concerning the injuries sustained by people who have come in contact with a crown of thorns starfish. These date back to 1705. Two conclusions can be drawn on the information now available. Firstly, the majority of injuries occur on the fingers, hands and feet of individuals. Secondly it would appear that while this starfish is indeed toxic the reactions of victims may be quite variable.

The first, and often most severe, symptom recorded by victims who have been injured by a spine is pain, which is usually felt immediately after the injury. Reports suggest that the pain can be very intense and may be likened to a burning sensation.<sup>16</sup> This, often throbbing, sensation may last for several hours after which it fades to a dull ache. Multiple punctures or wounds into joints may prolong this period of more intense pain. Injuries experienced by PJM to the side of a knee and to the tip of a finger were still very tender and uncomfortable (Figures 2, 3 and 4) for about a month after they occurred.

Severe injuries involving multiple punctures may result, not only in intense pain, but also nausea and protracted

vomiting. The latter may continue for several days. In such cases, localised suppuration may occur.

Reports also indicate that the wound turns a purplish blue colour soon after an injury has been sustained. Often this grows larger and is surrounded by an area of tissue which is light red in colour.<sup>18</sup> This region of erythema may persist for several days if not weeks. This may cause some victims to think that a portion of spine has remained in the wound. In addition to erythema, some victims may suffer from extensive oedema. Multiple punctures to a hand or foot may result in oedema occurring over the entire surface. This condition may last for up to a week during which time the wounds may leak serous fluid, numbness of the extremities may be felt<sup>16</sup> and swelling of the lymphatic glands may occur.<sup>8</sup> In such cases, victims



Figure 2. Injury to finger from crown of thorns starfish spine showing bleeding entry wound on pulp.



Figure 3. Injury to finger from crown of thorns starfish spine (side view).

Figure 4. Injury to finger from crown of thorns starfish spine seen from above.

also may find it difficult to move the injured limb and the area around the wound may become extremely itchy whilst the oedema persists. These symptoms, experienced by PJM, suggest that certain susceptible victims respond in an allergic manner to injuries from the crown of thorns starfish. Odom and Fischelmann<sup>16</sup> have suggested that there was even the potential for anaphylactic reaction in susceptible persons.

It should be emphasised that victims may differ quite widely in their reactions to injuries from crown of thorns starfish. This is probably for a number of reasons. Firstly, it depends on the site and extent of the injury that is sustained. Secondly victims appear so display differing sensitivities to the toxin or toxins present in the starfish. Finally, the animal itself may differ in its ability so produce a toxic reaction in victims. For example, observations indicate that injuries from small starfish are not nearly as painful or uncomfortable as those from much larger specimens.

# TREATMENT

Despite the fact that injuries caused by the crown of thorns starfish may be distressing there are no treatments presently available (ie. specific antivenoms) which are effective in neutralising the symptoms experienced by a victim. The most useful measures have been listed recently by Williamson<sup>19</sup> and are the same as those used to treat stonefish injuries. The experience of PJM is that submerging the wounded area in hot water (or cold water) somewhat relieves pain for short periods of time (up to an hour). Application of antihistamine creams or similar topical drugs did not appear to alleviate any of the symptoms (particularly oedema and itchiness). Longer term relief of pain may be obtained using more potent analgesics. As the starfish produces a mucus over its exterior, it is necessary to wash the wound carefully and to check whether any fragments of spine are still embedded in it. A xeroradiograph of the injury should be performed if it is suspected that spine fragments have been retained in the wound. These treatments are necessary to reduce the occurrence of secondary infections. Apart from treating the physical symptoms apparent in patients it is also important to alleviate their concern, given that the reactions may be severe and occur over relatively long periods of time.

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#### IMMUNOLOGY AND JELLYFISH VENOMS

# John Williamson

Jellyfish envenomation is a worldwide occurrence, producing effects ranging from mild local skin irritation  $^1$  to rapid endotoxic  $^2$  or less commonly, anaphylactic death.  $^3$ 

# **TOXIC REACTIONS**

Jellyfish venoms are complex mixtures of polypeptides and enzymes, structurally akin to the venoms of snakes, insects and spiders, that are pathogenic to man by toxic or antigenic mechanisms. That the principal clinical reactions to jellyfish envenomation appear to be toxic, rather than allergic, is suggested by the following evidence:

- (a) no immune resistance has so far been reported
- (b) repeated stings cause no difference in symptoms
- (c) venom injections into different mammals induce similar clinical results

Most human toxic reactions are also characterised by a direct correlation between the total venom dose (ie. size of the sting) and the severity of the clinical effects. Such toxic reactions include:

immediate skin pain with tentacle contact acute inflammatory skin reactions, up to actual skin death and scarring localised and regional tissue oedema regional lymphadenopathy muscle pains vomiting breathing distress impairment of consciousness

#### respiratory failure

Other severe documented effects that have been labelled as toxic are:

gangrene haemolysis renal failure

myocardial failure

Modern immunological research techniques are beautifully applicable to the study of both the toxic and allergic mechanisms of jellyfish venoms. The techniques include:

skin biopsy

immunofluorescence

radioallergosorbent test (RAST)

- enzyme-linked immunosorbent assay (ELISA)
- immunochromatography using monoclonal antibodies

Using such techniques, the following information has been revealed to date:

- 1. Partially purified extracts from the venoms of Hair Jelly (Cyanea capillata), Portuguese Man-O'War (Physalia physalis), Sea Nettle (Chrysaora quinquecirrha) and the Northern Australian Box jellyfish (Chironex fleckeri) have demonstrated cardiotoxicity to spontaneously beating cultured chick cardiocytes.<sup>4,5</sup>
- 2. The cardiotoxic components of venoms from Physalia physalis, Chrysaora quinquecirrha and Chironex fleckeri have their actions significantly intercepted by the presence of the calcium antagonist verapamil.<sup>6,7</sup>
- The composition of some jellyfish venoms may alter with the climactic seasons of a single year.<sup>8,9</sup>
- 4. Several common world jellyfish venoms (Chrysaora, Physalia and Pelagia noctiluca (Mauve Stinger)) generate antibodies in humans that exhibit some cross-reactivity; further, it appears that the venoms of Chrysaora and Physalia, the brown recluse spider (Loxosceles reclusa), and purified cholera toxin all share common antigenic sites.<sup>9,10</sup>

# ALLERGIC REACTIONS

It is of interest that the scientific study of allergic disease actually began with the use of coelenterate protein,<sup>11</sup> when Pertier and Richer in 1902 induced anaphylaxis in dogs with injections of coelenterate venoms. Despite this early clue, over the ensuing decades repeated documentation and apparent contradiction occurred concerning so-called "harmless" jellyfish stings from different parts of the world, and the "unexpected" systemic nature, severity, and/or persistence of the clinical effects. It was really only with the demonstration in the serum of stung patients, of immune-specific and cross-reacting antibodies to Chrysaora and Physalia venoms taken from the Atlantic Ocean, in 1980, 1981 and 1983,<sup>12,13,14</sup> that the allergenic potential of jellyfish venoms was given

scientific objectivity. Finally, the rare but potentially fatal anaphylactic reaction to a jellyfish sting (probably Pelagia noctiluca from the Eastern Mediterranean Sea) was first adequately documented in 1985.<sup>3</sup>

In common with many other allergen sources, jellyfish venoms can be described as multi-allergenic systems, containing several of perhaps many allergenic macromolecules. It is now clear that the visible, in particular cutaneous, response to jellyfish envenomation may on occasions be allergic in nature. The differential diagnosis of such a response from the more toxic one is necessary for its effective management.

Allergic reactions to jellyfish stings may vary widely in their clinical presentation:

Cutaneous Reactions (erythema and/or urticaria)

may be

local or generalised, immediate or delayed (days to weeks), persistent or recurrent, occurring at the sting site, or at sites distant from the primary sting.

They may be with or without

exaggerated local oedema itching, clear watery discharge vesicle formation.

<u>Generalised Hypersensitivity</u> (extracutaneous and/or anaphylactic)

include

fever, sweating, chills, diarrhoea, nausea, tachycardia, hypotension, difficulty with respiration, loss of consciousness.

Some clues have been obtained from studies, so far confined to either venom extracts or human envenomation from American Sea Nettle (Chrysaora quinquecirrha), the Atlantic Portuguese Man-O' war (Physalia physalis), the Eastern Mediterranean Mauve Stinger (Pelagia noctiluca), and the Cabbagehead Jellyfish (Stomolophus meleagris),<sup>10</sup> of patients who have exhibited allergic reactions of either a cutaneous, extracutaneous or anaphylactoid nature to jellyfish envenomation, and whose sera have been examined.

- A. Both specific and cross-reacting IgG and IgE antibodies to these venoms have been detected in the patients' sera.<sup>15</sup>
- B. The sera from patients exhibiting the more severe reactions contain higher specific IgG and IgE antibody levels.<sup>14</sup>
- C. Detectable antibody levels were not influenced by the number of stings sustained by the individual.<sup>14</sup>

- D. Elevated levels of these immunoglobulin antibodies may persist for up to 5 years, at least.<sup>15</sup>
- E. Increased histamine release from venom-challenged basephil cells taken from a seriously sensitised patient, has been documented. There was strong evidence that this was specific IgE-antibody mediated.<sup>3</sup>
- F. Recent work supports the hypotheses that T cell function may be important in the pathogenesis of at least cutaneous lesions, following coelenterate envenomation.<sup>16</sup>

In this regard it is noteworthy that experimentally purified, so-called 'lethal', extracts of several jellyfish venoms are more powerfully antigenic, and curiously no case of serum sickness after jellyfish envenomation, either by natural tentacle contact or by ingestion, has been reported to date.<sup>17</sup>

As has been pointed out in the literature previously,<sup>3,18</sup> beta-adrenergic blocking drugs have the potential to increase the severity of an anaphylactic reaction; this is pertinent in view of the current widespread use of such drugs.

# FUTURE DEVELOPMENTS

Further similar analysis of Chironex venom, the introduction into this research approach of the venoms of Chiropsalmus quadrigatus, Carukia barnesi (Irukandji ), and other Carybdeid medusae, and the study of the skin, sera and blood collected from selected patients in this (and other) countries affected by a jellyfish problem, can be expected to shed further light on the subject. The tools to study the nature and prevalence of both toxic and allergic jellyfish sting reactions are now available and on theoretical grounds at least, the development of preventative immunotherapy is entirely feasible. There is strong indication for such an approach, given the ubiquitous and expanding association of man with the sea.

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# THE MANAGEMENT OF STINGS BY JELLYFISH, OTHER THAN CHIRONEX

#### Peter Fenner

The term cubomedusan, simply translated, means box shaped jellyfish. There are so many box shaped jellyfishes that the deadly North Australian box jellyfish should always be referred to by its generic name of Chironex. This would then save any confusion from the terms box jellyfish or stingers, so loosely used by North Queenslanders for the deadly Chironex fleckeri. There have been recent problems with the Irukandji (Carukia barnesi) and the Morbakka, both of which are box jellyfish.

All types of jellyfish are derived from the Phylum Coelenterata and the biological classification for cubomedusans discussed in this article is presented on the next page.

Cubomedusans can also be simply classified as those with only four tentacles, ie. having only one tentacle per pedalium (corner) and those with many tentacles at each corner.

# **PREVENTION OF MARINE STINGS**

The wearing of protective clothing helps to give protection against any sting. The new and popular "stinger suits" are ideally suited as the thickness prevents the thread tube, through which the venom is injected from the nematocyst, or stinging capsule, coming close enough to the skin to be able to puncture it to sufficient depth to reach underlying blood or lymph vessels.<sup>1</sup>

In the same way two pairs of pantihose, as used to be worn by northern lifesavers is just as effective although perhaps not as aesthetic! One pair is used normally, with the feet cut out (otherwise they fill with sand!) and the others are worn upside down with a head hole cut in the crutch and the arms fit into the legs of the pantihose.

# TREATMENT OF MARINE STINGS

Vinegar has been proven to prevent further firing of nematocysts on remaining adherent tentacles of most species, particularly the life-threatening ones such as Chironex.<sup>2</sup> It does not inactivate the nematocysts of Cyanea (hair jelly) or Chrysaora (Sea nettle which is found in the USA). As Pelagia (the little mauve stinger) is similar to these two species, vinegar may well be of little use in the treatment of its stings also.

Note that vinegar, when used as treatment for Chironex fleckeri, the North Australian box jellyfish, or any other painful jellyfish sting <u>DOES NOT HELP PAIN</u>. It only prevents more nematocysts firing off and so possibly causing further envenomation.<sup>2</sup>

# FOUR TENTACLED CUBOMEDUSANS

#### **IRUKANDJI** (Carukia barnesi)

This stinger is very much in the news recently as there have been many reported cases, far more than usual.

Dr John Williamson's address is MSO Box 5695, Townsville QLD 4810, Australia.

#### CLASS CUBOZOA (Order Cubomedusae)

FAMILY Chirodropidae

Genus

Chironex Chiropsalmus

<u>Species</u> Chironex fleckeri North Australian box jellyfish

Chiropsalmus Quadrigatus

North Australian and South East Asian

It is a very nasty little jellyfish! Or it may be a syndrome, ie. a number of similar symptoms, caused by a number of different types of jellyfish, presumably all cubomedusans!

Usually the bell is only 2 cm diameter and 2.5 cm deep although the tentacles may extend out to 65 cm! It is probably no more common than usual and the recent large number of stings may be due to two factors. The first is the normal periodicity. In some years they are much more common than usual and this year may have been one of those years. Many factors may influence this although one of the more likely ones is the climatic conditions. The second factor is the construction of stinger resistant enclosures. Normally people will not swim in the North in the summer months. However with the advent of the stinger resistant enclosure they were able to do with relative safety from the large and lethal Chironex, and so many people started swimming, only to be faced with stings from the smaller Irukandji. Presumably the Irukandji is small enough to get through the protective mesh on the outside of the enclosure.

# Sting and symptoms

The initial sting may often be disregarded as being a minor skin irritation although some are described as being a somewhat painful sting. Classically the symptoms start with backache about 20-40 minutes after the sting. This then usually progresses to severe backache, with muscle and joint pains and maybe abdominal pains and chest pains. In the past has been mistaken for a heart attack and appendicitis!<sup>3</sup> Severe headache is often present along with severe nausea and vomiting. Patients often feel they "want to die". As the late Dr Jack Barnes once said, "I'd rather be stung by a small Chironex than an Irukandji.

The victim may be very restless, have localized sweating, particularly over the painful muscles and localized pilo-erection. They may have cold extremities, a decreased urine output and some also develop hypertension and/or cardiac arrhythmias.<sup>4,5</sup> As yet there have been no reported fatalities from an Irukandji

FAMILY Carybdeidae

Genus

Carybdea Tamoya Morbakka? Carukia

<u>Species</u> Carukia rastoni Jimble

Tamoya haplonema

Carukia barnesi Irukandji Morbakka

sting but there is a potential problem in the patient who is elderly or who may have heart problems and they should be assessed carefully.

#### Treatment

As the initial sting takes place some time before the patient usually presents there are usually no nematocysts left on the skin and so treatment with vinegar is of little benefit. Some patients have very little reaction and only require re-assuring. Others may need admission to hospital for intramuscular analgesia or anti-emetics. Treatment may be needed to lower blood pressure in some patients.<sup>5</sup>

There is reason to believe that immediate rest after the sting may produce far fewer and less severe symptoms although this has not yet been proven in practice. However, because the sting is often mild, the patient often continues to exercise.

My personal experience has shown that anti-histamines, and cortisone injections do not help at all. However a trial of Chironex fleckeri anti-venom gave pain relief within 2 minutes, and this lasted for up to 30 minutes before pains started to become severe again. Intravenous and intramuscular pethidine had the effect of stopping the severe pain but the patient was still aware of a discomfort coming on in waves for several hours later.

The use of Chironex fleckeri antivenom is not recommended as it produces only a transient effect. It is also too expensive for routine use, costing \$50 per ampoule), and it has the potential for anaphylaxis, although it must be stressed that no case of reaction has been seen as yet after many years and several hundred ampoules used. Furthermore pethidine is extremely effective in pain relief although very high doses need to be used at times, often necessitating the use of a drip to control the dose to achieve pain relief without side effects. I suggest 25-30 mg per hour after the initial dose which is approximately 50 mg intravenously for an adult.<sup>5,6</sup>

All the above symptoms seem to be related to the excessive release of catecholamines and this led to the recent use of phentolamine, an alpha adrenergic blocker. A paper detailing the use of these treatments has recently been prepared for publication and further details should be available later.<sup>5</sup>

# MORBAKKA

Previously referred to as the Moreton Bay stinger, or the fire jelly. Also wrongly classified until recently as Tamoya haplonema.<sup>7,8</sup> It looks similar to Irukandji except that it is much larger. The bell may measure up to 12 cm across and 16 cm deep and the tentacles, one in each corner, may be up to 1 metre in length and as thick as the flat 3 core electric cable used by electricians wiring houses. The bell is covered with pinkish looking mammilations which are clumps of nematocysts and so envenomation can also occur by handling the bell.<sup>8</sup> Fortunately the symptoms are not as bad as those of Irukandji despite the much larger size of the Morbakka. However the number of recorded stings is low with little documentation and more symptoms may be confirmed in the future.

# Sting and symptoms

Called the fire jelly the Morbakka lives up to its name. The area touched by tentacles is covered with raised white wheals with a bright red erythematous flare. The skin looks as if it is on fire and the sting area is very itchy and burns. Other symptoms include a cough, lump in the throat and mild backache.

# Treatment

Tentacles and nematocysts are inactivated by vinegar within 30 seconds. Usually pain is relieved by simple pain-killers such as paracetamol and further treatment is unnecessary. However there are unconfirmed reports of respiratory depression and marked prostration and so the victim should be carefully monitored.

# JIMBLE (Carybdea rastoni)

Looks like a small Chironex except it has only the single tentacle in each corner. It may be up to 3.5 cm diameter across the bell and the tentacles may extend to 30 cm.

# Sting and symptoms

It produces a painful welt with no general effects.<sup>1,9</sup>

#### Treatment

Vinegar neutralizes the tentacles although they are not usually adherent. Ice is the best for the relief of pain. There have been no reported systemic effects. The weal may sometimes break down in the centre a week or so later and these ulcers occasionally become infected.

# MULTI-TENTACULAR CUBOMEDUSANS

# CHIROPSALMUS QUADRIGATUS

The North Australian variety looks fairly similar to Chironex except that it is smaller, has finer more thread-like tentacles which are usually fewer in number than Chironex of a similar size, and the pedalium, the corner of the box from which the tentacles arise, has a different configuration in the central canal. Inside the bell are smaller, more rounded gonads.<sup>11</sup> Probably it is not lethal but it causes severe localized pain and weals that look similar to Chironex.

The South East Asian variety is very similar to Chironex in size and appearance. It causes deaths in the Philippines, up to 30-50 per year, and probably elsewhere in South East Asia (reports from trips by Fenner and Williamson to the area in September 1985).

Differentiation between this species and Chironex is even more difficult and probably only noticeable to the trained observer. From personal observation they include the finger like gonads compared to the grapelike configuration of the gonads of Chironex, and the more gentle hook-shape central canal, more like a volcano, present in the pedalium.<sup>10</sup>

# UNKNOWN CUBOMEDUSANS

We do not know of all types of box jellyfish. There are probably many others and in the last few years we have had reported a couple of new ones about which nothing is known. One is a cubomedusan where the pedalium is divided into two with tentacles off each part. It was found in a juvenile form by Phil Alderslade, Director of Coelenterates of the Northern Territory Museum in June 1985. The other looks similar to the Irukandji (Carukia barnesi) or the Jimble (Carybdea rastoni) and was discovered by Bob Hartwick several years ago in Alma Bay, Magnetic island. It attaches to structures with a suction cap and has been responsible for several stings to divers although no systemic effects have been reported.

# **OTHER COMMON JELLYFISH**

# HAIR JELLYFISH (Cyanea capillata)

This is the world's most common jellyfish which may grow up to 2 metres diameter in the Arctic.<sup>1,8,11</sup> Fortunately it is much smaller in Australian waters although commonly specimens are found up to 35 cm in diameter. There were many around in north Queensland in the 1985/86 summer season. Described as a "mop hiding under a dinner plate" they have a centre cone of thick tentacles underneath the "blubber" and many fine tentacles trailing behind for several metres. They have a characteristic fishy smell from mucous produced by the tentacles.

#### Sting and symptoms

A florid sting with many white weals surrounded by a vivid red flare which fortunately looks worse than it actually is! After the initial fear associated with the sting and then the look of the skin the patient actually only complains of a mild local discomfort and has no real systemic symptoms.

# Treatment

Usually there are no adherent tentacles but if any are present they should be washed off with sea water as recently vinegar has been shown to cause the nematocysts of this species to discharge. However this is of little practical importance as the sting is not serious.<sup>12</sup> No other treatment is usually needed although ice applied locally may help the irritation.

# LITTLE MAUVE STINGER (Pelagia noctiluca)

This is common worldwide but seems to be rare these days in north Queensland waters although there have been reports of two possible cases from Hamilton Island recently.

# Sting and symptoms

The sting causes an irregular shaped weal looking like an allergic rash. There is often intense local pain at the site of the sting, and there may also be a cough and occasionally pain on breathing.<sup>9</sup>

# Treatment

Normally there is no real danger to life. However an allergic reaction with near death has been reported from the eastern Mediterranean where these stings are common.<sup>13</sup> Ice should help the pain. The victim should be watched for signs of breathing difficulty. Vinegar is of doubtful value.

# **BLUBBER** (Catostylus mosaicus)

This is fairly common around Australia. It has no actual tentacles. It has been described as "a mushroom wearing frilly knickers" and the "frills" sting!

# Sting and symptoms

A mild sting with pins and needles only.

# Treatment

The best treatment is locally applied ice.

# **BLUEBOTTLE** (Physalia physalis)

This is not a true jellyfish (Schyphosan), but is a hydrozoan and is really a collection of animals rather than a single one.<sup>1</sup> One of the animals forms the floating air sac which is usually 3 to 6 cm in diameter but may grow to 15 cm. There are many intensely blue tentacles underneath and usually one of these is very long being up to several metres long. However there may be another species which has many long tentacles. Like everything else in the jellyfish world at present the taxonomy is in a state of flux.

# Sting and symptoms

Usually these are lines of white, separate, weals with a surrounding red flare. There is quite marked localized pain and itching. Often there is quite a marked pain in the lymph glands draining the stung area.

Large stings may cause nausea and vomiting, drowsiness and occasional pain on inspiration.<sup>9</sup>

# Treatment

Use vinegar to neutralize the nematocysts and then the tentacle can be removed. Local ice will reduce the pain of the sting and simple analgesics and rest may be necessary to help the pain in the glands.

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For further information on this subject with colour illustrations of the jellyfish and their stings discussed in this article, the reader is referred to "The Marine Stinger Book" by Dr John Williamson. This is available from the distributors:

Queensland State Centre of Surf Life Saving PO Box 36 NEWSTEAD QLD 4006 Australia

# OBJECTS OF THE SOCIETY

To promote and facilitate the study of all aspects of underwater and hyperbaric medicine.

To provide information on underwater and hyperbaric medicine.

To publish a journal.

To convene members of the Society annually at a scientific conference.

#### MEMBERSHIP

Membership is open to medical practitioners and those engaged in research in underwater medicine and related subjects. Associate membership is open to all those, who are not medical practitioners, who are interested in the aims of the society.

The subscription for Full Members is \$A35.00 and for Associate Members is \$A25.00. New Zealand members' subscriptions (\$NZ 45.00 and \$NZ 30.00 respectively) should be sent to Dr P Chapman-Smith, Secretary/Treasurer of the New Zealand Chapter of SPUMS, 67 Maunu Road, Whangerei.

Membership entitles attendance at the Annual Scientific Conferences and receipt of the Journal.

Anyone interested in joining SPUMS should write to the Secretary of SPUMS,

Dr David Davies Suite 6, Killowen House St Anne's Hospital Ellesmere Road MT LAWLEY WA 6050

# DIPLOMA OF DIVING AND HYPERBARIC MEDICINE

The requirements for the Diploma of Diving and Hyperbaric Medicine are

- 1. To have completed both the introductory course and the advanced course in Underwater Medicine at the Royal Australian Navy School of Underwater Medicine.
- 2. To have completed the course in Hyperbaric Medicine at the Prince Henry Hospital, Little Bay, Sydney, New South Wales.
- 3. To have completed six months full time, or equivalent part time, employment in diving or hyperbaric medicine.
- 4. To present a satisfactory thesis (suitable for publication, usually in the SPUMS Journal) for consideration.

The decision to award the Diploma lies with the Diploma Committee which is comprised of the President of SPUMS, the Officer in Charge of the Royal Australian Navy School of Underwater Medicine and the Director of the Hyperbaric Unit at Prince Henry Hospital.

Applications should be directed to the Secretary of SPUMS,

Dr David Davies Suite 6, Killowen House St Anne's Hospital Ellesmere Road MT LAWLEY WA 6050

# SPUMS JOURNAL

# Instructions to Authors

Contributions should be typed in double spacing, with wide margins, on one side of the paper. Figures, graphs and photographs should be on separate sheets of paper, clearly marked with the appropriate figure numbers and captions. Figures and graphs should be in a form suitable for direct photographic reproduction. Photographs should be glossy black and white prints at least 150 mm by 200 mm. The author's name and address should accompany any contribution even if it is not for publication.

The preferred format for contributions is the Vancouver style (Br Med J 1982; 284: 1766-1770 [12th June]). In this Uniform Requirements for Manuscripts Submitted to Biomedical Journals references appear in the text as superscript numbers.<sup>1-2</sup> The references are numbered in order of quoting. The format of references at the end of the paper is that used by The Lancet, The British Medical Journal and The Medical Journal of Australia. Page numbers should be inclusive. Examples of the format for journals and books are given below.

- 1 Anderson T. RAN medical officers' training. SPUMS J 1985; 15(2): 19-22.
- 2 Lippmann J, Bugg S. The diving emergency handbook. Melbourne: JL Publications, 1985.

Abbreviations do not mean the same to all readers. To avoid confusion they should only be used after they have appeared in brackets after the complete expression, eg. decompression sickness (DCS) can thereafter be referred to as DCS.

Measurements should be in SI units. Non-SI measurements can follow in brackets if desired.

# **Reprinting of Articles**

Permission to reprint original articles will be granted by the Editor, whose address appears on page 2, provided that an acknowledgment giving the original date of publication in the SPUMS Journal is printed with the article.

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For further information write to SPUMS 80 Wellington Parade EAST MELBOURNE VIC 3002 Australia

# MINUTES OF THE COMMITTEE MEETING

Held on Saturday, 22 February 1986 at 80 Wellington Parade, East Melbourne.

# Present

Drs C Acott (President), D Davies (Secretary), G Barry (Treasurer), D Walker (Editor), C Lourey (Past President), J Knight, A Sutherland, J Williamson and Mr A Newly (Allways Travel).

# Apologies

Nil.

# Minutes of the last meeting

10 August 1985, Naval and Military Club, Melbourne. Minutes read and confirmed.

# **Business arising**

1. Liaison between UMS and SPUMS should be encouraged. The Secretary to write to the new Secretary of UMS to obtain early information about its activities so these can be published,

2. The Committee noted that several organizations are proposing to train doctors in diving medicine. A sub-committee of three, Drs Knight, Lourey and Williamson will investigate the possibility of SPUMS taking on this role.

3. The Committee supports the concept of SPUMS endorsed seminars for the sports diving community. The aim is both education of divers and increasing the profile of SPUMS as an active organization.

4. A regional branch, the New Zealand Chapter, has been formed with over 90 enthusiastic members. A successful meeting has been held at Great Barrier island with Dr J Knight as Guest Speaker. The Committee recommended that there be a permanent place on the Committee for a delegate from New Zealand.

5. The Treasurer reported a sound financial position though money was being held in several accounts. It was agreed to amalgamate all these into one account under his sole control.

6. Organization of the AGM in Moorea is well under control with about 95 Australian delegates attending plus an unknown number from New Zealand.

7. Various aspects of the SPUMS Journal were discussed. The Committee agreed that in an attempt to defray cost, advertising would be acceptable. Dr Williamson suggested that a more formal format be considered. The Editor reported there is still an inadequate contribution from members.

# **New business**

1. Dr Williamson reported that the Townsville Hospital Emergency Centre now had a toll free number for advice on marine biological injuries. This could be used either for emergencies or for routine enquiries and reports.

2. A copy of the proforma of the database for investigation of diving accidents was shown to the Committee. This will be used as the basis for putting the information onto the National Safety Council computer. The Committee agreed that divers are reluctant to fill in forms, especially after an accident and there are a great many omissions, often of significant information.

3. Dr Williamson spoke on the proposed publication by the AUF of a booklet on Diving for the Disabled. The subject was discussed and the Secretary instructed to contact the AUF to convey the Committee's reservations on the advisability of publication.

# Meeting closed

16.15 hours

# Next meeting

The next meeting will be held in conjunction with the Annual Scientific Meeting in Moorea.

# MINUTES OF THE ANNUAL GENERAL MEETING

Held on Thursday 5 June 1986, at the Hotel Ibis, Moorea.

# Chairman

Dr C Lourey, Immediate Past President

# Attendance

All members attending the Scientific Meeting

# Apologies

C Acott, A Bridger, W Hurst, J Mannerheim, D Walker, J Williamson.

# Minutes of the previous meeting

These had been published and distributed to all members.

Moved J Knight, seconded P McCartney. Confirmed.

# **Business arising**

Nil

# Correspondence

Nil

# **Treasurer's Report**

The Treasurer tabled figures that showed the Funds of the Society were being depleted. The unaudited figures showed that \$11,369.77 had been spent in

the last year and that funds in hand were \$8,799,21. There were, however, some expenses of a nonrepetitive nature, but he suggested that the Committee needed to consider a lift in membership fees.

Moved D Brownbill, seconded J Knight. Confirmed.

# **President's Report**

In the absence of the President this was read by the Secretary.

# Secretary's Report

Membership of the Society is increasing at the rate of about 20 people per month. There are currently 500 Australians, 110 New Zealanders and 100 overseas members.

The Society is negotiating to obtain representation on the committee to discuss standards on Safety for Sports Divers.

A call for nominations for the committee will be made to all members shortly after returning to Australia.

# **Editors Report**

Dr Knight called for more contributions from members. The change in colour of the cover is an improvement but is too dark at the moment and will be altered with the next edition.

# Meeting closed

17.30 hours

# SPUMS ANNUAL SCIENTIFIC MEETING 1987

The Annual Scientific Meeting will be held during the first week in June at Honiara, Solomon Islands.

# CALL FOR PAPERS

All members wishing to present a paper should write to the Convenor of the Scientific Meeting:

Dr David Davies Suite 6, Killowen House St Anne's Hospital Ellesmere Road MT LAWLEY WA 6050

They should specify the topic and the ancillary equipment (overhead projector, 35 mm slide projector etc.) that they require and the expected duration of the presentation.

Thank you for all attending. I am sorry that Judy and I are not with you this year but our family commitment has changed in the past six months. The three of us will be attending next year.

At the last committee meeting we decided to "advertise" SPUMS more with the diving community. Therefore SPUMS will be sponsoring a diving seminar on diving safety to be held in Rockhampton on 11 and 12 October 1986. It will be called "ROCKDIVE" and if it is a success we hope to repeat similar seminars in other states.

We are in the preliminary stages of planning our own course in underwater medicine. More details of this will be announced later.

The New Zealand Chapter is flourishing due to the excellent work of Alan Sutherland and others in New Zealand. I welcome all our new members from New Zealand here today.

You may have noticed that the format of the Journal is undergoing a slow change. We are looking for original articles to publish, so why not put pen to paper? Hopefully, we will be able to publish seminar issues when the material is available. Our thanks are due to Douglas Walker and John Knight for their work in producing the Journal.

Again I apologise for not being with you this year, but I look forward to seeing you next year.

Chris Acott

#### CONSTITUTIONAL CHANGE

The Secretary has received no objections to the proposal that the Chairman of the New Zealand Chapter of SPUMS be an ex-officio member of the Executive Committee (SPUMS J. 1986; 16(2): 61).

The proposal is therefore deemed to have been carried and the constitution will be altered accordingly.

#### ELECTION OF EXECUTIVE COMMITTEE

When nominations closed the Secretary had received the following nominations:

President	Dr Chris Acott
Secretary	Dr David Davies
Treasurer	Dr Grahame Barry
Editor	Dr Douglas Walker
Committee	Dr John Knight Dr Peter McCartney Dr John Williamson

As there are the same number of candidates and vacancies the above are declared elected to the positions nominated.

These gentlemen will be joined on the Executive Committee by the Past President, Dr Chris Lourey and the Chairman of the New Zealand Chapter, Dr Allan Sutherland. Some copies of a few past issues are available at \$2.00 each including postage.

The relevant issues are

- 1983 Vol. 14, No 3 (4 copies) This contains the 1982 provisional report on diving related deaths in Australia.
- 1984 Vol 14, No 1 (23 copies) This contains Professor Brian Hill's paper on "Decompression Physiology" presented at the 1983 Annual Scientific Meeting.
- 1984 Vol 14, No 2 (20 copies) This contains papers presented at the SPUMS-RAN Meeting in August 1983 and at the ANZICS-SPUMS Meeting in Rockhampton in October 1983.
- 1984 Vol 14, No 3 (18 copies) This contains further papers presented at the ANZICS-SPUMS Meeting in Rockhampton in October 1983.
- 1985 Vol 15, No 4 (23 copies) This contains papers from the 1985 Annual Scientific Meeting in Bandos and from the New Zealand Chapter of SPUMS Meeting in November 1985, including an account of the formation of the New Zealand Chapter.

Orders, with payment, should be sent to

SPUMS 80 Wellington Parade EAST MELBOURNE VIC 3002 Australia

# DIVER EMERGENCY SERVICE

# 008-088-200

The duty supervisor of the Intensive Care Unit at the Royal Adelaide Hospital will answer the telephone and when told that it is a diving emergency will contact the on-call diving doctor. The call will be diverted to the diving doctor who will offer the caller expert advice. Civilian and naval doctors experienced in the treatment of diving accidents from all over Australia will be taking part in DES.

The diving casualty should contact DES on 008088-200. In most cases he will be advised to attend the local hospital unless he has easy access to one with a hyperbaric unit. That hospital will be contacted by DES with advice. The hospital will notify the nearest hyperbaric unit and arrange a hospital to hospital transfer. It will also notify the local ambulance service. If necessary the hyperbaric unit will alert the retrieval agency, such as the National Safety Council of Australia (Victorian Division) who have portable recompression chambers and aircraft to carry them. If specialist transfer is necessary the local ambulance service will arrange it with the retrieval agency.

# ELECTION OF EXECUTIVE COMMITTEE FOR 1987-1988

Nominations will be called for the positions of President, Secretary, Treasurer, Editor and three committee members in December 1986.

Each nomination has to be proposed and seconded and countersigned by the nominee.

Nominations are to be in the hands of the Secretary of SPUMS (Dr David Davies, Suite 6, Killowen House, St Anne's Hospital, Ellesmere Road, MOUNT LAWLEY WA 6050) by 1 March 1987.

Those elected will take office at the 1987 Annual General Meeting.

# DOCTORS WITH TRAINING IN UNDERWATER MEDICINE

We publish below a list of current members of SPUMS resident in Australia who have completed at least the Royal Australian Navy School of Underwater Medicine introductory course or who have notified the Secretary as requested in the SPUMS Journal (1985 15(2): 3) that they have had equivalent training.

The list has been compiled with the cooperation of the School of Underwater Medicine (SUM) and includes all members that can be identified from the SUM records as well as those who wrote in. As a result it may include doctors who no longer do diving medicals.

The addresses given are those to whom the Journal is sent and so may not be their professional rooms.

Those who have had equivalent training and whose names are not in this list are asked to write to the Secretary of SPUMS giving details of their training.

# Australian Capital Territory

Dr GD Davies 28 Berne Crescent MACGREGOR NSW 2615

# New South Wales

Dr JM Anderson 105 Karalta Road ERINA NSW 2250

Dr T Anderson School of Underwater Medicine HMAS Penguin BALMORAL NAVAL PO NSW 2091

Dr G. Barry Box 268 NEWPORT BEACH NSW 2106

Dr C Edmonds 25 Battle Boulevard SEAFORTH NSW 2092

Dr C Finlay-Jones 165 Morgan Street MEREWETHER NSW 2291 Dr R Gray 21 Coombar Close COFFS HARBOUR NSW 2420

Dr R Green 47 Shorter Avenue BEVERLEY HILLS NSW 2209

Dr TJ Horgan 232 Mona Vale Road ST IVES NSW 2075

Dr P Kolisch 33 Mann Street NAMBUCCA HEADS NSW 2248

Dr R Lloyd-Williams 102 Yanko Road WEST PYMBLE NSW 2073

Dr C Lowrey 233 Raglan Street MOSMAN NSW 2088

Lcdr CJ McDonald PO BOX 875 BALLINA NSW 2478

Dr W Pettigrew C/- Lidcombe Hospital LIDCOMBE NSW 2141

Dr CP Pidcock 39 Stockton Road NELSON BAY NSW 2315

Dr F Summers 56 Hickson Street MEREWETHER NSW 2291

Dr A Temperley 16 Normanhurst Road NORMANHURST NSW 2076

Dr I Unsworth Hyperbaric Unit Prince Henry Hospital PO Box 333 MATRAVILLE NSW 2036

Dr A Vane Police Medical Officer NSW Police Headquarters GPO Box 45 SYDNEY NSW 2001

Dr DG Walker 1423 Pittwater Road NARRABEEN NSW 2101

Dr DB Wallace 1/26 Aubin Street NEUTRAL BAY NSW 2089

Dr KJ Wishaw 5 Clearly Avenue CHELTENHAM NSW 2119

# Queensland

Dr Chris Acott 39 Oswald Street ROCKHAMPTON QLD 4700 Dr JW Cairns 65 Potts Street BELGIAN GARDENS QLD 4810

Dr I Gibbs PO Box 131 MACKAY QLD 4740

Dr J Orton Townsville General Hospital TOWNSVILLE QLD 4810

Dr D Pashen 3 White Street INGHAM QLD 4850

Dr D Richards 5/25 Ascog Terrace TOOWONG QLD 4066

Dr P Sullivan 33 Rutledge Street COOLANGATTA QLD 4225

Dr RL Thomas 39 Kersley Road KENMORE QLD 4069

Dr M Unwin 8 Fulham Road PIMLICO QLD 4810

Dr RM Walker Gold Coast Hospital Nerang Street SOUTHPORT QLD 4215

Dr J Williamson 137 Wills Street TOWNSVILLE QLD 4810

Dr K Woodhead Suite 9 Milton Shopping Centre Baroona Road MILTON QLD 4064

# South Australia

Dr D Gorman Hyperbaric Unit Royal Adelaide Hospital ADELAIDE SA 5000

Dr W Heddle 19 Alexander Avenue ASHFORD SA 5035

Dr G Rawson 4 Brierbank Terrace STONYFELL SA 5066

Dr AW Swain 46 The Parade NORWOOD SA 5067

#### Tasmania

Dr D Griffiths 9 Topham Street ROSEBAY TAS 7015 Dr V Hallet 12 Moirunna Road LINDISFARNE TAS 7015

Dr M Martin Dept of Anaesthesia Royal Hobart Hospital HOBART TAS 7000

Dr P McCartney PO Box 1317N HOBART TAS 7001

# Victoria

Dr G Broomhall 472 Belmore Road NORTH BOX HILL VIC 3129

Dr CBE Davis 8 Ascot Street North BALLARAT VIC 3350

Dr J Knight 80 Wellington Parade EAST MELBOURNE VIC 3002

Dr C Lourey 25 Hastings Street FRANKSTON VIC 3199

Dr JE Mannerheim 25 Wellard Street BOX HILL VIC 3128

Dr I Millar National Safety Council of Australia (Vic Division) 1 Chickerell Street MORWELL VIC 3840

Dr R Moffitt 1170 Main Road ELTHAM VIC 3095

Dr LJ Norton 44 Eleanor Street FOOTSCRAY VIC 3011

Dr K Shepherd 7 Young Street BRIGHTON VIC 3186

Dr J Silver PO Box 140 WILLIAMSTOWN VIC 3016

Dr G Zimmerman 46 Begonia Road ELSTERNWICK VIC 3185

# Western Australia

Dr AR Adams 24 Gilford Road DUNSBOROUGH WA 6281

Major P Alexander RAP Special Air Services Regiment Campbell Barracks SWANBOURNE WA 6010

# 106

Dr G Carter 254 Canning Highway EAST FREMANTLE WA 6158

Dr DE Davies Suite 6 Killowen Mouse St Anne's Hospital MOUNT LAWLEY WA 6050

Dr G Deleuil 135 Dunedin Street MOUNT HAWTHORN WA 6016

Dr H Oxer 331 Riverton Drive SHELLEY WA 6155

Dr J Rippon 764 Canning Highway APPLECROSS WA 6153

Dr A Robertson Sick Quarters HMAS Stirling PO Box 228 GARDEN ISLAND WA 6168

Dr J Taylor PO Box 498 EXMOUTH WA 6707

Dr R Wong 34 Loftus Street NEDLANDS WA 6009

Errors in this list should be notified to the Secretary of SPUMS

Dr David Davies Suite 6 Killowen House St Anne's Hospital Ellesmere Road MT LAWLEY WA 6050

#### PROJECT STICKYBEAK

This project is an ongoing investigation seeking to document all types and severities of diving-related incidents. Information, all of which is treated as being CONFIDENTIAL in regards to identifying details, is utilised in reports and case reports on non-fatal cases. Such reports can be freely used by any interested person or organization to increase diving safety through better awareness of critical factors. Information may be sent (in confidence) to:

Dr D Walker PO Box 120 NARRABEEN NSW 2101

# TRAINING STANDARDS FOR THE RECREATIONAL DIVER

#### Wal Williams

On 18-19 June 1986, The Standards Association of Australia (SAA) called a meeting of the Working Group SF/17/-/14, Scuba Diving, Basic Requirements, in Townsville. This Working Group is part of Committee SF/17, Work in Compressed Air, and was formed as a result of a request from the Australian Underwater Federation (AUF) to produce an acceptable Australian standard for a scuba diver. This standard will be the basic requirement for any diver whether recreational, scientific, search and rescue or professional.

Present at the meeting were representatives from the AUF, the Federation of Australian Underwater Instructors (FAUI), the National Association of Underwater Instructors (NAUI), the Professional Association of Diving Instructors (PADI), the Australian Institute of Marine Science (AIMS), Sydney University, the New South Wales Department of Sport, Leisure and Tourism, and the New South Wales Police Divers. The AUF was represented by myself and Frank Poole who was also representing the Scuba Divers' Federation of Australia (SDFA).

The meeting received many submissions from bodies with an interest in diving, but the two main submissions were forwarded by the AUF, on the National Coaching Accreditation Scheme (NCAS), (our Standards and Procedures) and the combined submission from FAUI, NAUI and PADI (called the Australian Scuba Council (ASC)).

Despite pressure from the representative of FAUI to have the Australian Scuba Council submission of a draft standard used as the basic document, the SAA used the AUF "Philosophy Behind the Australian Recreational Diving Standards" (ARDS) paper as the base document for the discussions.

After two days in which we were forced to accept some reductions and in turn forced some improvements to the ASC draft standards some form of agreement was reached. The draft standard produced was:

# Theory

To be the same as ARDS requirements.

#### **Basic Water Skills**

To be able to swim 200 metres any style, with no time limit, and then tread water for 2 minutes.

# **Snorkel Tests**

These were all reduced because they are also tested while on scuba. The only remaining snorkel test is a breath-hold dive demonstrated during the scuba surface swim.

# **Open Water Scuba Tests**

- 1. Prepare, assemble and fit all equipment correctly.
- 2. Enter the water correctly from beach, boat, jetty, rocky shore, or through surf and demonstrate alternative methods of entry.

3. Demonstrate all compulsory hand signals. There is a disagreement between the 9 compulsory World Underwater Federation (CMAS) signals and the 25 US signals which have been accepted by the ASC. We agreed on 8 signals to be made compulsory for the course and which are the same in both organisations. These are OK, OK on surface, Distress on the surface, Up, Down, Something Wrong, Buddy-Breathe, and Stop (see figure).





- 7. Swim 200 metres on the surface wearing, but not using, scuba. During the swim demonstrate skill in submerging and snorkel clearing techniques.
- 8. In at least 3 metres of water buddy breathe with buddy and swim for a least 25 metres while buddy breathing.
- 9. In at least 3 metres of water swim with buddy for at least 25 metres using an alternative air supply.
- 10.Demonstrate neutral buoyancy with buoyancy compensator (BC) deflated at the surface.
- 11.Demonstrate proper use of BC at depth.

# Rescue

- 1. Achieve the Royal Life Saving Society (RLSS) 'A' resuscitation certificate or carry out the equivalent training.
- 2. Using scuba, dive to recover a simulated nonbreathing scuba diver in 3 metres of water, surface and tow the diver 25 meires while conducting inwater expired air resuscitation (EAR), call for help and prepare to land. This test can be examined in a pool.







- 4. In at least 3 metres of water, swim 25 metres withouL a mask, replace the mask and clear it.
- 5. In at least 3 metres of water demonstrate the ability to remove and replace all equipment except the weight belt.
- 6. The student is to demonstrate ability to navigate underwater.

# **Open Water Diving Under Direct Supervision**

The compulsary dives conducted **after** the above tests are a **minimum** of four dives. One dive from the shore and one from a boat. Three dives are to be to deeper than 9 metres, one of which must be deeper than 15 metres. The total bottom time of the four dives is to be at least 140 minutes.

# Instructor/Student Ratios

It was agreed that the maximum instructor/student ratio will be 1/8. It was made quite clear that this ratio can only be used in optimum conditions and that any instructor who abused this ratio did so at his or her own risk, ie. "instructor beware".

The results of this working group will be presented to the main committee where it is expected that it will be accepted as a first draft. This first draft will then be sent out for public comment.

It is expected that it will take up to two before the full process of draft, comment, re-draft, etc. will produce an acceptable code. Therefore we have time to work towards upgrading any area found unacceptable by the AUF.

Lt Col WA Williams is the Chairman of the Technical Committee of the AUF. His address is 46 O'Rourke Street, WEETANGA ACT 2614.

The article below applies to the United Kingdom. Neither course is suitable, unaltered, for Australian conditions where doctors examining sports divers must be able to advise them about diving safety.

# DIVING MEDICAL ADVISORY COMMITTEE

RECOMMENDATIONS ON THE TRAINING AND REFRESHER TRAINING OF DOCTORS INVOLVED IN THE EXAMINATION OF PROFESSIONAL DIVERS AND IN THE TREATMENT OF DIVING-RELATED ILLNESSES

# 1. INTRODUCTION

We have been concerned for some time at the lack of positive guidance on the standards to be attained, and maintained by doctors undertaking the examination of professional divers, and the treatment of diving-related illnesses.

The recommendations which follow have been formulated from expert opinion drawn from many doctors who are actively engaged in diving medicine in the UK, Norway and elsewhere.

We would like to express the hope that they will be endorsed by appropriate Government Departments, and as a result, that any training establishment which purports in the future to offer doctors courses in these subjects will be obliged to comply as a minimum with these recommendations. We feel this to be essential in the long-term interests of the diving industry, and particularly of the divers themselves.

Our recommendations as to the content of each course have been arrived at after much thought and discussion, and are therefore firm. We recognise however, that the duration and order in which they appear in each course timetable may have to be adjusted in the light of local circumstances.

# 2. SCOPE DEFINITION

The recommendations throughout this paper are related to the three categories of doctor involved in one aspect or more of diving medicine. These categories are based on the 1981 EDTC Guidelines, as follows:-

#### A. Examining Medical Doctor For Professional Divers A doctor trained to conduct medical examinations on professional divers for fitness to dive.

- B. **Diving Emergency Medical Doctors** A doctor trained to work with divers and in particular, to cope with the medical aspects of every kind of diving emergency. He must be fit to go under pressure.
- C. Specialists
  - Specialists in Diving Medicine A doctor generally recognised in the international diving community as being well experienced in aspects of diving medicine, such as a medical doctor who is consulted on difficult or unusual cases by Examining Medical Doctors for Divers, and by Diving Emergency Medical Doctors, and who has an expert knowledge of diving physiology.
  - ii. <u>Associated Specialists</u> A specialist in some particular field (other than diving) who has an expert knowledge of the diving aspects of his special subject.

# 3. INITIAL TRAINING FOR GROUP A - EXAMINING DOCTOR

In our examination of this requirement, we have considered carefully whether to restrict the syllabus to normal office hours, or whether as appertains in at least one UK training establishment, have generally informal evening sessions most days, and thus offer longer tuition time, and additional opportunities for exchange of experience, etc.

So far as this particular course is concerned, we have come down firmly in fayour of a 5-day course with tuition hours not exceeding 7 hours per day. In reaching this conclusion, we have taken account of the fact that it would be a mstake to overestimate the learning capacity of the students on this course, most of whom will be unfamiliar with most aspects of diving medicine and their related commercial importance.

It is clear that since this course is intended for 'examining' doctors, everything on the syllabus must be geared to helping such doctors to acquire as much background knowledge as possible on diving and the diving industry, as well as a basic understanding of diving physics and physiology.

Given the above. together with an appreciation of the inter-relationship between 'normal' medical conditions and the diving environment, it ought to be possible by the end of the course for students to have a much fuller understanding of all these matters. They ought thereby, to be better equipped thereafter to come to a decision on whether or not a man is fit to dive, which has been based on a sound knowledge of the possible consequences which could follow that decision.

We feel that the emphasis must be on a syllabus designed for doctors examining professional divers rather than sports divers, and should include the items appearing under Group a. - Examining Doctors in Appendix E.

A proposed timetable and syllabus appear in detail in Appendices A and E.

# 4. INITIAL TRAINING - GROUP B - DIVING EMERGENCY DOCTOR

We have carefully considered the question of an appropriate length of time for this course, bearing in mind the pressures and the other demands which will undoubtedly be made on the time of those doctors attending.

We are convinced, however, that there is no way in which justice can be done to the subject matter, or that lasting benefit can accrue to those attending, unless the course lasts for 2 full weeks. Again, we have come down in favour of restricting tuition sessions to not more than 7 hours per day, although some of the sessions could well run on informally into the evening.

Our experience suggests that the revision of background physics and physiology, a pre-requisite to any therapy training; some first-hand experience of the diving environment; familiarisation with the clinical aspects of diving medicine, together with exhaustive discussion and analysis of case histories, all add up to a course of two weeks' minimum duration. We are convinced that a course of shorter duration would be a waste of time and effort.

We recognise the desirability of having a period of practical experience after completing this course, before a doctor could be considered fully qualified to give expert advice in a diving emergency. Although there is no substitute for practical experience, it is difficult to see how this could be built into any course syllabus. We feel that this requirement can best be met by the handling of simulated diving emergencies in the course syllabus proper.

Further, we are convinced that all doctors likely to be pressurised in surface decompression chambers should be examined and certified fit to do so.

A proposed timetable and syllabus for this course appear in Appendices B and E respectively.

# 5. INITIAL TRAINING - GROUP C (i) AND (ii) SPECIALISTS

There is obviously no specific requirement here, but see paragraphs 8 and 9.

# 6. REFRESHER TRAINING FOR GROUP (a) - EXAMINING DOCTORS

We feel that it is essential to have different refresher courses for the different groups, and this

paragraph deals only with refresher courses for examining doctors.

One of the principal difficulties about short refresher courses is that if they are too short, students will not feel that it is worthwhile to travel far to attend. On the other hand, making then sufficiently long to meet this requirement, brings the added responsibility of ensuring that all the content is still relevant, and not in any way padding.

We would see about 12 hours as being adequate refresher time for Group (a), and would suggest the following timetable:

Thursday 1030 - 1815 + Evening session including dinner with invited guests. Friday 0915 - 1500

The syllabus should include the following:-

- Introductory talk bringing students up-todate with developments in the field of diving medicals, <u>not</u> therapy.
- (ii) We feel that short refresher courses of this kind can be made much more interesting if all those who are to participate write in about three weeks beforehand with specific problems which they have encountered. Directing staff will examine these in the interim, and the ensuing answering and discussion can almost certainly be of value to all.

We are of the opinion that examining doctors should attend these refresher courses every three years.

A proposed timetable and syllabus appears in detail in Appendix C.

# 7. REFRESHER TRAINING FOR GROUP B -DIVING EMERGENCY DOCTORS

We are of the opinion that refresher courses for this Group need to be longer than those for Examining Doctors, and our recommendation is for 5 days, with tuition not exceeding 7 hours per day. Again, however, there will be occasions when some of these day-time sessions run on informally into the evening.

We attach great importance on this course to case history discussion, as it would appear to us that this would be particularly beneficial to those attending, experienced as they would be in the subject before coming on the course.

It is our view that diving emergency doctors should attend these refresher courses every three years.

We recommend that every doctor in this category should undergo a practical session in a chamber. We recognise, however, that this may impose practical difficulties at some training establishments. In these circumstances, alternative arrangements for this chamber session should be agreed with the course organiser, prior to the commencement of the course.

A proposed timetable and syllabus for this course appears in detail in Appendix O.

# APPENDIX A

# INITIAL TRAINING FOR GROUP A - EXAMINING DOCTORS 33 HOURS

(The numbers against each session relate to the lectures listed in Appendix E attached)

	0915	1015	1030	1130	1230	1400	1500	1515	1615	1715	1830	1930	
	1015	1030	1130	1230	1400	1500	1515	- 1615	- 1715	1815	1930	2130	
MON	(i)	COFFEE	(ii)	(iii) (half)	LUNCH	(iv) (half)	TEA	(iv)	(v)(a)	(v)(b) (v)(f)	DINNER	_	
TUE	(v)(e)	COFFEE	(vi)	(viii) (half)	LUNCH	(viii) (half)	TEA	(ix)(a)	(ix)(b)	(x)	DINNER		
WED	(xiii)	COFFEE	(xiv)	(xiv) (cont)	LUNCH	(xiv) (cont)	TEA	(xiv) (cont)	(xv)	(xviii) (xvi)(c)	DINNER		
THU	(xxiv)	COFFEE	(xix)	(xix)	LUNCH	(xxi)	TEA	(xxi) (cont)	(xxi) (cont)	(xxi) (cont)	DINNER		
FRI	(xxii)	COFFEE	(xxii)	(xxii)	LUNCH	(xxii)	TEA	(xxv)	_	_	_	_	

# APPENDIX B

# INITIAL TRAINING FOR GROUP B - DIVING EMERGENCY DOCTORS 65 HOURS

(The numbers against each session relate to the lectures listed in Appendix E attached)

	0915	1015	1030	1130	1230	1400	1500	1515	1615	1715	1830	1930	
DAT	- 1015	1030	- 1130	- 1230	1400	1500	- 1515	- 1615	- 1715	- 1815	1930	2130	
MON	INTRO + (i)	COFFEE (cont)	(iii)(d)	(iii)(d)	LUNCH	(ii)	TEA	(iv)	(iv) (cont)	(iv) (cont)	DINNER		
TUE	(v)	COFFEE	(v) (cont)	(v) (cont)	LUNCH	(v) (cont)	TEA	(vi)	(vii)	(vii) (cont)	DINNER	—	
WED	(viii)	COFFEE	(viii) (cont)	(viii) (cont)	LUNCH	(viii) (cont)	TEA	(viii) (cont)	(xv)	(xv) (cont)	DINNER	—	
THU	(xi)	COFFEE	(xi) (cont)	(ix)	LUNCH	(xviii)	TEA	(xx)	(xx) (cont)	(xii) (xiii)	DINNER	—	
FRI	(xxiv)	COFFEE	(x)	(x) (cont)	LUNCH	Review of the week	TEA	_	_	—	—	_	

# WEEKEND BREAK

MON	(xvii)	COFFEE	(xvii) (cont)	(xvi)	LUNCH	(xvi) (cont)	TEA	(xxii)	(xxii) (cont)	(xxii) (cont)	DINNER	_
TUE	(xxii)	COFFEE	(xxii) (cont)	(xxii) (cont)	LUNCH	(xxii) (cont)	TEA	(xxii) (cont)	(xxii) (cont)	(xxii) (cont)	DINNER	(xxii) (cont)
WED	(xxvi)	COFFEE	(xxvi) (cont)	(xxvi) (cont)	LUNCH	(xxvi) (cont)	TEA	(xxvi) (cont)	(xxvi) (cont)	(xxvi) (cont)	DINNER	_
THU	(xxvi)	COFFEE	(xxvi) (cont)	(xxvi) (cont)	LUNCH	(xxvi) (cont)	TEA	(xxvi) (cont)	(xxvi) (cont)	(xxvi) (cont)	DINNER	(xxiii) (1/2 hr only)
FRI	(xxii)	COFFEE	(xxii)	(xxii)	LUNCH	(xxv)	TEA	_	_	_	_	—

# APPENDIX C

# **REFRESHER TRAINING FOR GROUP A - EXAMINING DOCTORS**

# 12 HOURS

DAY	0915	1015	1030	1130	1230	1400	1500	1515	1615	1715	1830	1930	
	- 1015	- 1030	- 1130	- 1230	- 1400	- 1500	- 1515	- 1615	- 1715	- 1815	- 1930	- 2130	
тни	_	COFFEE	(1)	(7)	LUNCH	(8)	TEA	(2)	(3)	(4)	DINNER	(11)	
FRI	(5)	COFFEE	(9)	(10)	LUNCH	(6)	—	—	—	—	_	—	
<u>KEY</u>													
1.	. Introductory Talk, bringing students up-to-date, by ? Diving Specialist/Diving Emergency Doctor?												

3.) Role-playing syndicate sessions on problems sent in earlier by students, but also mainly directing
 4.) staff pre-set exercises.

5. Debrief on (2), (3) and (4).

2.)

6. Self-assessment examination.

7. Case histories on problems arising from medical examinations, plus revision session on standards of fitness.

8. Revision lecture on the Physics of Diving and the Physics of Gases.

9. Revision lecture on Safety - psychology, selection, drugs, alcohol, diet, fatigue and training.

10. Revision lecture on Diving Related Medical Conditions - The Sick Diver and The Injured Diver.

11. Evening session/Dinner with invited guests and guest speaker from HSE?/operators/diving contractors/ local doctor.

# APPENDIX D

# REFRESHER TRAINING FOR GROUP B - DIVING EMERGENCY DOCTORS 32 HOURS

DAY	0915	1015	1030	1130	1230	1400	1500	1515	1615	1715	1830	1930	
	1015	1030	1130	1230	1400	1500	- 1515	- 1615	- 1715	1815	1930	2130	
MON	(1)	COFFEE	(2)	(3)	LUNCH	(4)	TEA	(5)	(6)	(7)	DINNER	_	
TUE	(8)	COFFEE	(9)	(10)	LUNCH	(11)	TEA	(12)	(13)	(14)	DINNER	—	
WED	(15)	COFFEE	(16)	(i7)	LUNCH	(18)	TEA	(19)	(20)	(21)	DINNER	(34)	
THU	(22)	COFFEE	(23)	(24)	LUNCH	(25)	TEA	(26)	(27)	(28)	DINNER (33)	(33)	
FRI	(29)	COFFEE	(30)	(31)	LUNCH	(32)	TEA	_	_	_	_	_	

<u>KEY</u>

1. Introduction and General Review of recent clinical and technological development.

2.-7. Revision periods on Physics of Diving, Physics of Gases, Physiology of Diving, etc.

8.-14. Case histories/Syndicate Work.

15.-21. Practical Diving Instruction, including Chamber Dive to 50 metres on air.

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- 22.-28. Case histories/Syndicate Work (cont'd).
- 29.-31. It would be unwise to be too specific about allocating subject matters to these periods. As any course progresses, weaknesses become apparent and extra time is needed. This is particularly relevant on refresher courses. These periods, on the last morning of the course have been left to the discretion of Directing Staff for this reason.
- 32. Self-assessment examination.
- 33. This would seem to be very suitable for an end-of-course semi-formal dinner, with an appropriate guest speaker, either speaking at the dinner, or at an evening session thereafter. Local diving doctors should also be invited to the dinner.
- 34. Optional evening session on cardio-pulmonary resuscitation, putting up drips, etc.

	<u>APPENDIX E</u>		
		Group A Examining Doctor	Group B Diving Emergency Doctor
		<u>Hours</u>	<u>Hours</u>
(i)	<u>Types and History of Diving</u> (General, breathhold, scuba, surface supplied, bounce, saturation, excursion)	1	1/2 (review)
(ii)	<u>Diving Systems and Equipment</u> (theory), including Dive Planning and Safety Procedures	1	1
(iii)	<ul> <li><u>Physics of Diving</u></li> <li>(a) Pressure</li> <li>(b) Partial pressure</li> <li>(c) Effects of change of pressure on gas-containing spaces (including sinus, teeth, chest)</li> <li>(d) Review of diving physics</li> </ul>	1	2
(iv)	<u>Gases</u> (a) Air, nitrogen, narcosis (b) Oxygen and HBO (c) Carbon dioxide, carbon monoxide, helium (d) Gas laws, partial pressure calculations	2	3
(v)	<ul> <li><u>Physiology of Diving</u></li> <li>(a) Respiration, including gas density and resistance (immersion effects, respiratory and circulatory), correlated with known medical disorders</li> </ul>	1	1/2
	<ul> <li>(b) HPNS</li> <li>(c) Thermal balance - heat and cold</li> <li>(d) Diet and metabolism</li> </ul>	1/2	1/2 1/2 1/2
	<ul> <li>(e) The ear in diving</li> <li>(f) Vision</li> <li>(g) Immersion, drowning and near-drowning</li> </ul>	1 1/2	1
(vi)	D <u>ecompression Theory and Tables</u> Haldane and modern	1	1
(vii)	Immediate Evaluation of the Diving Casualty		2
(viii)	<ul> <li><u>Decompression Sickness</u></li> <li>(a) Diagnosis (presentation and monitoring)</li> <li>(b) Therapy</li> <li>(c) DCS including inner ear and CNS</li> <li>(d) Barotrauma, including diagnosis and treatment of pulmonary barotrauma and arterial gas embolism)</li> <li>(e) Treatment tables</li> </ul>	1/2 1/2 1/2 1/2	1 1 1 1
(ix)	<u>Diving Related Medical Conditions</u> (a) The sick diver (b) The injured diver	1 1	1 1

				113
		Group A Examining Doctor	Group B Diving Emergency Doctor	_
		<u>Hours</u>	<u>Hours</u>	
(x)	<u>Use of Drugs including Adjuvant Therapy</u> (see also (xv))	1	2	
(xi)	Management of the Patient at Pressure		1	
	Anaesthesia, use of ventilators in chambers, coincidental illness and injury			
(xii)	Dangerous Marine Life		1/2	
(xiii)	<u>Aseptic Bone Necrosis</u> (and any other long-term alleged effects)	1	1/2	
(xiv)	Standards of Fitness	4		
	<ul> <li>(a) The examination, including emphasis on pre-disposing factors, residual symptoms of signs which might during routine examination</li> <li>(b) Evaluation of examination</li> <li>(c) Conditions restricting or preventing diving activity</li> </ul>			
(xv)	Safety	1	2	
	Psychology, selection, drugs of abuse, alcohol, diet, obesity, fatigue training			
(xvi)	Diving Accidents		2	
	<ul> <li>(a) The pathology of diving accidents</li> <li>(b) Accident investigation</li> <li>(c) Litigation vis-a-vis the medical profession</li> </ul>	1/2		
(xvii)	Organisation of a Diving Emergency Service		2	
	<ul><li>(a) Communications</li><li>(b) Training</li></ul>			
(xviii)	<u>Design and Acceptance Criteria</u> for personal diving equipment, such as underwater breathing apparatus	1/2	1	
(xix)	<u>Current legislation</u> , Government and other authoritative recommendations, etc., as they affect the Examining Doctor, with particular reference to those countries in which he may work	2		
(xx)	<u>Current legislation</u> , Government and other authoritative recommendations, etc., as they affect the Diving Emer Doctor, with particular reference to those countries in which he may work	gency	2	
(xxi)	Visit to a Chamber/Diving Vessel	4		
(xxii)	Case Histories/Syndicate Work	4	15	
(xxiii)	Relevance of Current Research Trends		1/2	
(xxiv)	Sports Diving/Women in Diving	1	1	
(xxv)	Self-assessment examination	1	1	
(xxvi)	<u>Practical Diving Instruction</u> , including chamber dive to 50 metres on air		14	
	TOTALS	33	65	

Name: \_\_\_

\_\_ Dive School:\_\_

# **MEDICAL EXAMINATION OF PROSPECTIVE SCUBA DIVERS**

# Advice to the Examining Physician

Issuing an itemised account (so enabling the patient to claim medicare benefits) for diving medicals is prohibited by paragraph 25 (page 1b-4) of Section 1, Part B of the Notes for the Guidance of Medical Practitioners in the Health Benefits Schedule Book, dated 1st February, 1984.

Depends Schedule Book, dated ist rebroary, 1997. Diving is a sport carried on in a non-respirable environment, the sea, using breathing apparatus. Sudden unconsciousness underwater is usually fatal when using Scuba equipment, as the relaxation of muscle tone accompanying unconsciousness results in the breathing regulator falling from the victim's mouth. The diver's next breath will then be water. This makes any condition which can cause sudden unconsciousness an absolute bar to diving. Such conditions include epilepsy and diabetics on insulin.

A further problem with the water environment is that pressure increases very rapidly with descent — one atmosphere extra pressure for every 10m of depth in the sea. The use of breathing apparatus, providing gas at ambient pressure, prevents problems of pressure-volume imbalance in the lungs during descent. However the middle ears and sinuses will develop problems on descent unless the pressure in these spaces equals ambient. There is no way of establishing the patency of sinus ostia by clinical examination. However, patency of the Eustachian tubes, and so the ability to equalize the middle ear pressures, can be established easily. Observation of the tympanic membrance while the patient holds his (or her) nose, shuts the mouth and blows (Valsalva Manoeuvre) will reveal ingress of air to the middle ear by movement of the drum. The Eustachian tube opening in the naso pharynx is normally closed. Swallowing opens the ostium. Therefore, a combination of a Valsalva and swallowing during the manoeuvre will give the best chance for air to travel up the Eustachian tube. Another way of opening the Eustachian tube is to protrude the jaw and wriggle it from side to side while performing a Valsalva manoeuvre. Failure to auto inflate a middle ear is an absolute bar to diving until the person can auto-inflate.

A further set of pressure related problems also occur in diving. These are related to decreasing ambient pressure, i.e. the ascent phase of the dive.

If an air-filled space cannot vent when the surrounding pressure is reduced, two things can happen. A space with elastic sides can expand but if the space has rigid walls the pressure in the space, remaining at the original pressure, becomes higher than ambient. The chest wall is elastic, but after a certain expansion the stretching of the lungs results in tears of the lung substance. Air can then enter the pulmonary venous drainage, pass through the left heart and be carried to the brain as air emboli. Unconsciousness and death can result. Thus any condition preventing normal emptying of the lungs is an absolute bar to diving.

Lung cysts, bullae, and other areas that empty slowly or not at all are an absolute bar to breathing air under pressure. These conditions are best detected by taking an X-ray of the chest in full inspiration and another in full expiration. Asthma is another such condition. It is in order to detect expiratory airway obstruction that a Vitalograph (or similar) test is required. Experience in the navies of the world with submarine escape training of many thousands, has shown that a disproportionate number of those suffering burst lungs have FEV<sub>1</sub>/VC ratios of below 75%. Such people do not need to hold their breath on ascent to damage their lungs; all they have to do is rise too rapidly. People with a FEV<sub>1</sub>/VC ratio below 75% cannot be considered fit for diving.

A normal FEV<sub>1</sub>/VC % but clinical signs of bronchospasm, especially on forced deep, rapid ventilation, is an indication of unfitness to dive.

Treatment with drugs is not suitable as:

(a) the effects can wear off underwater,

(b) the combined effects of pressure and broncho-dilatory drugs are uncertain.

It is hoped that the foregoing makes this list of absolute and relative contraindications to diving logical and comprehensible:

#### ABSOLUTE CONTRAINDICATIONS

Conditions causing unconsciousness Epilepsy Diabetics on insulin

#### LUNG CONDITIONS

Asthma Lung cysts Previous spontaneous pneumothorax Obstructive lung disease Lungs which empty unevenly (X-ray appearance) Previous Thoracotomy

# ENT CONDITIONS

Inability to autoinflate the middle ears Previous middle ear surgery with insertion of prosthesis to replace any of the ossicles.

#### RELATIVE CONTRAINDICATIONS

FEV<sub>1</sub>/VC ratio less than 75% Poor physical condition Previous myocardial infarction Pregnancy.

If in doubt about a candidate's fitness, it is safer for the candidate to be classed as unfit than fit to dive. Difficult decisions should be referred to a doctor experienced in Diving Medicine. These are to be found in each State.

#### **RECOMMENDED READING:**

EDMONDS, C., LOWRY, C. and PENNEFATHER, J.
Diving and subaquatic Medicine. 2nd Edition, 1981. Revised 1983.
Diving Medical Centre, Sydney.
The South Pacific Underwater Medicine Society exists:

(a) to promote and facilitate the study of all aspects of underwater and hyperbaric medicine;
(b) to provide information on underwater and hyperbaric medicine.

Enquiries should be addressed to

The Secretary, SPUMS,
C/- 80 Wellington Parade,
EAST MELBOURNE,
VICTORIA. 3002.



# SCUBA DIVING MEDICAL FORM FOR SPORTS DIVERS

ENOURIES: F.A.U.I. P.O. BOX 246 TUART HILL WESTERN AUSTRALIA 6060

COR P.O.B TUAR

IAEDICAL HISTORY - TO BE FILLED IN BY CANDIDATE

DATE OF BIRTH	IVORCED []						ELLENT	
	MARRIED D			NAME OF CONDITION:	TYPE OF DRUG:	DESCRIBE		
AMES	GLE D			O YES	IJ YES	LI YES	D FAIR	
OTHER N	SEN	ADDRESS:		ONE	ON D	ONLI	E POOR	
SURNAME	ADDRESS	NEXT OF KIN:	PRINCIPAL OCCUPATION	HAVE YOU ANY DISEASE OR DISABILITY AT PRESENT?	ARE YOU TAKING ANY TABLETS, MEDICINES OR OTHER DRUGS?	DO YOU PARTICIPATE II ANY SPORT OR LEISURE ACTIVITY?	HOW WOULD YOU RATE YOUR FITUESS?	

HAVE YOU EVER SUFFERED FROM OR DO YOU NOW SUFFER FROM ANY OF THE FOLLOWING:

	MEDICAL HISTORY	Q	YES
	Do you wear any glasses or contact lenses.		
N	Eye or visual problems.		
e	Hay fever.		
4	Sinusitis.		
ŝ	Any other nose or throat trouble.		
G	Deafness or ringing noises in ear.		
~	Discharging ears or other intection.		
æ	Operations on ears.		
6	Giddiness or loss of balance.		
ō.	Motion sickness (car, plane, sea).		
÷	Have you any disability when flying in aircraft.		
12	Dentures.		
13	Dental procedures (within the last month).		
ž	Severe headaches or migraine.		
5.	Fainting, blackouts, fits or epilepsy.		
16.	Unconsciousness.		
17	Concussion or head injury.		
ĝ	Sleep walking or frequent nightmares.		
19.	Severe depression.		
R	Claustrophobia.		
21.	Any other mental illness.		
32	Any heart disease.		
2	High blood pressure.		
24	Rheumatic fever.		
25	Swollen or painful joints.		
Ŕ	Abnormal shortness of breath.		
27.	Bronchitis or pneumonia.		
28.	Pleurisy or severe chest pains.		
29	Coughing up blood.		
g	T.B. (Consumption).		
31	Chronic or persistent cough.		
32.	Pneumothorax (collapsed lung).		
33	Asthma or wheezing.		
8	Any other chest complaint or chest injury or		
	operation on chest, lungs or heart.		_
35	Kidney or bladder disease.	_	_
35.	Diabetes.		
37.	Indigestion or peptic ulcer.		
R	Vomiting blood or rectal bleeding.		
2	Recurrent vomiting or diarrhoea.		_

	MEDICAL HISTORY (Contined)	<u>0</u>	YES
<b>1</b> 0.	Jaundice or hepalitis.		
41.	Malaria or other tropical disease.		
42	Venereal diseaso.		
<del>5</del> .	Severe loss of weight.		
4	Hernia or ruplure.		
45.	Haemorrhoids (piles).		
46.	Any skin disease.		
47.	Any reaction to drugs or medicines.		
48	Any other altergies.		
49	Any major joint or back injury.		
50	Any fractures (broken bones).		
51.	Any paralysis or muscular weakness.	_	
52.	Have you had any operations?		
ß	Have you been in hospital or mental institution		
	lor any reason?		
5	Have you ever been rejected for insurance?		
22	Have you been unable to work for medical reasons?		
99	Have you ever been on a pension?		
57.	Have you ever lived with a person with T.B.?		
58.	Has any member of your family had T.B.? or		
59	Attempted suicide or had mental illness? or		
8	Had lits or epilepsy?		
61.	Do you smoke?		
62	Approximate number of cigarettes a day.		
3	Do you drink alcohol?		
64.	Approximate daily consumption.		
65.	Have you any other illness or injury not mentioned		
	in this list?		
Ē	MALES ONLY		
8	Are you now pregnant?		
67.	Have you any incapacity during or before periods?		

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NOTES ON HISTORY (PHYSICIAN'S USE ONLY)

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DIVING MEDICAL HISTORY -- TO BE COMPLETED BY THE PROSPECTIVE DIVER

of list snorkel dive.	of first compressed air (scuba) dive.	per of compressed air dives since.	h of any dive.	ion of any dive.	
Approx. date of lirst snorkel div	Approx. date of first compresse	Approx. number of compressed	Greatest depth of any dive.	Longest duration of any dive.	
-	~i	e.	4	Ś	

HAVE YOU EVER SUFFERED, OR DO YOU NOW SUFFER FROM ANY OF THE FOLLOWING DISORDERS RELATED TO DIVING?

		2	3	
	Severe ear squeeze.			
	Rupture of eardrum.			
۱ <u></u>	Dealuess.			_
1	Giddiness or dizziness.			
١_,	Sovere sinus squeeze.			
۱.	Severe lung squeeze.			
	Ruptured lung (burst lung).			
1	Emphysema.			_
	Pneumothorax.			
1.2	Air embolism.			_
	Nitrogen narcosis.			~
<u>.</u> .	Decompression sickness (bends).			
<u></u>	Near drowning.			
-	Severe marine animal injury.			
<u>.</u>	Oxygen toxicity.			
	Carbon dioxide toxicity.			_
	Carbon monoxide toxicity.			
5	Dysbaric osteonecrosis (bones)			_
نسا	Any other diving incidents.			_
l				

NOTES ON HISTORY (PHYSICIAN'S USE ONLY)

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115

I CERTIFY THAT THE ABOVE INFORMATION IS TRUE AND COMPLETE TO THE BEST OF MY KNOWLEDGE.

 SIGNED DATE: \_

	-		_	
	7 Respiratory Fu	nction Test	REA	MARKS
	Vital Capacity			
	Percentage			
	Frequency H:			l
250	500 1000	0001 0002	6000 3	000
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XAMINATION	Nor- A	Abnor- No	OTES ON ABN	ORMALITIES
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	<ul> <li>Signature of</li> </ul>	physician		Date.
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	. Address of p	hysician		
	Telenhone			Postcode.
	n cech 20	ech n n n d(1) bia bia bia bia bia bia bia bia	in     in     Frequency Hz     Percentage       in     250     500     1000     1000       in     100     1000     1000     1000       in     1000     1000     1000	r frequency Hz Frequency Hz

The above form, produced by the Federation of Australian Underwater Instructors (FAUI), is a welcome step forward. For some years FAUI instructors have been giving their pupils copies of medical history and examination forms from the Standards Association of Australia AS 2299-1979 Underwater Air Breathing Operations. These forms were of little help to doctors who knew nothing of diving medicine.

The new form includes, on the first page, Advice to the Examining Physician, a short synopsis of the major contradictions to diving and why they are dangerous. This synopsis was first offered to FAUI by Dr John Knight in 1980. The other pages, based on AS 2299, Include questions about exercise taken and fitness, which were recommended in the SPUMS Journal (Vol 14 No 4: 6-15) in 1984.

We congratulate FAUI on their initiative in producing the new form, which will help non-diving doctors in their attempts to assess their patients. It is encouraging to find an instructor body publishing such an informative and comprehensive Form, which even includes two mentions of the fact that diving medicals are not rebateable.

The address of the Federation of Australian Underwater Instructors (FAUI) is PO Box 246, TUART HILL WA 6060.

# Continued from page 109

# 8. REFRESHER TRAINING FOR GROUP C(i) -SPECIALISTS IN DIVING MEDICINE

What refresher training can be considered necessary for an internationally acknowledged expert in diving medicine?

Surely this can only be accomplished by postgraduate study, by attendance at international diving medicine symposia, and by a continuing regular involvement in diving medical therapy.

Failure to maintain any of these three criteria, but particularly the latter, would undoubtedly in due course mean the fall of a star from the firmament, albeit perhaps only as far as the Heaviside Layer.

# 9. REFRESHER TRAINING FOR GROUP C(ii) - ASSOCIATED SPECIALISTS

We do not consider that we are qualified to make recommendations for refresher training for specialists in these other areas.

The address of the Diving Medical Advisory Committee is 28/30 Little Russell Street, London WC1A 2HN, England.

# **ORIGINAL PAPERS**

#### COMPARISON OF VINEGAR AND METHYLATED SPIRITS AS TREATMENT FOR CYANEA CAPILLATA (HAIR JELLYFISH) STINGS

#### Peter J Fenner and Peter F Fitzpatrick

#### SUMMARY

Vinegar has already been proven to cause de-activation of the nematocysts of many jellyfish and other Cniderians including the deadly Chironex fleckeri, and as such is the ideal immediate treatment on the beach.<sup>1</sup>

More recently it has been shown that Chyrsaora quinquecirrha (the North American sea nettle) reacts in the opposite way and vinegar causes discharge of the nematocysts<sup>2</sup> and because of its similarity to Chyrsaora , Pelagia noctiluca (little mauve stinger) must also be suspected of having nematocysts discharged by vinegar.

Recent tests with isolated tentacles of Cyanea capillata (the "hair jelly" or "snottie") also show that the nematocysts are actively discharged by vinegar but not by methylated spirits. However, there is no difference to the clinical effects of the sting and because of the life saving benefits of vinegar in Chironex stings, the initial first aid must continue to be vinegar poured in copious quantities all over the envenomated area.

## INTRODUCTION

All jellyfish, and many other Cniderians, possess the nematocyst, a small stinging cell by which they poison, paralyze and consequently trap their prey. Most of these nematocysts have different morphological features and by microscopic study, it is often possible to identify the envenomating animal.<sup>3</sup>

The nematocyst is a specialized capsule which contains the specific venom of the animal. Inside this capsule is a coiled thread tube which is hollow to allow the passage of venom.

On the outside of the nematocyst is a cnidocil which is a small 'trigger mechanism' which when touched causes the thread to uncoil itself quickly and it fires, rather like a harpoon, deep into the tissues of its prey. Hydrostatic pressure causes the vigorous 'firing' of the thread tube.<sup>3,4</sup>

The venom then passes through the hollow centre of the tube and is deposited into the body of the prey where it causes its specific effects.

The action of vinegar and methylated spirits is to dehydrate the tentacle, although it is obvious that different chemicals can have different effects on the nematocysts of different species.

Whilst trying different solutions for fixing and then studying the isolated tentacle of Cyanea capillata (the "hair jelly"), it was noticed that on a tentacle fixed in vinegar, most of the nematocysts had discharged.



Schematic drawing of a nematocyst firing

## LABORATORY EXPERIMENTS

Isolated pieces of Cyanea tentacle were mounted in solutions of sea water, vinegar (4-6 per cent acetic acid) and methylated spirits and studied under the microscope.

The slides with the tentacles in the sea water and the methylated spirits showed that very few of the nematocysts had discharged. Conversely, the slide with the tentacle fixed in vinegar showed that most of the nematocysts were discharged.

The experiment was also tried with tentacles still attached to the whole animal to see if there may be a 'nervous' response that could cause a reflex discharge of the nematocysts, but the results remained exactly the same ie. vinegar causes discharge of the nematocysts, whereas methylated spirit does not.

#### DISCUSSION

Vinegar is found to cause discharge of the nematocysts of Cyanea capillata whether or not these are attached to the animal or on isolated tentacles.

The sting of the Cyanea although florid in appearance, causes very little pain or discomfort and no systemic effects.<sup>3,5,6</sup> There is therefore little actual clinical effect of pouring vinegar on to an envenomated victim. There is a great clinical effect if vinegar is not used in victims stung by Chironex. By using vinegar you may save a life if the sting is from Chironex fleckeri while you will not cause any medical problems by using vinegar on Cyanea stings.

First aid teaching must remain simple if it is to work. The Surf Life Savers in North Queensland will be taught to continue the life saving technique of

Vinegar + Resuscitation = Life

# REFERENCES

- 1. Hartwick RJ, Callanan V and Williamson JAH. Disarming the box jellyfish. Med J Aust 1980; 1: 15-20.
- 2. Burnett JW, Rubinstein H and Calton GJ. First aid for jellyfish envenomation. Sth Med J 1983; 76: 870-872.
- 3. Cleland Sir JB and Southcott RV. Injuries to man from marine invertebrates in the Australian region. Canberra: NHMRC, 1965; Special Report Series No. 12.
- 4. Hyman LH. The Invertebrates. Protozoa through Ctenophora. New York: McGraw Hill Book Co., 1940: 382.
- 5. Williamson JAH. The marine stinger book. 3rd ed. Queensland State Centre, Surf Lifesaving Association of Australia, 1985.
- 6. Sutherland SK. Australian Animal Toxins: the creatures, their toxins and the care of the poisoned patient. Melbourne: Oxford University Press, 1983.

Dr PJ Fenner's address is PO Box 34, North Mackay QLD 4740.

Dr PF Fitzpatrick's address is PO Box 1284, Mackay QLD 4740.

A letter to the Medical Journal of Australia (1986 ii: 174) on this subject from these authors appeared in August. This paper is published to bring the information to a wider audience.

#### THE MORBAKKA -

#### ANOTHER SPECIES OF BOX JELLYFISH

#### Peter J Fenner

#### SUMMARY

There is a large cubomedusan in the family Carybdeidae that has previously been known as Tamoya haplonema or fire jelly. It has been reported throughout Queensland from Port Douglas to Moreton Bay where it is known as the "Moreton Bay stinger". After a recent case of severe envenomation in South Queensland and the examination of two specimens caught in Mackay Harbour, it has been recognized as being a new species.<sup>1</sup>

Until it is formally identified, the name 'morbakka' has recently been suggested by Dr Ron Southcott<sup>2</sup> after "Moreton Bay carybdeid medusa" the area in South Queensland where they were first reported.

Vinegar has been shown to inactivate the undischarged nematocysts (stinging cells) of this species and is recommended as the initial first aid treatment.<sup>1</sup>

# INTRODUCTION

On 18 and 23 April 1985, two large specimens of a morbakka were found in Mackay Harbour. The larger specimen was 130mm in diameter across the body of the bell, and the height of the bell was 180mm (Figure 1). The other specimen was 120mm in diameter and had a bell height of 150mm.



Figure 1. Morbakka caught in Mackay Harbour. Note the gloved hand. Nematocysts are present on the bell as well as on the tentacles.

Each specimen had four large mauve tentacles, one attached to each corner of the bell. The tentacles on the larger specimen were well preserved and reached a length of 600mm when extended, they contracted to a length of 200mm. After preservation in 10 per cent formalin and sea water they contracted even further to a length of 60mm. The tentacles were ribbon shaped 10mm wide but only 3mm thick. They had a multiple transverse bar pattern similar to, but much larger than those of the Chironex fleckeri. Unlike those of Chironex the central canal in the pedalium did not have the "hook" or "rose thorn" appearance.<sup>3</sup>

Also, unlike Chironex, the bell was covered with numerous warty mauve mamillations. Each of these contained hundreds of nematocysts which were capable of causing even the thick skin of the palm to tingle when the animal was picked up. The bell is transparent in the natural state but after preservation in formalin sea water becomes completely opaque.

# An envenomation in Moreton Bay

At 11:00 am on 20 January 1984, whilst swimming at Margate Beach, Moreton Bay, in one metre deep murky water, a 12 year old girl was stung by a "Moreton Bay Stinger". At the time there was a strong on-shore wind.

On surfacing from a dive under the water she received a large sting from a tentacle of a morbakka that stretched over her shoulder and down the front of her chest and her back. The predominant symptom at this time was a severe "burning" pain, which felt like the skin was on fire. Within a few minutes the skin had raised white weals with a surrounding red flare where the tentacles had touched and within half an hour she had developed a cough, backache and a feeling of a lump in the throat, symptoms which lasted for the next 24 hours. Oral paracetamol 500 mg and dexchlorpheniramine 2 mg were given but had little effect apart from that of sedation.

24 hours later the skin lesions were still red and raised but had stopped burning and become somewhat itchy and tender to touch. The lesions became paler by the third day and had a papulo-vesicular appearance which lasted another week before settling with no scarring.



Figure 2. Sting on the chest of a boy soon after he left the water. The raised white wheal is surrounded by a bright red flare.

# Use of vinegar

Experiments have been carried out by Dr Robert Hartwick which showed that weak (3-10 per cent) acetic acid solution (vinegar) inactivated the unfired nematocysts in the tentacles and bell of this species.<sup>1</sup> These tests were patterned on previous work by Hartwick et al,<sup>4,5</sup> for nematocysts of Chironex fleckeri and Physalia physalis.

# DISCUSSION

As the morbakka is of the Class Cubozoa (formerly called the Order Cubomedusae) it is a type of box jellyfish although not to be confused with the deadly Chironex fleckeri which is THE Northern Australian box jellyfish.

It has previously been incorrectly referred to as Tamoya haplonema, but as it lacks gastric cirri, one of the identification features described by Muller in 1859 for the Genus Tamoya<sup>6</sup> it has to be classified as a species of its own. However until it can be formally identified, Dr RJ Southcott suggested the name morbakka derived from Moreton Bay carybdeid medusa as it is from that area that several specimens have been described and a number of envenomations reported.<sup>2</sup> However specimens have also been caught as far north as Port Douglas as well as in Mackay and with a growing number of hearsay reports it is becoming obvious that this species is more prevalent than is generally known.

Vinegar has been shown to be efficient in disarming undischarged nematocysts in the adherent tentacle. thus preventing further envenomation. This role of vinegar is already proven for other species of jellyfish.<sup>4,5</sup> As it is non-flammable it is safe to use, and being cheap and readily available it has to be recommended as the immediate first aid treatment on the beach.

# REFERENCES

- 1 Fenner PJ, Fitzpatrick PF, Hartwick RJ and Skinner R. "Morbakka", another cubomedusan. Med J Aust 1985; 143: 550-555.
- 2 Southcott ŃV. The "morbakka". Med J Aust 1985; 143: 324.
- Southcott RV. Studies on Australian cubomedusae, including a new Genus and species apparently harmful to man. Aust J Marine Freshwater Res 1956; 7: 254-280.
   Hartwick RJ, Callanan V and Williamson JAH.
- 4 Hartwick RJ, Callanan V and Williamson JAH. Disarming the box jellyfish: nematocyst inhibition in Chironex fleckeri. Med J Aust 1980; 1:15-20.
- in Chironex fleckeri. Med J Aust 1980; 1: 15-20. 5 Hartwick RJ, Callanan V and Williamson JAH. Disarming the box jellyfish. Med J Aust 1980; 4: 335-338.
- 6 Muller F. Zwei neue quallen von Santa Catharina (Braisilien). Abhandl Naturf Ges Halle 1859; 5: 1.

Dr PJ Fenner's address is PO Box 34, NORTH MACKAY QLD 4740.

# A NEW HYPERBARIC UNIT FOR VICTORIA

# John Knight

The hyperbaric unit maintained by the National Safety Council of Australia, Victorian Division (NSCA) at Morwell has closed.

This closure was dictated by economics (the NSCA carried all the costs of treatment without any contribution from the Victorian government) and medical considerations. The chamber at Morwell was approximately 145 km from the nearest hospital with full intensive care facilities. There has been a need for

such facilities in the Morwell chamber at times. It is no longer acceptable to have a hyperbaric treatment complex situated outside the confines of a large hospital. Once the NSCA's contractual obligations to provide hyperbaric chamber cover were met by installing a chamber on their support craft the rationale for maintaining the Morwell chamber operational disappeared.

In August a deputation consisting of Dr CJ Lourey (Anaesthetist, Intensivist and Past President of SPUMS), Dr David Brownbill (Senior Neurosurgeon at Royal Melbourne Hospital), Dr Des Gorman (Director of the Royal Adelaide Hospital Hyperbaric Unit and guest speaker at the 1986 Annual Scientific Meeting) and a representative of the NSCA, Mr Andrew Wilson, saw the Minister for Health, Mr D White. They presented the case for siting the Morwell Chamber complex in a teaching hospital. The NSCA were willing to donate chambers and all ancillary equipment to the hospital for the costs incurred in transporting the chambers to their new site. The NSCA was also willing to enter into a service agreement with the hospital to provide staff to operate the chamber. The extra costs proposed for medical and nursing staff would be for a full time Director, and at least three nursing staff. This was based on the Adelaide experience.

The Minister for Health and the Victorian Government have accepted the advice of the deputation and the generous offer of the NSCA to donate their chambers has also been accepted.

The facility will be installed at the Alfred Hospital in Melbourne and it is hoped to have it working by the beginning of summer (December 1986). It is expected that the position of Director of the unit will be advertised around the world.

I understand that the complex will consist of a two compartment saturation capable chamber, designed for 200m, to which is mated a circular chamber to which is attached an oblong chamber with transfer under pressure facilities for the Drager Duocom two man transportable chamber.

The Health Department has issued an Interim Protocol for Management and Transport of Patients Requiring Hyperbaric Treatment. This protocol and part of the letter that accompanied it are reproduced below.

At present there are approximately 40-50 patients in Victoria each year who need to receive emergency treatment in a hyperbaric chamber, due .to problems associated with deep sea diving and certain other conditions such as carbon monoxide poisoning, cyanide poisoning and anaerobic infections.

In order to ensure that such patients continue to receive that level of care where indicated, interim arrangements have been made for them to be referred to interstate facilities linked with the Diver Emergency Service (telephone 008 088 200).

Until further notice, arrangements for the management and transport of these cases should be undertaken in accordance with the attached interim protocol.

As with any other Ambulance transport, charges will be made to the patient or, in the case of interhospital transfers, to the referring hospital by the Ambulance Service in accordance with standard ambulance charge rates.

# INTERIM PROTOCOL FOR MANAGEMENT AND TRANSPORT OF PATIENTS REQUIRING HYPERBARIC TREATMENT

In the event of a casualty presenting to a hospital with a condition for which emergency hyperbaric treatment is required, the following protocol should be followed:

1. The attending doctor will seek advice from the Diver Emergency Service by telephoning 008 088 200. The hyperbaric specialist answering the call will advise on management of the case and if necessary will arrange for admission to the appropriate hyperbaric unit.

2. If transfer to a hyperbaric unit is required, the hospital doctor will contact the local Ambulance Service to request ambulance transport of the casualty requiring hyperbaric retrieval to the nominated hyperbaric unit.

3. The local Ambulance Service will contact the Diver Emergency Service (telephone 008 088 200) and consult with the hyperbaric specialist on-call, to

- a. confirm that the unit is expecting a casualty
- b. discuss the most appropriate transport medium c. confirm that a retrieval under full hyperbaric
  - conditions will be required

4. If hyperbaric retrieval is required, the local Ambulance Service will contact the National Safety Council of Australia (telephone (051) 49 2333) and request that the retrieval be undertaken on behalf of the Ambulance Service.

5. The National Safety Council of Australia will undertake the subsequent retrieval, with the cooperation of the local Ambulance Service which will co-ordinate all movements and organise ground transport where appropriate.

6. If, following consultation between the hyperbaric specialist and the local Ambulance Service, it is decided that transport in a recompression chamber is either not necessary, or not practicable, the most appropriate method of transport will be determined. In this decision, the local Ambulance Service will be guided by the hyperbaric specialist. having in mind the unique requirements for the transport of these casualties.

In these circumstances the local Ambulance Service will also be responsible for advising the National Safety Council (051 49 2333) that hyperbaric retrieval is not required.

NOTE: Experience has shown that a significant number of diving casualties make initial contact with the National Safety Council of Australia at Morwell, rather than with a local medical officer or local hospital. As the diving casualty will almost certainly require immediate medical treatment, he or she will be directed to seek aid from local resources (eg. ambulance to nearest hospital). Having ascertained the local hospital, National Safety Council of Australia will contact the hospital to advise of the expected arrival of a diving casualty. The diving casualty will be instructed to request the local hospital to contact the Diver Emergency Service, as subsequent evacuation may be required.

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