CHAPTER 5

MARINE ANIMALS THAT ARE POISONOUS TO EAT

(TOXIC)

Barracouta Poisoning
Bacterial and Viral Food Poisoning
Ciguatera Poisoning
Crustacean Poisoning
Hallucinatory Fish Poisoning
Mercury Poisoning
Pollution
Puffer Poisoning
Scombroid Poisoning
Seal Liver Poisoning
Shark and Ray Poisoning
Shellfish Poisoning – Acute Yellow Atrophy of Liver
Shellfish Poisoning – Allergic
Shellfish Poisoning – Gastrointestinal
Shellfish Poisoning – Paralytic
Turtle and Tortoise Poisoning

BARRACOUTA POISONING

SPECIES : *Leonura atun, Lepidocybium flavobrunneum*
SYNONYM : Gempylotoxic poisoning

The diarrhoea occasionally produced by eating these fish is probably the result of a direct effect on the bowel. No abdominal cramps or generalised symptoms are found.

TREATMENT

1. Lomotil (diphenoxylate with atropine) 4 tablets immediately, with 2 after each bowel motion (maximum = 12 per day).
2. Other anti-diarrhoea medications, e.g. Mist opii, Kaomagma, etc.
3. Attention to fluid and electrolyte status.

BACTERIAL AND VIRAL FOOD POISONING

This can occur with prepared fish foods, as it can with all other foodstuffs. As it is not specific or characteristic of fish foods, it is not dealt with further, and reference should be made to the established textbooks of medicine. The types of outbreak that have been reported in association with marine foods include:

- Botulism
- Cholera
- *Escherichia coli* gastroenteritis
- Infective hepatitis
- Poliomyelitis
- *Staphylococcal* gastroenteritis
- Typhoid and paratyphoid fever
- Viral gastroenteritis.

Many other infectious agents can cause disease by contaminating drinking or bathing water, without the necessity of being carried in a marine animal. They include a variety of infectious organisms whose description is also beyond the scope of this text – such as hookworm, hydatids, giardiasis, amoebiasis, schistosomiasis, pathogenic algae, etc. Amoebic encephalitis from *Naegleria aerobii* is probably the only disease of this nature not well documented in the medical texts.

CIGUATERA POISONING

This term is derived from the original "cigua" – poisoning due to the ingestion of a marine snail by early Spanish settlers in the Caribbean. It is now known to be much more widespread and is the commonest form of fish poisoning in the Australian area.

DISTRIBUTION AND TYPE OF FISH

Most of the tropical and temperate regions of the South Pacific are susceptible to disease. The most southern areas of Australia reported to have affected fish are Brisbane on the east coast and Geraldton on the west (Lat. 28°S). Evidence from other parts of the world suggests that this fish poisoning could occur as far south as 34° Latitude (Sydney – Perth). Certain fish caught on the Barrier Reef, e.g. the chinaman, the red bass and the paddletail, are particularly dangerous and are not accepted by most fishing co-operatives for sale. Occasional problems are encountered with coral trout, Spanish mackerel, reef cod, barracuda, emperor, groper, surgeonfish and yankee whiting. Rarely the Maori wrasse and the barramundi are incriminated.

Apparently any large fish is a potential danger, but especially if it inhabits reefs. Pelagic school fish are least affected. Ciguatera poisoning occurs sporadically and unpredictably, and may gradually spread from one area to another.

SEASONAL INCIDENCE

Sub-tropic (Brisbane to Gladstone) – especially September and October.

Tropics (north of Gladstone) – especially autumn and winter.

GENERAL

This is one type of Ichthyosarcotoxic poisoning. The victims are often anglers or their families, celebrating a particularly large "catch". The affected fish is often the largest of the batch caught, and is usually of the carnivorous type. The poison "Ciguatoxin" probably originates in a benthoic organism, is transferred to herbivorous fish, and thence to carnivorous fish, where it is accumulated. The liver and testes are particularly toxic, as are other viscera, while the flesh is less so. The freshness of the fish has no bearing on its toxicity, nor does cooking. The toxin is heat stable and partly water soluble. It is probably a triglyceride (mw = 1500 approx.), and includes a lipid containing a quaternary nitrogen atom, with anticholinesterase activity in vitro, and not dissimilar to organophosphorus (dieldrin) poisoning in some of its effects. The anticholinesterase effects are doubtful "in vivo", and effects of the toxin in experimental animals suggest a "cholinomimetic" action in which stimulation of autonomic ganglion seems to be the most obvious initial effect.

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The demonstrated pharmacological effects of the toxin include: a diminished respiratory rate with increased amplitude, irregular and Cheyne-Stokes respiratory patterns prior to apnoea; a diminished blood pressure which returns to normal in the less severe cases, bradycardia or arrhythmias resulting in tachycardia; partial neuromuscular blockage. It is noteworthy that in such experiments the dose required to produce failure of spontaneous respiration is about half that required to produce cardiovascular collapse, hence the importance of mouth to mouth respiration as a first aid measure and of positive pressure respiration during medical treatment. Ciguatoxin has been reported to cause demyelination in peripheral nerves, spinal cord and brain in animals. It depolarizes excitable membranes "in vitro" at low concentrations by increasing resting Na^{+} permeability and this depolarization has been shown to be antagonized by raising the Ca^{++} concentration of the bathing medium. While it is not yet clear that this toxin's primary action is mediated by its effects on sodium permeability, there is no valid reason to believe that "in vivo" poisoning is produced by anticholinesterase action of this toxin.

Gymnothorax toxin is absorbed after ingestion of eels and the result is identical to ciguatera poisoning (Halstead), as is paralytic shellfish poisoning from eating affected shellfish (Russell). The "ciguatoxin" isolated from eels has the empirical formula $C_{35}H_{65}NO_8$ and is very unstable. Moray eels are also incriminated as having ichthyohaemotoxic poisons, but this has not yet been reported in the Australasian region. The eel form of ciguatera poisoning may result in prolonged paralysis, over one week.

CLINICAL FEATURES

ACUTE

- Usually symptoms are noted from 2 to 12 hours (0—30 range) after ingestion of the food. Severe cases may occur earlier and mild cases may be precipitated later by alcohol ingestion.
- General weakness and dull aches in limbs (especially knees) and head.
- Paraesthesia. Tingling, then numbness, around the lips, tongue, mouth and throat, face, hands or feet, noted in half the cases, and lasts up to 7 days. A severe pruritis, occurring days after the ingestion, may spread from the extremities and have nocturnal exacerbations leading to insomnia.
- Metallic taste, dry mouth, thick tongue, aching of teeth or tightness of muscles around mouth.
- Muscle pains, weakness and/or cramps. These pains differentiate this disorder clinically from Puffer fish poisoning.
- Anorexia, nausea, vomiting, diarrhoea and/or abdominal pain, in half the cases. This may last up to 4 days.
- Red, itchy rash over palms of hands and soles of feet and sometimes with weals (a punctate erythema with vesicles or urticaria). It is severe for 2—3 days and subsides after 4—5 days. The rash is aggravated by alcohol ingestion. Hair and nail loss may occur.
- Reversal of temperature perception — cold articles feel hot and vice versa. This may be present for months, with the drinking of chilled beer producing a burning sensation in the throat.
- Neurological disturbances — insomnia, apprehension, delirium, visual disorders (double vision, dilated pupils, etc.), commencing in lower limbs, inco-ordination and ataxia, occasional athetoid movements, convulsions, coma and death.
- Respiratory failure, with tightness in chest, laboured breathing, cyanosis (bluish colour), etc.

- Cold, clammy appearance with hypotension, cardiac arrhythmias, bradycardia or tachycardia, with extra systoles, etc.
- Serum and red blood cell cholinesterases are normal.
- Death in some non-Australasian series is approximately 10%. In Polynesia the mortality rate may be less than 0.1%.

PROGRESS

In a mild attack the major symptoms may clear in 24—36 hours, although residual weakness, paraesthesia and the reversal of temperature perception may persist for longer

In a severe case the serious manifestations usually subside within the week, although total recovery may take many months or even years. Recurrent burning sensations may also be precipitated by alcohol, nicotinic acid and other vaso active drugs. There may be an associated erythema. The body, face and genitals are often affected, intermittantly, for many months. Insomnia and gastrointestinal symptoms may also persist over this period.

Exacerbation of the illness may follow stress, or the ingestion of certain fish. Immunity does not occur, and it is possible that repeated poisonings may be even more serious.

FIRST AID

1. If the patient is fully conscious, induce him to vomit by inserting fingers in his throat. Syrup of Ipecac U.S.P. 8 ml orally may aid in this. Treat any other potential victims (e.g. others who ate the fish) in the same way.
2. Rest and reassurance.
3. Resuscitation if needed. This will usually be in the form of mouth to mouth respiration (page 197) in those patients who develop severe respiratory distress, unconsciousness with cyanosis (bluish colour), etc.
4. Hospitalisation and observation are always needed until recovery.

MEDICAL

1. First Aid as above. Gastric lavage, or apomorphine 2—8 mg s.c. or other emetics, if the laryngeal reflex is unimpaired.
2. With respiratory involvement, the ideal treatment is to perform endotracheal intubation and control respirations. The use of an endotracheal tube will also prevent the aspiration of vomitus, particularly likely under the conditions of a bulbar paralysis with gastrointestinal symptoms. If this is not achieved, then maintain respirations by any method at your disposal.

   Assisted intermittent positive pressure respiration may be all that is required when there is only a mild impairment. If there is a rising arterial CO_2 level or an increasing respiratory rate, assistance with respiration is required but oxygen supplementation is not needed.

   When there is a more severe degree of respiratory depression with symptomatic distress and/or cyanosis, an increasing arterial CO_2 and a decreasing arterial O_2, it would be prudent to completely control respirations by the use of endotracheal intubation and mechanical ventilation. Monitoring of serial arterial O_2, CO_2 and pH levels is required. The patient should be maintained on the regime for at least 6 hours and then gradually weaned from the respirator over the next 12—24 hours.
3. Ensure fluid and electrolyte replacement and administer medication by intravenous means (record vital signs, serial haematocrit, S.G. electrolytes, C.V.P., E.K.G., urinary output and analyses, etc.).

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4. Symptomatic treatment of convulsions — treated as in status epilepticus.
5. 10% Calcium gluconate i.v. may rapidly relieve neuromuscular and neurological features, and increase muscle tone.
6. Sedation should be achieved with non-respiratory depressants, e.g. diazepam 10 mg i.v. repeated as required. Small doses of opiates may be needed for pain.
7. Steroid cover, e.g. hydrocortisone 100 mg i.v. 6 hourly during the danger period is possibly of value — but is non-specific in its effect.
8. Specific pharmacological treatment is not available. Atropine i.v. may be required to control vagal tone and counter the muscarine manifestations of the toxin. Edrophonium (Tensilon) and neostigmine are successful in some cases, although they had previously been considered as being contraindicated due to the cholinesterase inhibition. Pralidoxime (P.A.M., Protopam) 1—2 g oral, i.m. or i.v. has been proposed — to be repeated as required. This is not yet proven in sufficient experimental trials and has not been of value in clinical practice, despite the theoretical indications. Nikethamide, vit. B, and many other drugs have been suggested, but are best omitted in preference to general medical care. Nicotinic acid preparations have been known to prolong the clinical manifestations for many months.

PREVENTION

1. Do not eat the fish mentioned above.
2. Treat all oversized fish with suspicion.
3. Try out a sample on the neighbour's cat, and observe for a few hours.
4. One approach is to feed the older members of the family first, and if they remain unaffected after several hours, the fish may be fed to the children with safety. Some cultures may reverse this process.
5. If one is compelled to eat potentially harmful fish, then avoid roe, intestines and viscera, or abnormally large fish. Cut the flesh into thin slices and soak it in several changes of water (fresh or salt) for one hour each. Discard the water and do not use it for cooking or drinking purposes. Eat only small quantities, and never eat tropical moray eels.

CRUSTACEAN POISONING

PHYLUM : Arthropoda
CLASS : Crustacea
Crayfish
Prawns
Shrimp
Lobster
Crabs
Yabbies

GENERAL

These arthropods, so well liked amongst our gourmets, appear to be able to induce similar reactions to the shellfish poisonings. Paralytic poisoning (page 192) has been reported, and possibly gastrointestinal poisoning (page 190) may occur following the ingestion of these crustaceans. Probably the commonest form of crustacean poisoning is of the allergic type — with an identical clinical picture to that of the Allergic Shellfish Poisoning (page 188) and requiring the same first aid and medical treatment.

HALLUCINATORY FISH POISONING

DISTRIBUTION AND TYPE OF FISH

This rare disorder occurs in scattered localities, either in epidemics or endemically. One such area is Norfolk Island, off the eastern coast of Australia, where fish which are known locally as the "dreamfish" are ingested and cause troublesome nightmares. Fish that have been incriminated include mullet, goatfish, unicornfish, surgeonfish, rudderfish, damselfish, drummers, rockcods.

The toxin is not destroyed by cooking.

CLINICAL

- There is a latent period which varies between a few minutes and 2 hours.
- Disturbances of vision and hearing vary from misinterpretations (i.e. illusions) to frank hallucinations.
- The disturbed conscious state waxes and wanes for many hours.
- Neurological signs may be transitory, and affect coordination, muscle strength, etc.
- Sleep is disturbed, with nightmares and bizarre dreams.
- Sensations of constriction to breathing, impending death, gastrointestinal and skin disturbances.
- The victim may develop paranoid feelings that people are trying to harm him.
- The symptoms usually terminate spontaneously within 24 hours.
- Symptoms of other fish poisoning may be present (see ciguatera, scombroid, etc.).

FIRST AID

1. Reassurance, in a protective, well-lighted environment, by friends and relatives whom the patient trusts.
2. Give tranquillisers if available, but do not give stimulants (coffee, tea, etc.) or sedatives (alcohol, barbiturates, etc.).
3. Treat physical disorders as for other fish poisonings.

MEDICAL

1. First Aid, as above.
2. Phenothiazines, e.g. thioridazine (Melleril) 100–200 mg statim and repeat p
   Diazepam 10 mg i.v. may also be of value in quieting the patient. As it is not kno
   whether the toxin is related to STP, chlorpromazine is best avoided.
3. Treat other associated fish poisonings on their merits.

PREVENTION

Take note of the local knowledge, otherwise a planned holiday may result in an extend
"trip".

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MERCURY POISONING

SYNONYM : Minamata Disease

GENERAL

Between 1953 and 1960, 111 cases of mercury poisoning were reported in people eating contaminated shellfish and fish from Minamata Bay, in Japan. Forty-three of these people died. The mercury was originally derived from industrial effluent. In Minamata Bay, values as high as 1—10 micrograms per litre were recorded. This was later followed by an outbreak in Sweden, due to ingestion of contaminated fish from fresh water lakes polluted with mercury by paper and pulp mills. Other industries with similar effects include the manufacturers of chlorine, caustic soda, electric batteries, fluorescent lamps, plastics, fungicides and seed dressings. Commercial fishing has been prohibited in parts of the Great Lakes of Canada and some rivers in the United States of America and Sweden, because of this mercury contamination. If the surface water mercury concentration exceeds 0.2 micrograms per litre, significant contamination is present. In the marine environment, the mercury compounds decompose into the inorganic form. It is then converted to methyl mercury, and this is the toxic substance which accumulates in fish. The mercury level is increased in those marine animals which are major predators, e.g. tuna, shark and the larger crustaceans. Paradoxically, the mercury levels of tuna and swordfish caught in 1878 and 1946 and preserved in museums are not much different from those detected today.

Mercury poisoning in man occurs when the blood level reaches 50—100 micrograms per 100 ml, and the safe limit has been estimated as 10 micrograms per 100 ml. This limit is thought to be reached with a daily consumption of 100 micrograms of mercury, which happens to be the content of one 200 gram can of tuna fish contaminated with 0.5 ppm mercury. The World Health Organisation recommendation is for a safe limit is 0.05 ppm. The biological half life of mercury in humans is about 70 days. The neuropathological manifestations of mercury poisoning include cellular degeneration of the cerebellum, basal ganglia, hypothalamus, midbrain and cerebral cortex. Methyl mercury, and other mercurial compounds, may be especially hazardous to the foetus because of the tendency to cross the placenta and become concentrated in foetal blood. Probably the maximum safe level of methyl mercury for pregnant females is 30 micrograms per day.

CLINICAL FEATURES ACUTE

With acute mercury poisoning, pain occurs in the ear, throat and larynx. Abdominal cramps with nausea and vomiting develop with 15 minutes.

Mercury is concentrated in the kidneys, where it damages the tubules. This produces an increased urine flow within the first 2 to 3 hours, but then proceeds to total cessation of urine formation.

The combination of vomiting, dehydration and tubular damage in the kidneys, etc., leads to renal failure and uraemia.

Mercury is also excreted in the colon and produces severe enteritis with bloody diarrhoea and pain.

Death is usually from renal failure and uraemia.

SUB ACUTE

Gradual development of renal failure (oliguria and anuria).

CHRONIC POISONING

The following manifestations may be noted.

- Salivation, sore mouth, diarrhoea, etc.
- Numbness in periphery of legs and arms, progressively extending centrally to include the tongue and lips. Visual fields may constrict.
- Weakness of the muscles, with tremors, jerking, rigidity, etc. In late cases the muscles become wasted.
- Irritability, agitation, moodiness, depression and sometimes overactivity.
- Insomnia, confusion, delirium and headaches.
- Inco-ordination, parkinsonian tremor and other extrapyramidal movements, vertigo and disturbances of gait.
- Difficulty with speech, swallowing, vision and hearing.
- Neurological evidence of damage to cerebellum, cerebrum, extra-pyramidal and mid-brain areas.
- Death may be due to the neurological involvement, starvation or secondary infections.

The biological significance of small concentrations of mercury are not known, however has been suggested that general vitality and reproductive capacity may be affected.

MEDICAL TREATMENT

1. Binding of the mercury by the use of parenteral dimercaprol (BAL) or penicillamine is recommended. The latter is effective for both inorganic and phenyl compounds.
2. Symptomatic treatment along customary medical lines for the clinical manifestations.
3. Neurological damage may be permanent.

PREVENTION

1. Elect only local regional and national governments which have forward planning and constructive policies on environmental pollution.
2. Regular sampling and chemical analysis of fish from waterways.
3. Rigid control over the release of industrial waste effluents into the sea.

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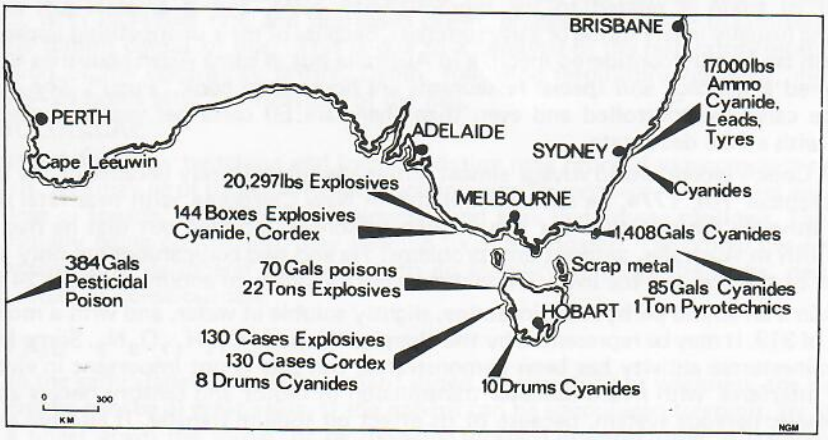
POLLUTION

Over the last decade there has been an awareness of the danger of polluting the waterways and seas with industrial wastes and chemicals, pesticides, radioactive materials, mercury, etc. These must bring, in their wake, a reprehensible and diverse array of dangers to those populations reliant on the seas for their existence. It is estimated that 20,000 tons of DDT is present in marine life, although it is not known whether this is likely to cause clinical manifestations in humans who consume the affected fish.

The outpouring of inadequately treated sewage and solvents from industrialised societies is a cause of concern to marine ecologists and public health authorities alike. Most proposals made by government departments seem designed to allay apprehension and divert blame, more than to remedy a tragic situation. Nor is this limited to one country. The Minamata disease of Japan 1953–60 (page 173), the French atomic tests in the Pacific (1970–74), the ditching of radioactive atomic waste of the U.S.A. (1970–71), and the sewerage and solvent pollution of Australian bays and sea shores ensures that this attitude has no national boundaries.

Australia is one of the 71 members of Intergovernmental Oceanographic Commission and is involved indirectly (very indirectly until this time) in a worldwide anti-pollution regime. At about the same time that this information was made known to the Australian public (*The Australian*, 11 October, 1971), the Great Barrier Reef was being destroyed by the *Acanthaster planci,* unencumbered by governmental interest, the Australian sea food industry was given 15 years of life before pollution wiped it out "if present trends are not checked" (*The Australian*, 7 September, 1971), and Botany Bay, the cradle of the Australian settlement was given 5 years before the ecology was irreparably ruined (*The Daily Mirror*, 4 September, 1971).

The sea, which holds both the past and the future of mankind, deserves respect and thought by those who would exploit it. The mineral, food and energy potential may be of enormous benefit, and the judicious extraction of these valued materials can be performed without the catastrophic effects that are now being perpetrated under the guise of industrialisation. To despoil the oceans with solvents, plastics and other non-biodegradable materials is tantamount to building a sewerage pit for our children and their children to live in. We are fouling the beauty of the undersea world, slaughtering its inhabitants and destroying our national heritage.



PUFFER POISONING

TYPE OF FISH INVOLVED : Toadfish, Puffer, Sharp nosed Puffer, Blowfish, Globefish, Porcupinefish, Swellfish, Toado, Balloonfish, Sunfish and many others.

ORDER : Tetraodontiformes (Plectognathi)

SYNONYM : Tetraodotoxic Poisoning, Fugu

GENERAL

This occurs following the ingestion of puffers, ocean sunfishes or porcupine fishes. The name "Puffer" comes from the fish's ability to inflate itself by taking in large quantities of air or water. They are regarded as poisonous throughout the world. Some can inflict crushing bites, but are not venomous. The body has no true scales, but may have projecting spines or armour-like plates. The Tetraodontoidei have only 4 fused teeth.

They are chiefly tropical or sub-tropical fish, however they do extend all around Australia. The toxin is the most lethal of all the ichthyosarcotoxic types. It is concentrated mainly in the gonads (especially around the ovaries), the liver and the intestines. Lesser amounts occur in the skin, but the body musculature is usually free of poison. The appearance and amount of toxin is related to the reproductivity cycle, and is greatest just prior to spawning (usually in late spring or early summer). Because of their unappetising appearance these fish are usually considered inedible in Australia but in some Asian countries they are considered a delicacy and special restaurants are licensed to cook "Fugu". The cooking must be carefully controlled and even then there are 50 cases per year reported from Japan, with a 60% death rate.

Captain Cook's second world voyage almost terminated prematurely because of the puffer. On September 7th, 1774, he sampled this fish in New Caledonia, with near fatal results. Cook, although warned that the fish may be poisonous, pointed out that he had eaten similar fish in Australia, without any problems! He and two companions ate only a small amount of the roe and the liver. The victims were fortunate to endure the next 24 hours.

The toxin is an amino perhydroquinazoline, slightly soluble in water, and with a molecular weight of 319. It may be represented by the chemical formula $C_{11}H_{17}O_8N_3$. Some in vitro anticholinesterase activity has been demonstrated, but this is not important in vivo. The poison interferes with neuromuscular transmission in motor and sensory nerves and the sympathetic nervous system, because of its effect on sodium transfer. It also has a direct

depressant effect on medullary centres, on skeletal muscle (reducing its excitability) and on intracardiac conduction and the contractile force of the heart. It does not cause depolarisation of nerve and muscle, and does not reduce the sensitivity of the end plate to acetylcholine. Hypotension is due either to the effects on the preganglionic cholinergic fibres or the direct effect on the heart. Respiratory depression may be due to the effect on nerve, neuromuscular function, musculature or a medullary action.

In animal experiments, oral introduction produces lethargy, muscular weakness, inco-ordination and ataxia. Paralysis is observed first in the hind limbs and later in the fore limbs. Deep reflexes are lost and convulsions may occur. The respirations become laboured, and the animal becomes cyanosed. Retching and vomiting are often severe. In cats the oral LD_{50} of the toxin is in excess of 200 mcg/ kg body weight. The minimal lethal dose in mice is approximately 8.0 mcg/kg. Death in mice can be prevented by the injection of sufficient quantities of cysteine to bind the toxin, within 10—30 minutes of ingestion.

Tetrodon fish are immune to the effects of tetrodotoxin.

CLINICAL FEATURES

GENERAL

- The onset and severity of symptoms vary according to the amount of the toxin ingested.
- Within 5 to 40 minutes (or as long as 3 hours) the patient may note weakness, malaise, pallor, dizziness, inco-ordination and ataxia. During this time paraesthesia of the lips and tongue (described as tingling or prickling) may extend to the extremities or become generalised.
- Increased salivation, sweating, chest pain and headache may also be noted.
- Gastrointestinal symptoms of nausea, vomiting, diarrhoea and pain are sometimes observed.
- A decrease in temperature, blood pressure and pulse rate may also be noted.
- A haemorrhagic diathesis has been reported, with bleeding into the skin and mucosa, haemorrhagic blister formation and desquamation (peeling of the skin). Vomiting of blood and other bleeding tendencies are possible.
- The death rate is approximately 60%.

RESPIRATORY

After the paraesthesia, respiratory symptoms dominate the clinical state. Respiratory distress, with increased rate and decreased depth, proceeds to severe breathlessness and cyanosis (bluish colour of lips). This is due to a paralysis of the respiratory muscles and a depression of the respiratory centre. Death, following respiratory paralysis, may occur within 6—24 hours after the ingestion of the fish.

NEUROLOGICAL

Generalised muscular twitching and inco-ordination may proceed to a complete muscular paralysis. This may be of the bulbar type involving interference with speech and swallowing (with loss of speech, difficulty in swallowing, and later inability to swallow). The pupils, after initially being constricted, may become fixed and dilated. The bulbar paralysis may then extend to a peripheral paralysis, with an inability to move the limbs, despite the victim retaining consciousness.

FIRST AID

1. In the early stages, before the patient shows any paralysis or weakness, the use of an emetic may be of great value. If the patient can be encouraged to vomit, by inserting a finger down the throat, or by ingesting mustard in warm water, it is possible that

much of the poison will be removed in the vomitus.

2. In cases of paralysis resulting in respiratory distress, the use of mouth to mo... respiration (page 197) may be lifesaving, but ensure that the airway is clear ... vomitus, and that the patient is not allowed to develop a bluish colour (cyanosis...

3. Obtain medical assistance.

MEDICAL TREATMENT

1. In the early stages, before evidence of paralysis occurs, the insertion of a tube f... gastric lavage seems axiomatic. Preparation must be made against the possibility ... aspiration of stomach contents into the bronchial tree, i.e. full anaesthetic readine... and efficient suction. Apomorphine, 2–8 mg s.c., may be of value, as may oth... emetics such as Ipecacuanha syrup.

2. With respiratory involvement, the ideal treatment is to perform endotrache... intubation and control respiration. The use of an endotracheal tube will also prev... the aspiration of vomitus, particularly likely under the conditions of a bul... paralysis with gastrointestinal symptoms. If this is not achieved, then mainta... respiration by any method at your disposal.

   Assisted intermittent positive pressure respiration (e.g. with a Bird respirator) may b... all that is required when there is only a mild impairment. If there is a rising arte... CO_2 level or an increasing respiratory rate, respiratory support is required, bu... oxygen supplementation is not needed.

   When there is a more severe degree of respiratory depression with symptoma... distress and/or cyanosis, an increasing arterial CO_2 and a decreasing arterial O_2 ... would be prudent to completely control respirations by the use of endotrache... intubation and mechanical ventilation. Monitoring of serial arterial O_2, CO_2 and p... levels is required. The patient should be maintained on this regime for at least 6 hou... and then gradually weaned from the respirator over the next 12–24 hours.

3. The presence of fixed dilated pupils in an apnoeic unresponsive patient is expect... in this disorder and is not a sign of "cerebral death", merely of temporary paraly...

4. Specific pharmacological therapy is not available. Atropine may control vagal to... Intravenous calcium gluconate (10%) has been recommended as a non-specific sti... lant which could augment the action potential of neurones. Anticholinestera... such as pyridostigmine or edrophonium may have a beneficial effect after th... paralysis commences to abate. They have no value during the acute phase. Cardi... respiratory stimulants have been suggested, and these have included caffe... nikethamide, cardiasol, etc. There is little justification for these, although unc... some animal experiment conditions, both lobeline and pentylenetetrazol have ha... some value.

5. Ensure fluid and electrolyte replacement and administer medication by intraven... means (record vital signs, serial haematocrit, S.G., electrolytes, C.V.P., urinary ou... put and analysis, etc.). Plasma expanders, in the initial stages, may be required t... counter the vasodilation. With the possibility of haemorrhages, cross matched blo... should subsequently be made available.

6. Monitoring procedures such as E.K.G., E.E.G., respiratory parameters, etc., wou... seem indicated. Serial estimations of arterial blood gases and pH should be the ba... on which respiratory therapy is decided. With the development of pulmona... oedema, it may be necessary to increase the inspiratory pressure up to 60 cm ... water.

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7. Because of the possibility of consciousness being retained in the absence of skeletal or respiratory movement, the periodic administration of a minor tranquilliser such as diazepam would seem humane, and the patient should be given continuous reassurance and explanation. Discussions between the medical personnel should be guarded, in the patient's presence.
8. On general principles, i.v. steroids (e.g. hydrocortisone 200 mg, repeated as indicated) could be beneficial in the severe cases.
9. General nursing care, with attention to pressure areas, eye and mouth toilets, etc., is axiomatic in these paralysed and debilitated patients.

PREVENTION

1. Do not eat scaleless fish unless they are known to be harmless.
2. If one is forced to eat fugu in Japan, it should be purchased at a first class restaurant with a licensed "puffer" cook. All the visceral organs and skin from the fish must be removed. Although the testes are usually non-toxic, it must be realised that these can be confused with the highly toxic ovaries.
3. Cooking by frying, stewing, baking, boiling, etc., does not inactivate the toxin.
4. If one is forced to eat these fish to survive, the following is recommended. The fish should be eviscerated and only the musculature selected. The meat should then be cut or torn into small bits and soaked in water for at least four hours. The fish should be kneaded during this time and the water changed at frequent intervals. The toxin is partly water soluble, and this soaking may remove it. Do not eat more than is required to maintain life.
5. Exposure to the toxin does not produce immunity.

"These ye shall eat of all that are in the waters; all that have fins and scales shall ye eat; And whatsoever hath not fins and scales ye may not eat; it is unclean unto you."
Mosaic sanitary law, Deuteronomy 14, 9–10.

SCOMBROID POISONING

SYNONYM : Scombrotoxic Poisoning

There are few problems from this type of fish poisoning in Australia, because of the tendency to eat canned tuna or heavily cooked fish. It is a possible hazard wherever the mackerel-like fishes, tuna, bonito or albacore, are caught and eaten without adequate preparation, and wherever Asian type restaurants (e.g. Japanese) are frequented. It has also occurred in epidemics due to contaminated canned tuna.

GENERAL

These fish, which are normally safe to eat, become poisonous if handled incorrectly. If left for several hours at room temperature, or in the sun, the histidine in their muscular tissues is able to be changed by bacterial action, into saurine – a histamine-like substance. The bacteria involved include *Proteus morganii, Clostridium, Salmonella, Escherichia,* etc.

Laboratory verification of contaminated fish, is obtained by demonstrating a histamine content of 100 mg per 100 g of fish muscle.

CLINICAL FEATURES

- The taste of the fish may be characteristic – "sharp or peppery".
- A latent period of some 20–60 minutes precedes the other symptoms.
- Nausea, vomiting, diarrhoea and upper abdominal distress develops.
- Headache is noted. It is relieved by venous pressure (obstruction on the jugular vein by a medical practitioner) and it diminishes later, if hypotension occurs. Throbbing of the cranial arteries is experienced, and is abolished by transitory carotid pressure on that side (to be applied by a medical practitioner only).
- Palpitations may be associated with rapid weak pulse.
- Dry mouth, thirst, burning sensation in throat, inability to swallow.

Usually the above symptoms are noted in the first two hours, and are followed by:

- Generalised red colour, itching and blister (urticaria) formation over the whole body.
- Face is flushed and swollen, eyes inflamed.
- "Common cold" symptoms.
- Bronchospasm, respiratory distress, cyanosis.
- Fever, chills, tremors.
- Malaise, muscular weakness, pain, a metallic taste in the mouth.

- Cold, clammy appearance with rapid pulse and unconsciousness (syncope) on standing. Cardiovascular shock with hypotension, and EKG evidence of ST depression.
- Death is possible, but unlikely.
- Usually symptoms disappear within 12–16 hours but severe cases can last days.

FIRST AID

1. If the patient is fully conscious, get him to vomit by inserting fingers down the throat. Syrup of Ipecac U.S.P. 8 ml orally may aid in this. Treat any other potential victims (e.g. other members of the group who ate the fish) in the same way.
2. Rest and reassurance.
3. Resuscitation if needed. This will usually be in the form of mouth to mouth respiration (page 197) in those patients who develop severe respiratory distress, unconsciousness with cyanosis, etc.
4. Hospitalisation and observation are always needed until recovery.results.

MEDICAL

1. As in First Aid above. Apomorphine 2–8 mg s.c. may be used as an emetic if laryngeal reflex is unaffected.
2. Steroids are very effective, hydrocortisone 100 mg i.v. is repeated every few hours as required, for 24–48 hours. This counters most of the harmful effects of the scombroid poisoning and is probably the drug of choice – and it may be used with adrenalin, isoprenaline and/or antihistamines.
3. Adrenalin (epinephrine) 1 in 1,000, 0.1 ml per minute s.c. (maximum = 1 ml) or other sympathomimetic drugs such as isoprenaline, will counter most of the symptoms if given early. These are of great value for the respiratory manifestations.
4. Antihistamines have been proposed and may be of benefit. Mepyramine (Anthisan) may be given 100–200 mg i.m. or i.v. and repeated as an oral dose every 6 hours.
5. Ergotamine preparations or the inhalation of 100% oxygen may give symptomatic relief of headaches.
6. Ensure fluid and electrolyte replacement by intravenous means (record vital signs, serial haematocrit, electrolytes, C.V.P., E.K.G., urinary output and analysis, etc.).

PREVENTION

1. Prepare the fish correctly (refrigeration) and do not leave them in the sun, or exposed to room temperature.
2. Eat only canned tuna.
3. If there is any evidence of pallor of the gills, or an odour, or staleness, discard the fish.

"A piscine allergy-like reaction."

SEAL LIVER POISONING

SYNONYM : Hypervitaminosis A

TYPE OF ANIMAL INVOLVED :

- Polar Bear Liver
- Seal Liver
- Porpoise and Dolphin Liver
- Whale Liver
- Shark Liver
- Scombroid (tuna and mackerel)
- Serranid (sea bass and groper)
- Sparid (snapper)
- Arctic Fox
- Husky

This disorder is due to the over ingestion of vitamin A in amounts of 1,000,000 IU. At an estimated concentration of 12,000–14,000 IU/g of bearded seal liver, it is believed that eating 80 g (or 40 g of polar bear liver) can cause this illness. The amount is easily exceeded in one meal. Some whales have also been incriminated. Seal livers can be obtained in the southern states of Australia and New Zealand and they are said to be more toxic in summer than in winter months. The symptoms and signs are reminiscent of arsenic poisoning and this disorder should always be considered in the differential diagnosis.

The validity of this clinical entity is questioned by some workers. Others claim that it is also seen with ingestion of fish and shark livers, scombroid (tuna and mackerel), serranid (sea bass and groper) and sparid (snapper).

Trichinosis has also been reported from eating the flesh of seals, Polar bears, white whales and walruses.

CLINICAL FEATURES

The symptoms may occur ½–7 days after ingestion of a single dose of vitamin A, but presumably subclinical manifestations from multiple ingestions may lead to subacute and chronic hypervitaminosis A.

- headache and drowsiness
- malaise and weakness
- blurred or double vision
- nausea, vomiting and abdominal pain

- central nervous system involvement — with epileptic convulsions, a confusional state, changes of sensation, weakness and paralysis, difficulty in speech and swallowing. Signs of raised intracranial pressure include papilloedema, increased reflexes, extensor plantar responses, etc.
- peripheral neuritis or numbness, tingling and weakness of the limbs.
- excessive perspiration, areas of increased pigmentation, and angular breaks in the skin at the corner of the mouth.
- gross peeling of skin, especially on palms and soles
- in subacute cases x-ray of bones may show periosteal new bone formation and clinically there may be painful or tender swellings on the bone.
- the manifestations of an acute attack may last from a few days to many weeks.
- serum vitamin A is elevated, e.g. in excess of 500 micrograms % (normal = 50—100).

FIRST AID

1. Not applicable. The vitamin A has been absorbed by the time clinical manifestations appear.
2. Enlist medical aid.
3. Rest and reassurance.
4. Stop ingestion of vitamin A foodstuffs.

MEDICAL

1. As above
2. Treatment is entirely symptomatic and includes analgesics, tranquillisers, anticonvulsants, etc., as for any other case of raised intracranial pressure and neurological syndromes.
3. Maintenance of fluid balance and electrolyte state, in those cases with vomiting or diarrhoea, along general medical lines.
4. Investigations include E.E.Gs, serial serum vitamin A estimations, x-ray of bones, etc.

PREVENTION

Do not eat seal livers.

"An arsenic poisoning-like syndrome."

SHARK AND RAY POISONING

SYNONYM : Elasmobranch Poisoning

Any shark (Order: Squaliformes) or ray (Order: Rajiformes) may be involved, especially in the tropic or temperate zones. Those specifically incriminated include:

Carcharhinus melanopterus — Black-tip Reef Shark
Galeocerdo cuvieri — Tiger Shark
Prionace glauca — Blue Shark, Great Blue Shark, Blue Whaler
Heptranchias perlo — Seven-gilled Shark
Carcharodon carcharias — White Shark, White Pointer
Sphyrna zygaena — Hammerhead
Aetobatus narinari — Spotted Duck-bill Ray, Spotted Eagle Ray, and other members of the Family Myliobatidae.

GENERAL

The toxin in this case is thought to be a parasympathomimetic substance. The ingestion of livers from tropical sharks, and from an arctic shark, is the commonest cause of this poisoning. The gonads are also toxic, but the musculature is less so and usually causes only a gastrointestinal upset. If the victim exercises the symptoms may be aggravated. Cooking does not destroy the poison, although it does appear soluble in water. Ciguatera poisoning (page 166) and hypervitaminosis A (page 182) are also possible from the eating of shark. Shark allergy, similar to skin reaction of allergic shellfish poisoning (page 188) is also possible, from contact with sharks, e.g. by fishermen.

CLINICAL FEATURES

- There is usually a latent period of 20 minutes or more after ingestion of the meat. Severe poisonings present more rapidly than minor ones.
- Anorexia, nausea, vomiting, abdominal pain and diarrhoea.
- Headaches, malaise, prostration.
- Joint pains.
- Rapid thready pulse.
- Numbness and tingling around the mouth with burning sensations of the tongue, throat and oesophagus.
- Skin is itchy and may peel off.

- Neurological and neuromuscular symptoms may develop, with weakness, inco-ordination, ataxia, visual disturbances, muscle cramps, lockjaw, paralysis, etc.
- Respiratory distress or failure with cessation of breathing, cyanosis, etc.
- Delirium, coma and death may result.
- Complete recovery may require 5–20 days.

FIRST AID

1. In those cases before any evidence of paralysis has occurred and in which the patient has not already vomited, this should be encouraged. If the patient is fully conscious, get him to vomit by inserting fingers down the throat. Syrup of Ipecac U.S.P. 8 ml orally may aid in this. Treat any other potential victims (e.g. other members of the group who ate the fish) in the same way.
2. Rest and reassurance.
3. Resuscitation if needed. This will usually be in the form of mouth to mouth respiration (page 197) in those patients who develop severe respiratory distress, unconsciousness with cyanosis (bluish colour), etc.
4. Hospitalisation and observation are always needed until recovery occurs.

MEDICAL

1. First Aid as above. Gastric lavage or an emetic, such as apomorphine 2–8 mg s.c., if the laryngeal reflex is unimpaired.
2. With respiratory involvement, the ideal treatment is to perform endotracheal intubation and control respiration. The use of an endotracheal tube will also prevent the aspiration of vomitus, particularly likely under the conditions of a bulbar paralysis with gastrointestinal symptoms. If this is not achieved, then maintain respirations by any method at your disposal.

   Assisted intermittent positive pressure respiration (e.g. with a Bird respirator) may be of value when there is only a mild impairment. If there is a rising arterial CO_2 level or an increasing respiratory rate, assistance with respiration is necessary, but oxygen supplementation is not needed.

   When there is a more severe degree of respiratory depression with symptomatic distress and/or cyanosis, an increasing arterial CO_2 and a decreasing arterial O_2, it would be prudent to completely control respiration by the use of endotracheal intubation and mechanical ventilation. Monitoring of serial arterial O_2, CO_2 and pH levels is required. The patient should be maintained on the regime for at least 6 hours and then gradually weaned from the respirator over the next 12–24 hours.
3. Ensure fluid and electrolyte replacement and administer medication by intravenous means (record vital signs, serial haematocrit, S.G., electrolytes, C.V.P., E.K.G., urinary output and analysis, etc.).
4. Symptomatic treatment of convulsions – treated as a status epilepticus.
5. 10% Calcium gluconate i.v. may rapidly relieve neuromuscular and neurological features.
6. Sedation should be achieved with non-respiratory depressants, e.g. diazepam 10 mg i.v., repeated as required. Small doses of opiates may be needed for pain.
7. Steroid cover (hydrocortisone 100 mg i.v. 6th hourly) over the danger period is possibly of value – but is non-specific in its effect.
8. Specific pharmacological treatment is not available.

PREVENTION

1. First feed a small portion of the flesh or liver to a dispensable test animal (e.g. the neighbour's cat).
2. By thorough washing then drying of the meat, over a considerable time.
3. Repeat washings of the meat, with disposal of the effluent water each time.

SHELLFISH POISONING ACUTE YELLOW ATROPHY OF LIVER

SYNONYM : Asari, Venerupin
PHYLUM : Mollusca
CLASS : Pelecypoda (bivalves) e.g. oysters
SPECIES : *Crassostrea gigas, Tapes (Venerupis) semidecussata*

GENERAL

Specific situations are known (e.g. Lake Hamera in Japan) where molluscs concentrate a hepatotoxin. It is only noted between December and April inclusive, because of the presence during those months of a toxic dinoflagellate *Exuviaella mariae-lebouriae*. The Japanese government has placed the affected areas under quarantine during the danger season. Ordinary cooking does not destroy the poison. As this disease has not yet been reported in the Australasian region, it will not be discussed in any length. The clinical features are those of acute yellow atrophy of the liver – gastrointestinal symptoms, a generalised haemorrhagic diathesis, jaundice and hepatic coma. These occur within 48 hours of ingestion. The toxic effect is dose dependent, requiring 50 shellfish to cause symptoms and 100 to cause death, in an adult.

SHELLFISH POISONING
ALLERGIC

SYNONYMS : Erythematous shellfish poisoning
Oyster allergy

PHYLUM : Mollusca

CLASS : Pelecypoda and others
Mussels
Clams
Oysters
Cockles
Scallops

GENERAL

This appears to be an immediate hypersensitivity reaction to a protein in the shellfish. It is likely that the victim has previously been exposed to the same or similar protein, and to which he has developed an antibody reaction. Symptoms develop after the second and subsequent exposures and are aggravated by exercise, heat and emotion. There may be a history of allergies to other foreign proteins, e.g. hay fever, antitoxins, horse serum, etc.

CLINICAL FEATURES

- There is usually a delay of ½–6 hours following ingestion of the shellfish.
- Diffuse reddening, swelling and itching spreads from the head and neck to the whole of the body.
- Blisters, weals (urticaria) and generalised swelling (oedema) can be extreme.
- Generalised shock state, with pale cold clammy appearance, rapid pulse rate and syncope on standing (fainting), may sometimes occur. Death can occur from this anaphylactic shock.
- Nausea, vomiting and abdominal pain may develop.
- Headache, muscular aches, flushing sensation and mild fever are common.
- Congestion of the respiratory passages may result in laryngeal obstruction, stridor and difficulty in breathing, cyanosis (bluish colour of lips) and death. Less impressive is the congestion and swelling of the nasal mucous membrane, tongue, etc.
- Angioedema is characterised by local oedema (swelling) — and usually involves eyelids, lips, sexual organs, mouth, tongue, larynx or gastrointestinal mucosa.
- Eosinophilia may be present in the blood film.
- Duration of the disease may extend for weeks.

FIRST AID

1. If the patient is fully conscious, get him to vomit by inserting fingers down the throat. Syrup of Ipecac U.S.P. 8 ml orally may aid in this.
2. Rest and reassurance.
3. Resuscitation if needed. This will usually be in the form of mouth to mouth respiration (page 197), in those patients who develop severe respiratory distress, unconsciousness with cyanosis, etc.
4. Hospitalisation and observation are always needed until recovery occurs.
5. If decongestants or antihistamines are available, they may be of benefit.

MEDICAL

1. First Aid as above
2. Hydrocortisone (100 mg i.v. every few hours for 24—48 hours) may be most effective, and can be reduced and stopped within a few days. It may be used instead of, or as well as the regimes noted in paras 3. and 4.
3. Ephedrine 30 mg q.i.d. and antihistamines, e.g. promethazine (Phenergan) i.v. 25 mg b.d. or Antistine i.v. or i.m.
4. Adrenalin (epinephrine) 0.1 ml of 1 in 1000 given per minute subcutaneously, up to 1.0 ml, may give prompt and dramatic relief of symptoms. Other parenteral sympathomimetics may be used instead of adrenalin, e.g. isoprenaline. They may be needed if respiratory distress and bronchospasm recur. Repeat doses may be needed until the antihistamines and ephedrine become effective.
5. In the severe cases of laryngeal or upper respiratory tract obstruction, emergency tracheostomy may prove lifesaving. Severe bronchospasm may require endotracheal intubation and intermittent positive pressure respiration with O_2, with or without isoprenaline.

PREVENTION

1. If allergic shellfish poisoning has occurred once, it is likely to recur — so avoid future contact with this type of food.
2. As this is an individual reaction to the shellfish protein, the fact that other people eat this food with impunity means nothing to the victim.

SHELLFISH POISONING GASTROINTESTINAL

PHYLUM : Mollusca
CLASS : Pelecypoda (Bivalves)
Mussels
Clams
Oysters
Cockles
Scallops

GENERAL

This disorder arises following the ingestion of shellfish which are contaminated b organisms capable of causing gastroenteritis in humans. Oysters are capable of ridding themselves of these organisms if allowed to live in purified water before ingestion. The types of infective gastroenteritis will vary according to the organisms concerned in each outbreak, and there have been cases of typhoid fever and viral hepatitis transmitted by ingestion of contaminated oysters. Marine vibrios have been incriminated as have viruses and other bacteria. *Escherichia coli* bacterial counts should be regularly performed on oysters intended for human consumption.

CLINICAL FEATURES

There is usually a long delay after eating the shellfish, this may be from 8–12 hours with some, and about 36 hours with others (marine vibrios). Presenting features include:

- Anorexia, malaise and weakness.
- Nausea, vomiting.
- Diarrhoea and abdominal pains, often of a cramping type.
- Headache, muscle pains, fever, etc.

Rarely do the acute gastrointestinal disturbances persist for more than 48 hours.

FIRST AID

1. Rest.
2. Encourage high fluid intake, but prohibit spicy foods, or foods difficult to digest.
3. Obtain medical assistance.
4. Use whatever home remedies one has available to reduce the incidence of diarrhoea or vomiting.

MEDICAL

1. As described in First Aid.
2. Antiemetics if indicated. Prochlorperazine (Stemetil) 12.5 mg i.m. is of value. Other drugs include antacids, antihistamines, hyoscine, phenothiazines, etc.
3. Diarrhoea is controlled by the use of Lomotil (4 tabs stat and 2 after each defaecation. Maximum = 12 per day). Morphine and opium derivatives are of value, especially codeine phosphate, mist opii, etc. Kaolin compounds (Kaomagma) may also be used.
4. Fluid and electrolyte balance is maintained by intravenous infusions, e.g. i.v. Hartmanns solution is a reliable first choice while waiting for laboratory results. Hypertonic saline may be needed if diarrhoea is severe, as may i.v. caloric fluids, amino acids, etc.
5. Antibiotics may be of value. Neomycin (Neogastrin) 20—40 ml q.i.d. acts locally in the bowel, as does dihydrostreptomycin (Streptomagma) tabs 1 q.i.d. Sulphonamides may also be used.

SHELLFISH POISONING
PARALYTIC
PSP

SYNONYM : Saxitoxin poisoning, Gonyaulax poisoning
PHYLUM : Mollusca
CLASS : Pelecypoda (Bivalves)
Mussels — Mytilus
Clams — Donacidae
Oysters — Ostrea, Crassostrea
Cockles — Cardiidae
Scallops — Pecten, Chlamys

GENERAL

This disorder is due to the ingestion of a neurotoxin P.S.P., (saxitoxin) in shellfish, especially the bivalve filter-feeding type. This poison is often associated with the "red tide" or "waterbloom" — a discolouration of the sea due to masses of dinoflagellates — and is accumulated and concentrated by the shellfish (mussels, clams, etc.) which filter these organisms from the infested water. It usually occurs in epidemics, with all people consuming the shellfish from the area being affected. The dose of poison needed to produce symptoms is approximately 5000 mouse units. Severe symptoms may accompany the ingestion of 20,000 mouse units, and death with 30,000. The formula is probably $C_{10}H_{15}N_7O_4$. The plankton protozoa incriminated is the species *Gonyaulax catenella*. The major effect of P.S.P. is respiratory paralysis due to a peripheral action, probably by blocking conduction in the motor nerves and a direct effect upon the respiratory muscles. There is little evidence to incriminate a medullary paralysis, and probably the poison does not pass the blood brain barrier. P.S.P. also has a vasodepressant action producing hypotension in experimental animals, together with conduction defects in the heart.

Deaths from this poisoning have been reported in the Australian/New Guinea region.

CLINICAL FEATURES

- There is a latent period which varies from 20 minutes to many hours, following the ingestion of the poison.
- Paraesthesia (tingling then numbness) is usually the first symptom and occurs around the mouth (88%) and hands (83%) and may spread over the body. There may be a tingling or burning sensation in the gums, tongue, teeth, lips, etc. The circumoral area

may become hypersensitive to touch.

- Weakness of upper (62%) and lower (71%) limbs.
- Floating sensation (66%)
- Inco-ordination and ataxia (57%). Involuntary movements from muscle twitching to convulsions.
- Difficulty in speech, vision, swallowing, breathing, etc., in serious cases. These may worsen for the next few hours.
- Aches in joints.
- Increased salivation and thirst
- Nausea, vomiting and diarrhoea are possible associated symptoms.
- Transitory hypertension has been noted.
- Serum creatine phosphokinase may be increased in those with predominantly muscular symptoms.
- Fatality rate varies from 1–10%, and is due to respiratory paralysis, usually within the first 12 hours. If the victim survives that period, the prognosis is good.

FIRST AID

1. In the early stages, before the patient becomes paralysed in any way, the use of an emetic may be of great value. If the patient can be encouraged to vomit, either by Syrup of Ipecac U.S.P. 8 ml oral, by inserting the finger down the throat, or by ingesting a solution of mustard in water, it is possible that a lot of the poison may be removed with the vomitus.
2. In cases of paralysis resulting in respiratory distress, the use of mouth to mouth respiration (page 197) may be lifesaving. Ensure that the airway is clear of vomitus, and that the patient is not allowed to develop a bluish (cyanosis) colour.
3. Obtain medical assistance.

MEDICAL

1. In the early stages, the insertion of a tube for gastric lavage seems axiomatic. Apomorphine 2–8 ml s.c. may be of value if there is no evidence of suppression of the laryngeal reflex.
2. Maintain respirations by any method required. Assisted intermittent positive pressure respiration may be of value when there is only a mild involvement of respiration. A Bird Respirator would perform this adequately. When there is any severe degree of respiratory paralysis, it would seem prudent to completely control respirations by the use of endotracheal intubation and hyper-ventilation. Respiration should be based on the monitoring of serial arterial O_2, CO_2 and pH measurements.
3. The use of an endotracheal tube will also prevent the aspiration of vomitus, which is likely with a partial bulbar paralysis and gastrointestinal symptoms.
4. Amphetamines (e.g. methylamphetamine) and Metrazol are said by some to considerably reduce the period required for recovery.
5. Maintenance of fluid balance could best be performed by intravenous infusions, of Hartmann's solution, with or without potassium supplementation as indicated by serial estimations of serum electrolytes, urinary output and specific gravity. High calorie and amino acid infusions are of value in extended cases. An indwelling urinary catheter is necessary.

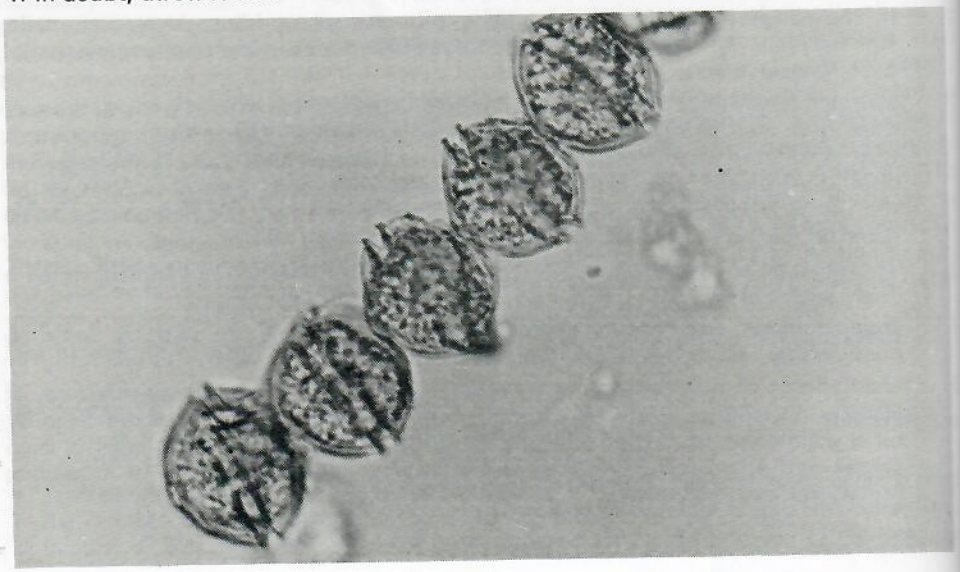
6. Monitoring procedures should include vital respiratory and cardiovascular signs, C.V.P., E.K.G., E.E.G., etc., arterial blood gases and pH.
7. Because of the possibility of consciousness persisting despite the absence of skeletal or respiratory movement, the repeated administration of a minor tranquilliser, e.g. diazepam 10 mg i.v. would seem humane.
8. Symptomatic medication may be required, e.g. analgesics, anti-diarrhoea preparations, etc.
9. On general principles, large doses of steroids (100 mg hydrocortisone i.v. 6th, hourly) could be beneficial in the severe cases.
10. Control of the epidemic. The public must be warned of the danger associated with the ingestion of shellfish. This can be achieved by the communications media (radio, T.V., newspapers) and by direct approaches to the fishing co-operatives and distributors.
11. Tracing the source from the patient to the distributors and fishermen to prevent further contaminated shellfish being supplied for public consumption.

PREVENTION

1. The unexplained presence of dead sea creatures (eels, sea birds, mollusc eating fish, etc.) is a good indication that something in the sea is poisonous.
2. Red tides (dinoflagellates) and luminescence of the water are also warning signs.
3. Cooking, and the discarding of cooking fluids afterwards, diminishes the amount of poison ingested.
4. Wherever there is any question of this, shellfish should be subjected to toxicity tests in the public health laboratories.
5. There is no way in which shellfish can be shown to be safe by visual inspection. Discolouration, folk tale techniques, and smell are unreliable guides.

"If in doubt, throw it out."

Photomicrograph of G. catenella



TURTLE AND TORTOISE POISONING

ORDER : Chelonia

SPECIES & SYNONYM : *Caretta caretta* — Loggerhead turtle
Eretmochelys imbricata — Hawksbill turtle
Chelonia mydas — Green turtle
Dermochelys Coriacea — Leatherback turtle
Pelochelys bibroni — Soft shell turtle

GENERAL

In Australia the marine species of Chelonia are termed turtles and the freshwater or land species are called Tortoises. Both have been used as food, and some of those in the north Australian waters weigh over 200 kilograms. One would have thought that the unpleasant consequences of ingestion would have made them obsolete as foodstuff, but still this animal is consumed by some ships crews, natives, etc., and is particularly reported around the Malay Archipelago and New Guinea.

There is no way of determining whether the turtle is poisonous except by trial and error. Some authors believe the poison to be similar or identical to ciguatera. Autopsy findings include hepatocellular damage, to the extent of acute yellow atrophy of the liver, necrosis of the kidney, haemorrhages and ulceration of the bowel.

CLINICAL FEATURES

- Symptoms commence a few hours to several days after ingestion of the turtle.
- Anorexia, nausea, vomiting, abdominal pain and diarrhoea in many cases.
- Abnormal sensations around the lips, mouth, tongue, throat, etc., may extend to include dryness or increased salivation and difficulty in swallowing, mouth ulcers and inflammation may supervene and become extensive — lasting for weeks or months before healing is completed.
- Weakness, sweating, pallor, vertigo, headache.
- A generalised red itchy rash may later peel.
- Difficulty in breathing, tightness in chest, may extend to severe respiratory distress, central cyanosis (bluish tingue to lips) and death.
- Liver damage may result in jaundice, liver enlargement and tenderness, coma and death.
- Other manifestations may mimic ciguatera poisoning (page 166).

- Mortality rate is over 25%.
- Renal failure may result in a decreased urinary output and then the development of uraemia over the next few days.

FIRST AID

1. If the patient is fully conscious, induce him to vomit by inserting fingers in his throat. Syrup of Ipecac U.S.P. 8 ml orally may aid in this. Treat any other potential victims (e.g. other people who ate the turtle) in the same way.
2. Rest and reassurance.
3. Resuscitation if needed. This will usually be in the form of mouth to mouth respiration (page 197) in those patients who develop severe respiratory distress, unconsciousness with cyanosis (bluish colour), etc.
4. Hospitalisation and observation is always needed until recovery.

MEDICAL

1. First Aid as above. Gastric lavage (apomorphine 2–8 mg s.c.) or other emetics if the laryngeal reflex is unimpaired.
2. With respiratory involvement, the ideal treatment is to perform endotracheal intubation and control respirations. The use of an endotracheal tube will also prevent the aspiration of vomitus, particularly likely under the conditions of a bulbar paralysis with gastrointestinal symptoms. If this is not achieved, then maintain respiration by any method at your disposal..

   Assisted intermittent positive pressure respiration (e.g. with a Bird respirator) may be of value when there is only a mild impairment. If there is a rising arterial CO_2 level or an increasing respiratory rate, assistance with respiration is required but oxygen supplementation is not needed.

   When there is a more severe degree of respiratory depression with symptomatic distress and/or cyanosis, an increasing arterial CO_2 and a decreasing arterial O_2, it would be prudent to completely control respirations by the use of endotracheal intubation and mechanical ventilation. Monitoring of serial arterial O_2, CO_2 and pH levels is required. The patient should be maintained on the regime for at least 6 hours and then gradually weaned from the respirator over the next 12–24 hours.
3. Ensure fluid and electrolyte replacement and administer medication by intravenous means (record vital signs, serial haematocrit, S.G., electrolytes, C.V.P., E.K.G., urinary output and analysis, etc.).
4. Sedation should be achieved with non-respiratory depressants, e.g. diazepam 10 mg i.v. repeated as required. Small doses of opiates may be needed for pain.
5. Steroid cover, e.g. hydrocortisone 100 mg i.v. 6 hourly during the danger period is possibly of value – but is non-specific in its effect.
6. Treat the bowel disorder symptomatically.
7. Monitor the clinical, biochemical and electroencephalographic manifestations of hepatocellular damage, and correct these by the customary medical dietetic and antibiotic techniques.
8. Monitor the clinical and biochemical manifestations of renal failure and correct these by dialysis as required.

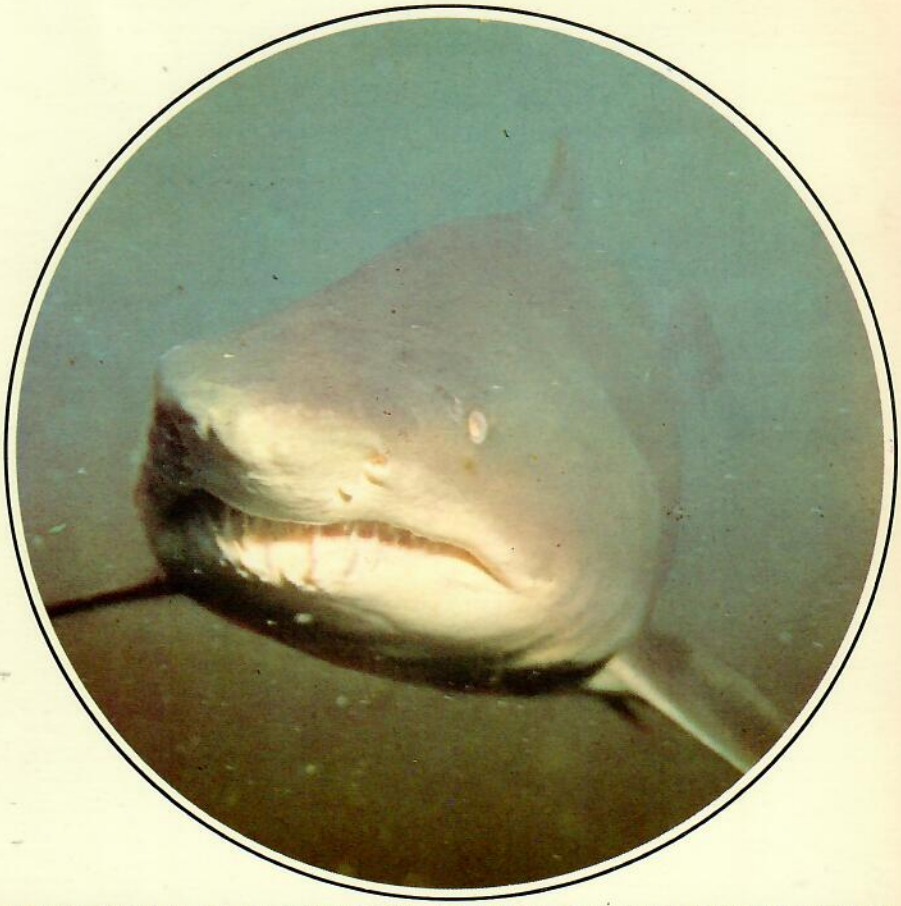
PREVENTION

Refrain from eating turtles.

DANGEROUS MARINE ANIMALS

OF THE INDO-PACIFIC REGION

by DR. CARL EDMONDS



DIVING MEDICAL CENTRE MONOGRAPH ON INDENTIFICATION
FIRST AID AND MEDICAL TREATMENT