Ciba-Geigy for their generous support with this conference and by bringing Dr Des Gorman from Australia. We have learnt much from Des's scientific approach and hope to benefit from his political success in co-ordinating Australia's diving medical facilities. Diving Emergency Service, (DES) (they tell me is not named after Des Gorman but form your own opinion), is an Australian-wide Diving and Envenomation Service co-ordinated in Adelaide by Des Gorman.

Next year, subject to your approval, the Executive proposes to run the scientific meeting in conjunction with OCEANS in mid-September on Auckland's North Shore. We also propose to co-support Carl Edmonds as guest speaker. A subsequent diving weekend will be held in November in 1987, Marlborough Sounds could be our conference venue! (Mikhail Lermontov) I would remind you that the SPUMS AGM is to be held in the Solomons in the first week of June 1987. Details will be forwarded to you by Anthony Newly of Allways Tours, arriving tonight.

Finally, I must thank the Conference Committee, Allan Wills, Jeff Olson and Peter Jansen, headed by Mark Fraundorfer, for their excellent organization of Annual Conference.

AFN SUTHERLAND
Chairman
New Zealand Chapter

NEW ZEALAND CHAPTER OF SPUMS ANNUAL CONFERENCE

5 to 7 April 1987

This meeting will be held at Tutukaka. For further information contact the Conference Convenor

Dr Peter Chapman-Smith
67 Maunu Road
Whangarei
New Zealand

LETTER FROM CMAS (WORLD UNDERWATER FEDERATION)

NO AGREEMENT BETWEEN CMAS AND PADI

During the last few months there have been articles in several diving magazines which gave the impression that agreements have been made between CMAS and PADI. **This is not true.**

Some organisations have made individual arrangements with PADI representatives/agencies, but these were not made on behalf of CMAS.

As an example, the Executive Bureau withdrew authorization for the New Zealand Underwater Association to issue CMAS certificates after they made such an agreement with PADI.

If there are any changes in the future, we will keep you informed.

Pierre Perraud
President, CMAS

SPUMS ANNUAL SCIENTIFIC MEETING 1985

PUFFER FISH POISONING

Struan Sutherland

Many fish are poisonous and a variety of toxins is involved. The flesh and organs of some fish are invariably poisonous, whereas others may show seasonal fluctuations in their toxic content. A useful general rule is to avoid eating the flesh of fish without true scales or large predatory fish. The former may be Toad or Puffer fishes, whose flesh and organs may contain the potent toxin, tetrodotoxin, and the latter may have accumulated the food chain poison, ciguatoxin. Both these toxins can cause death and neither can be removed by washing the fish or even by prolonged cooking.

ORDER TETRAODONTIFORMES
TETRODOTOXIC FISH

Tetrodotoxic fish are found in most sea waters of the world. In some countries, like Japan, the flesh of selected Toad fish, 'fugu', is a culinary delight, as traces of tetrodotoxin are considered to produce a pleasant tingling sensation. Officially fugu can only be prepared by specially trained people. Matsubara[1] considered that, once one had tried the dish, its popularity could easily be understood. He said: "A well-flavoured dish has thin slices of raw fish arranged in the form of a chrysanthemum, each slice representing a petal. The taste is delicate. To the author's knowledge, only one thing comparable in taste is crisp white Burgundy of a good year. Sometimes one feels slight numbness at the tip of one's tongue, although worrying, this enhances the delicacy of the dish. After all, the idea of sharing a potentially mortal risk with friends makes the whole evening exciting."

However, the risks of such pleasure are very high. Suenaga and Kotoku[2] state that from 1955 to 1975 three thousand individuals were poisoned by Puffer fish in Japan and 51 per cent died. Although both the ancient Chinese[3] and Egyptians[4] were aware of the dangers of many of these fish, Europeans were poorly informed. Captain Cook nearly died in 1774 after sampling a little of the roe and liver of a Toad fish in New Caledonia.[3]

Hundreds of species of tetrodotoxic fish exist throughout the world and some thirty species are found in Australia. Many of the smaller species are very common around jetties and in shallow water, where they are easily caught by fishermen. Most have the ability to inflate themselves into nearly perfect spheres, using either air or water, and thus they have common names such as Puffer fish, Toad fish, Globe fish, Toado, Swell fish and Balloon fish. Others like genus Diodon, the Long-spined Porcupine fish, have long spines all over their bodies and become spiky balls when inflated.

Characteristic features of these fish are the apparent absence of scales, the large eyes and the presence of four large teeth. In fact, all the teeth are fused together, but have an anterior division. The teeth are particularly strong and, according to the Melbourne Age of 17 April 1979, a 5-year-old girl had had two toes bitten off the previous day by a Toad fish in Shute

TABLE 1

LETHALITY OF CRYSTALLINE TETRODOTOXIN IN VARIOUS ANIMALS

TEST ANIMALS	ROUTE OF ADMINISTRATION	LETHAL DOSE (pg/kg)
Mice	subcutaneous	13 to 15 (LD50)
	intraperitoneal	11
	intravenous	8 to 10 (LD50)
	oral	180 (LD50)
	intracranial	0.3 (LD50)
Rats	subcutaneous	14
	intraperitoneal	12
	intravenous	10
	oral	147
Rabbits	subcutaneous	10
	intravenous	2
	oral	200
Cats	Intravenous	2
Dogs	subcutaneous	15
	intravenous	0.3 (minimum emetic dose)
	oral	70

Harbour, north Queensland. The average maximum length of Toad fishes varies with the species, most reaching about 15 cm. Gastrophysus scleratus (Gmelin), the Giant Toad fish which is found in all Australian states and grows to a length of 76 cm, was the species that Captain Cook sampled.[5]

Puffer fish are easily identified and fishermen generally treat them with revulsion and throw them back into the sea.

TOXIN

Toxicity

Tetrodotoxin (TTX) is one of the most toxic poisons known. Its intravenous LD_{50} in mice is 9 μg/kg.[6] Table 1 has been extracted from information supplied by the Sankyo Company with their crystalline TTX. It is interesting to note similar lethalities in the intravenous and subcutaneous routes, except in the case of dogs which are particularly prone to vomit. Obviously, the oral route either leads to reduced absorption of the purified toxin, or extensive detoxification, or a combination of both. However, when fish containing TTX is ingested, clearly plenty of toxin is rapidly absorbed and the clinical relevance of the experiments with ingested pure TTX described above is uncertain.

Biochemistry and Actions

Tetrodotoxin (TTX) was the name given by Tahara[7] to a purified toxic extract he obtained from the eggs of Puffer fish. The chemistry and historical aspects of TTX have been thoroughly reviewed by Courville.[8] TTX was isolated in a pure form from the ovaries of Puffer fish by Tsuda and Kawamura.[9] The structure of crystalline TTX was then studied by a number of workers in Japan and USA, and elucidation of the complex architecture of TTX was completed simultaneously in the Sankyo laboratories in 1964 (current Sankyo Company literature). TTX has an empirical formula of $C_{11}H_{17}O_8N_3$ and a molecular weight of 319.3. The reason it took so long to determine its structure is apparent from its unusual nature as illustrated in Figure 1. The oxygen molecule between C_5 and C_{10} marks a unique hemilactal link, which is necessary for activity.[5] Both the guanidinium group on the right hand side of the molecule and the hydroxyl groups are also essential for biological activity. TTX is insoluble in water and in ordinary organic solvents, but dissolves in weakly acidic solutions.

The most frequently quoted review of the actions of TTX is that of Kao.[3] Simpson[10] brought together reviews by a number of distinguished scientists, with individual chapters by the Australians Curtis and Gage. Blankenship[4] has also surveyed the literature. Because of these publications, only a summary of the actions of TTX is given here.

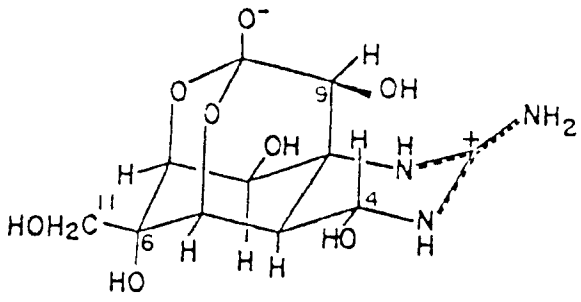

Figure 1. Structure of Tetrodotoxin
(Drawing kindly provided by Professor CY Kao.)

TTX is one of the most potent vasodepressors known and an intravenous dose can cause prolonged hypotension.[3] Clinically and experimentally, the most important effects of TTX are upon the nerve fibres, where blockage of excitability occurs. Because of the unique action of TTX, it has perhaps been more extensively studied by neurophysiologists than any other toxin. Narahashi et al[11] found that TTX stopped the passage of action potentials down nerves by selective inhibition of the sodium carrying mechanism, whilst the movement of potassium was not disturbed. The effect is selective for the sodium channel, not the sodium ion. Calcium dependent action potentials are not disturbed by TTX. This nerve blocking potential is greatest in peripheral nerves, but also effects sensory and autonomic nerve fibres. Skeletal and smooth muscle and, to a lesser extent, cardiac muscle have their excitability reduced by TTX. TTX also produces a central effect with depression of spinal reflex pathways and electroencephalographic activity.

TTX has not only been found in certain tissues of the tetrodotoxic fishes, but has also been isolated from the skin of Central American frogs of the genus Atelopus[12] and in the glands of the skin of the Californian newt of the genus Taricha.[13] TTX has been discovered in the posterior salivary glands of H maculosa by Sheumack et al,[14] as well as in the Pacific Goby.[15] There is no other animal toxin which has such a diverse distribution and the appearance of TTX in five such different animals has yet to be satisfactorily explained.

In tetrodotoxic fishes, TTX is mainly concentrated in the liver, ovaries, intestine and skin. The flesh is easily contaminated with TTX when the fish is caught or when it is being cleaned.

HUMAN INGESTION OF TTX

Usually within ten to forty-five minutes of eating part of a Puffer fish, signs and symptoms of poisoning have developed. Rarely, they may be delayed for several hours and usually the speed of onset and severity is in proportion to the amount of toxin swallowed.

Most cases have some nausea, but vomiting is uncommon. Even the mildest cases usually experience 'tingling' sensations and severe poisonings rapidly develop an alarming illness. The conscious state may remain unimpaired until near death; those who survive a near fatal episode may recall careless comments made by relatives or hospital staff (Case 2).[1] In Japan, last century, some severely paralysed victims recovered when facing imminent burial or cremation days after eating the fish. They could recall most details of conversations which had occurred before and after they became a 'corpse'.[16]

Fukuda and Tani[17] classified the four degrees of poisoning as follows:

Grade 1: Numbness around the mouth with or without gastrointestinal symptoms.

Grade 2: Numbness of tongue, face and other areas of skin. Evidence of early motor paralysis and incoordination. Speech slurred and patient may be thought to be drunk or drugged. Peripheral reflexes still intact.

Grade 3: Patient still conscious, but widespread paralysis, dyspnoea and hypotension well established and voice now lost.

Grade 4: Severe hypoxia, near complete paralysis and hypotension. Death due to respiratory failure.

Death may occur as rapidly as seventeen minutes after eating the tissues.[16] Sometimes widespread desquamation occurs during convalescence,

Deaths occurring from Puffer fish poisoning are reported in Australia from time to time. Southcott[5] reproduces an account from Sydney in 1821, in which a man 'who must have been a stranger to the Colony' died twenty minutes after eating Toad fish, which the locals knew to be poisonous. Some years ago a young Tasmanian couple, who had eloped, died in Gippsland. They were without money and ate whatever they could catch.

DIFFERENTIAL DIAGNOSIS

The diagnosis is usually straightforward, especially when a number of individuals is involved, as is often the case.

Ciguatera may present in a similar fashion but later, usually from two to twelve hours after the fish is eaten. In these cases, the offending fish or fishes is usually easily identified and is clearly not a Puffer fish. The ciguatera sufferer usually has reversed sensations, in particular hot objects feel cold and vice versa, but this phenomenon is absent in TTX poisoning. Mitchell[18] states that muscle pain may occur with ciguatera but not TTX poisoning. Puffer fish are not ciguatoxic fish. Other types of marine poisoning, or poisoning by eating contaminated food, usually lack the marked neurological signs produced by TTX.

FIRST AID

If the patient is conscious, he or she should be made to vomit by use of an emetic such as liquid ipecacuanha. If this is not available vomiting may be induced by inserting fingers into the back of the pharynx. Vomiting should not be induced in the unconscious patient, because of the risk of inhalation of vomitus. If respiration is inadequate, mouth to mouth resuscitation or more sophisticated maintenance of oxygenation should be commenced and hospital treatment sought as a matter of urgency.

CLINICAL MANAGEMENT

Apart from the use of gastric lavage, the care of the TTX victim, as outlined below, is applicable to other marine poisonings which produce a Flaccid paralysis, such as H maculosa the blue ringed octopus and venomous Cone shells.

In this extract from Torda et al[19] Professor Torda has kindly updated certain recommendations:

"There is no specific antitoxin or antidote to TTX. Treatment therefore must be largely symptomatic and empirical.

"On suspicion of TTX ingestion, or on appearance of symptoms, vomiting should be induced, provided there is no difficulty in swallowing or weakness of the

voice. The latter symptoms suggest that hazard of aspiration of vomitus exists. If that is the case, gastric lavage can be carried out after intubation with a cuffed endotracheal tube. Whether gastric lavage is indicated more than 3 hours after ingestion is uncertain. As gastric emptying may be slowed by TTX, it is probably safer to do it than not.

"Where symptoms are confined to paraesthesia and weakness not affecting the muscles of respiration and deglutition, probably no effective treatment is possible. Light sedation with benzodiazepine or other sedative in anxious patients may be desirable, and close observation must be maintained at least until symptoms begin to recede. In the presence of any difficulty in swallowing, oral intake must be suspended and an intravenous infusion set up to maintain hydration and prevent hypotension.

"Any difficulty in dealing with saliva or respiratory secretions is an indication for endotracheal intubation, as is increasing dyspnoea, a rising respiration rate, or progressive elevation of the arterial carbon dioxide tension. There are obvious dangers in oxygen administration to patients with failing ventilation, as it may mask a dangerous situation allowing gross elevation of carbon dioxide tension before the critical nature of the patient's respiratory status becomes obvious. We suggest that ventilatory insufficiency in patients breathing air is an indication for assisted or controlled ventilation rather than oxygen supplementation. (The importance of oxygen in resuscitation is of course not disputed). The short natural course of TTX intoxication makes tracheostomy unnecessary. Our preference is for nasal intubation, with a cuffed polyvinyl chloride tube. It is easier to fix firmly in place, causes less discomfort and is less liable to accidental dislodgement than an oral tube. To ease the passage of the tube and reduce the incidence of epistaxis, the nose is sprayed with 0.25% Neosynephrine. As these patients are conscious (unless acutely hypoxic), intravenously administered anaesthesia (eg. diazepam, 0.2 to 0.4 mg/kg) is desirable. As paralysis is likely to become complete, controlled ventilation is preferable to assisted, unless the ventilator used is able to 'take over' automatically if the patient fails to trigger it. Adequate humidification of the inspired gases is necessary. Initial sedation is helpful to allay the patient's fears, but recognition that he is likely to be conscious and full explanation of all procedures are even more important.

"Fluid administration is regulated according to arterial blood pressure, central venous pressure and urinary output. As TTX causes vasodilation (as well as central cardiac depression in larger doses), it is rational to infuse a plasma expander until urine output (measured by catheter and 100 ml collecting burette) exceeds 40 ml/hr. However, if the central venous pressure rises without restoration of urine output, inotropic agents such as dopamine or dobutamine are indicated. After urine output has been restored, 2 l of dextrose-saline will supply maintenance fluid and sodium for 24 hours in the case of an adult. Potassium chloride 40 to 60 mEq per day should be added after it has been ascertained that the patient is not hyperkalaemic.

"Nursing care includes hourly turning of the patient and attention to pressure areas. The eyes are protected by Vaseline gauze pads. Pulmonary changes of constant volume ventilation are minimized by hyperinflating the lungs 5 times every hour or by end expiratory pressure of 3 or 4 mm Hg. Tracheal toilet is performed only as needed.

"The electrocardiogram is monitored. Atropine is said to be ineffective in preventing TTX-induced bradycardia. The effect of isoprenaline on bradycardia or conduction disturbances appears to be uninvestigated. Complete atrio-ventricular dissociation may be an indication for insertion of a temporary pacemaker. Although TTX is effective in treating ouabain-induced arrhythmias, the effect of cardiac glycosides on the cardiotoxicity of TTX is unknown."

Anticholinesterase Drugs

In Case 2,[19] edrophonium given during recovery seemed effective in accelerating the return of muscle power. The improvement appeared to be maintained after the administration of neostigmine. The authors stated that this observation was at variance with previous reports and therefore needed confirmation. Their paper stimulated Sorokin[20] to report that he considered that anticholinesterases had produced clinical improvements in cases of Puffer fish poisoning he had managed in Fiji. Likewise, in the case of paralysis due to the toxins of either H maculosa or Conus shells the use of an anticholinesterase would appear worth consideration.

LABORATORY INVESTIGATIONS

Japanese workers have used several methods to analyse post mortem gastric and intestinal contents. Tsunenari et al[21] used a mouse bioassay to demonstrate the presence of TTX in the gastrointestinal tract and vomitus of an 80-year-old man who was fed Puffer fish ovaries by his daughter. Suenaga and Kotoku,[2] using gas chromatography, could detect TTX in both the gastric contents and the serum in five out of eight persons who died after eating Puffer fish. The concentrations detected ranged from 0.9 to 5.5 μg/g in stomach contents to 0.12 to 0.35 μg/ml in serum. These workers made the observation that their method had better reproducibility and was more objective than the biological assays.

CASE HISTORIES

Case 1[22]

"On December 6, 1950, a boy, SCM, aged eleven years, living at Castle Forbes Bay, was given five small fresh fish which had been beheaded and cleaned but not skinned. He took these fish home and his mother cooked four of them, the cat having stolen one. The fish were placed before SCM and his father, but the latter, after tasting a small portion, decided he did not fancy the fish and he gave his share to the cat. SCM asked his mother to cook his two fish a little longer, after which they were eaten at 6 pm.

"Tea was concluded at 6.20 pm and the boy went on his bicycle to do a message. When he returned at 6.40 pm he complained of feeling "queer"; his legs, feet and hands were numb and his body seemed to be "floating". Within five minutes he complained of feeling cold and said he could not move his arms or legs. His lips felt stiff. He was given an aspirin, but could not swallow it. The boy had lost consciousness by 7.30 pm and was moribund when examined by Dr HP Coats at Huonville at 7.50 pm. He died soon after 8.00 pm.

Post Mortem Findings

"Apart from some pulmonary oedema and some congestion of the organs, no abnormality was detected. The routine examination of histological sections did not assist in any way.

Further Investigations

"The fish immediately came under suspicion, in spite of the father's statement that they were "mountain trout". The suspicions were strengthened by the action of the family cat, for at 9.30 pm it was seen to be paralysed in the back legs, and it was found dead in the morning.

"A warning was issued to parents in that area, and Hobart detectives, using explosives, were able to obtain some more of these fish from the same tidal creek. They were identified by Dr Pearson, of the Tasmanian Museum, as belonging to the family Tetraodontidae puffer fishes having the teeth of each jaw coalesced into two tooth plates. The genus is abundant in species and numbers. The species in this case, Speroides liosomus, is fairly common in Tasmanian waters and sometimes enters tidal creeks, possibly for spawning.

"Aqueous extracts were made from several organs of the fish as well as from the stomach contents of the boy and the cat. All were found to contain a potent and fast-acting toxin when injected subcutaneously into guinea-pigs. A few drops of an aqueous extract of toadfish skin caused paralysis and death in a small guinea-pig in a matter of minutes.

"Police inquiry revealed a further interesting fact. The day before the tragedy three similar fish were caught and fed to three cats belonging to another family. The cats soon became sick, vomited and later died. Some hens picked at the vomited material, and as a result one died and two were seen to be paralysed. (It is not recorded whether anyone ate the dead fowl)."

Case 2

This is the most comprehensive account in the modern literature. The authors state that the boy's life was saved by the prompt and competent actions of Spivey at the Shoalhaven District Hospital.

"The patient, a healthy boy of 14 years, was on a camping holiday with his parents and two brothers, some miles south of Nowra (NSW). Fishing off some coastal rocks the family had caught over 20 small puffer fish, which were cleaned, gutted and left soaking in sea water overnight. The following day they were boiled in sea water and served to the family just before midday. One fish, different in appearance from all the others, was eaten by our patient. (The fish was later identified as Amblyrhynchotes richei).

"As the meal was being prepared a young, injured crow or magpie, which the family was rearing was given one fish. Shortly after, the bird was seen staggering about in the grass; it then fell, lay with its wings twitching and died in a few minutes.

"As the weather had deteriorated, the family broke camp after lunch. When the packing was completed, our patient complained of numbness in the tongue, a feeling of swelling in the lips and a general feeling of lightness. He sat in the car and they began their return towards Nowra. Some minutes later (perhaps half to three-quarters of an hour after the meal) he vomited.

"After his recovery, he prepared for us a written account of his experience from which we quote verbatim:

> '... Then I started to lie down feeling weak, tired and miserable with that funny up and down sensation if I moved. It became so critical that I couldn't be bothered with anything and I just lay there trying to expand and contract the diaphragm. My father tried giving me milk ... but I couldn't swallow. I could just move my mouth and I was cold, very cold ... I was losing air fast. It was slow and painful. I thought of panic but I became quite paralysed and then unconscious ...'

"The drive was about 15 miles. On arrival at the Shoalhaven District Hospital he was cyanosed and apnoeic. He was ventilated, intubated and sent on to Wollongong Hospital. We quote again from the patient's account:

> '... The next thing I remember was being in the ambulance receiving air and breathing. I could hear them talking but I couldn't move or anything. I was completely conscious ... and I really heard them. They were laughing and chattering and they even played with the hooter ...'

"At Wollongong on arrival he was described as " ... unreactive. Had flaccid paralysis and is areflexic. Pupils fixed and dilated.' His pulse rate was 80 blood pressure 130/80 mmHg, fundi normal. The electrolyte estimation and chest X-ray examination results were said to be normal and blood gases "showed resp. alkalosis only". He was catheterized, an intravenous infusion was commenced and he was given dexamethasone 2 mg intravenously and ampicillin 500 mg. He was thought to have suffered anoxic damage. Arrangements were made for his transfer to the Respiratory-Intensive Care Unit of Prince Henry Hospital where he arrived at 9.30 pm.

"Although he had no memory of events at Wollongong Hospital, he remembers part of the second journey clearly:

> '... I couldn't feel anybody touching me. One of the men in (the) front of the ambulance asked the nurse how I was and she said I still looked worse ... (arriving at Prince Henry Hospital) ... Again I heard them chattering for they were trying to find their way in where we were supposed to go. He stopped two times and on the third try they took me in. All I could feel was that funny up and down feeling ...'

"On arrival he was flaccid and areflexic. His pupils were fixed, central and dilated. The pulse rate was 90, blood pressure 120/80 mmHg. His hourly urine output varied from 25 to 200 ml, passing a total of 1340 ml from the time of catheterization in Wollongong to 8 am the following morning. His PaC02, by modified rebreathing technique, was 43 torr.

"He was given alternating 500 ml flasks of Hartmann's solution and 0.18% saline in 4% dextrose at the rate of 2 1 in 24 hours, with 10 mEq of potassium chloride added to each flask. Ventilation was continued with a Bird Mk VII ventilator driven by air and humidification

was supplied by a Fisher and Paykel heated humidifier. "Rebreathing" carbon dioxide estimations were done every 2 hours during the night. The ECG was monitored by oscilloscope. (No further corticosteroids or antibiotics were given.)

"His consciousness remained obviously clear:

> '... can't remember much at all except for hearing voices in the distant but it was only faint ... but I heard nurses later on and they were trying to talk to me specially one who said good morning and good night. I could also hear surgeons talking mumble jumble ... I tried to move and talk but that was impossible. I felt them spraying stuff on me (silicone aerosol) which turned from hot to cold around my legs and body. They usually told me what they were going to do ... it was terrible because they opened my eyelids every now and then and I found out I could see but just couldn't open my lids. They always used a torch which was very disturbing ...'

"At 6 am movement of the eyelids was noted. The patient could make no other movements, but communication was established:

> '... As time passed I always tried to move. Soon I was able to keep my eyes an eighth to a quarter inch open until I could finally open them. It was nice to see, believe me. I was able to communicate a bit. Doctor asked me if I could feel anything, then he pinched my ear, so I closed my eyes which meant yes. But at least they knew if I was with them or not ...'

"At 8 am he could move his lips and tongue. At 10 am he could move his limbs feebly and a vital capacity of 600 ml was measured with a Wright respirometer. The $PaC0_2$ (rebreathing) was 44 torr. A trial of unassisted respiration was begun. At 11 am he could lift his limbs against gravity, the vital capacity was 800 m., and the $PaCO_2$ was 34. The pattern of respiration resembled that of partial neuromuscular block, with tracheal tug, use of the accessory muscles and some intercostal recession. The respiration rate was 16 to 20 per minute and the patient reported experiencing no respiratory distress. Therefore the endotracheal tube was removed ('... soon you all gathered around and you took the darn thing out ...').

"As respiration still resembled that seen after incomplete reversal of neuromuscular block, a test injection of edrophonium, 10 mg, was given slowly, intravenously. The vital capacity improved to 1000 ml. Tracheal tug, use of accessory respiratory muscles and intercostal recession ceased. A further 10 mg given 15 minutes later resulted in no further increase of vital capacity or other detectable effect. Atropine 1.2 mg and neostigmine 2.5 mg were given. At 1.45 pm., the patient's vital capacity was 2400 ml, normal for his size.

"The cardiac monitor, with the electrodes in a lead II configuration did not show any abnormalities, at any time. A 12 lead ECG and 8 channel EEG taken about 9 am showed no abnormality. The pupillary light reflex was first detected after 8 am, more than 2 hours after eyelid movements were noted and consciousness of the patient established.

"The urinary catheter was removed. Unrestricted oral fluids were allowed 3 hours after extubation as the patient was then able to cough effectively. The following day a free diet was allowed and the patient was allowed out of bed. He was a little unsteady on his feet, but able to walk unassisted. The following day he was discharged from hospital completely recovered, as far as we could ascertain.

"The other members of our patient's family who shared his meal fared better. His father, who did not vomit, developed paraesthesiae, weakness of all limbs and neck, weakness of the voice and difficulty swallowing. He was hospitalized for two days and made an uneventful recovery. The patient's two brothers vomited after eating the fish. They both escaped with paraesthesia only. His mother did not develop symptoms at all."

TETRODOTOXIN POISONING IN ANIMALS

Cats sometimes eat Puffer fish and they may rapidly succumb to paralysis and respiratory failure. Like most other animals, they tend to vomit some of the meal. Milder cases may show weakness and incoordination. Deaths in birds and cats which have been fed Puffer fish have been mentioned in Cases 1 and 2. Atwell and Stutchbury[23] described two cases of Puffer fish ingestion in cats. Both presented with gross mydriasis, flaccid paralysis, tachycardia and depressed respiration. Significant improvement was produced in one cat by an intramuscular injection of 210 mg of etamiphylline camsylate. Both cats slowly recovered over a 24 hour period

Differential Diagnosis

In the coastal region of Queensland, Atwell and Stutchbury[23] list P texilis and I holocyclus envenomations as alternative diagnoses which should be considered, apart from primary or secondary neurological lesions.

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

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

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NEW ZEALAND CHAPTER OF SPUMS MEETING NOVEMBER 1985

A CASE OF CEREBRAL AIR EMBOLISM

Peter Robinson and Allan Sutherland

This 23 year old diver was referred to the Royal New Zealand Navy Hospital (RNZNH) on 24 October 1985, 3 days after her diving accident.

On the previous Monday evening, the 21st, she had made a rapid ascent from the base of a swimming pool while undergoing training with scuba. She had been practicing face mask retrieval and was breathing through scuba apparatus when things went wrong. She panicked and made a rapid ascent holding her breath. Immediately after the incident she was cold and frightened. With some reassurance, everything seemed to settle down. On her way home, she noted that her left middle finger was stiff, swollen and tense and later on that evening, it turned a dusky colour. Next day her left arm was cold and aching in the shoulder region and the finger was still a dusky colour.

Two days later she could not sleep because of the pain and aching in her left arm and shoulder. The finger had not improved, her left arm was cold with some altered sensation and clumsiness. There was no shortness of breath, no pleuritic pain and no precipitating factors which made the pain worse. At this stage on the evening of the 23rd she went to North Shore Hospital where several doctors looked at her, examined her arm only and x-rayed her hand, after which they diagnosed a case of arterial gas embolism, reassured her and sent her home.

She reached the RNZNH 24 hours later, on the Thursday evening, having mentioned to her diving instructor at Shore Watersports Ltd that she was having a few