1980s MARINE TURTLE
POISONING FILE
PART 3 OF 3 G.H. BALAZS

Turtle meal puts another three in hospital

FIJI TIMES APRIL 23, 1987

THREE other people from Nukudamu village, near Udu Point, were admitted to the Labasa Hospital from suspected food poisoning after eating turtle meat yesterday.

Admitted are Tomasi Tuinavadra, 24; Merewalesi Sese, 33; Uate Savirl, 22.

They ate the meat at a feast two weeks ago.

Last night the hospital's consultant physician Dr Gopal Krishnan said their condition was satisfactory.

Meanwhile seven others who were admitted to the hospital this week from the same incident were discharged on Tuesday.

Four people have died from the incident. They are 64-year-old Osea Naceva, Sevanaia Senibusi, Navitalai Setoki and Tuinavadra's wife Rinieta Pulu.

Reports from Nukudamu village said more people who ate the turtle meat have begun to show signs of the sickness.

A medical team which returned from Nukudamu on Monday were to make another visit yesterday but they were held back because there was no vessel to take them across .

Sub-divisional medical officer northern, Dr Asinate Boladuadua, who was to head the team said they will probably make a visit today.

Chapter XXV—VERTEBRATES

Class Reptilia

SEA TURTLES, SEA SNAKES

The class Reptilia is comprised of a group of poikilothermal vertebrates characterized by dry cornified skin with scales or scutes. Typically, there are four limbs, each with five clawed toes which are reduced or absent in some. The skeleton is bony, having one occipital condyle. Reptiles inhabit terrestrial, freshwater, or marine environments in warm, temperate, and tropical regions. There are about 6,000 species.

The living members of the class Reptilia are divided into four orders:

CHELONIA (TESTUDINATA): Turtles, tortoises, and terrapins.

RHYNCHOCEPHALIA: Beaked headed reptiles. Only one species remains, *Sphenodon punctatum*. The other members are known only from fossil remains.

SQUAMATA: Lizards and snakes.

CROCODILIA: Alligators, caimans, gavials, and crocodiles.

Only two of these orders are of interest to marine biotoxicologists: Chelonia, which includes marine turtles that are sometimes deadly poisonous to eat; and Squamata, which includes the venomous sea snakes.

Poisonous Sea Turtles

Reptiles of the order Chelonia (Testudinata) are characterized by a broad body incased in a bony shell comprised of a rounded dorsal carapace and a flat ventral plastron, joined at the sides and covered by polygonal laminae (scutes, scales) or leathery skin. The jaws are edentulous and equipped with horny sheaths. The quadrate bone is united to the skull. The ribs are fused to the shell, and the sternum is absent. All turtles (tortoises, terrapins) are oviparous in their reproduction. Although there are about 265 species in this order, only five marine species of turtles have been reported as poisonous to man.

The biology and systematics of turtles have been discussed by Stejneger (1907), Rooij (1915), Stejneger and Barbour (1933), Carr (1952), Caldwell (1960), Wermuth and Mertens (1961), Nikol'skii (1963), and others.

REPRESENTATIVE LIST OF MARINE TURTLES REPORTED AS POISONOUS

Phylum CHORDATA

Class REPTILIA

Order CHELONIA: Turtles

Family CHELONIIDAE

272 *Chelonia mydas* (Linnaeus) (Pl. 1, fig. a). Green turtle, rock turtle, meat turtle, sand turtle, right turtle, milk turtle (USA), tortuga (Latin America), gal kasbava, mas kasbava, vali kasvava, perr amai, pal amai (Sri Lanka).

DISTRIBUTION: All tropical and subtropical oceans.

SOURCES: Taylor (1921), Loveridge (1945), Halstead (1959).
OTHER NAMES: *Chelonia japonica, Chelonia virgata, Testudo mydas.*

272 *Eretmochelys imbricata* (Linnaeus) (Pl. 1, fig. b). Hawksbill turtle, scute turtle, shell turtle, comb turtle, spectacled turtle, fowl turtle (USA), tortuga (Latin America), pothu kasbava, lelli kasvava, pana kasvava, kanadi kasbava, kukulu kasbava, alunk amai (Sri Lanka), caret (France).

DISTRIBUTION: All tropical and subtropical oceans.

SOURCES: Bierdrager (1936), Deraniyagala (1939), Loveridge (1945), Carr (1952), Halstead (1959), Cooper (1964), Hashimoto, Konosu, and Yasumoto (1967).

Family DERMOCHELIDAE

272 *Dermochelys coriacea* (Linnaeus) (Pl. 1, fig. c). Leathery turtle, leatherback turtle, trunk turtle, harp turtle, luth turtle, ridge turtle, three-ridged turtle, bat turtle, oil turtle, ship turtle, boat turtle, seven-banded turtle (USA, Australia), tortuga (Latin America), dhara kasbava, thun dhara kasbava, vavul kasbava, thel kasbava, navu kasbava, mavalla, dhom amai, yelu vari, amai (Sri Lanka).

DISTRIBUTION: Largely circumtropical but occasionally taken in temperate seas off the coasts of North and South America, Mediterranean area, British Isles, and Japan.

SOURCES: Deraniyagala (1939), Halstead (1959).

BIOLOGY

Family CHELONIIDAE: The green sea turtle *Chelonia* usually inhabits water less than 25 m in depth and prefers areas sheltered by reefs where it feeds on algae. It is also common in bays and lagoons. Occasionally *Chelonia* will make its way into freshwater lakes. Green turtles are sometimes seen

basking on reefs and beaches of islands uninhabited by man. They are omnivorous but primarily vegetarian, feeding upon *Cymodocea, Thalassia, Zostera, Halophila*, and other algae. When kept in captivity they seem to show a preference for a diet of meat and fish. Green turtles nest between the latitudes 30° north and 30° south of the equator. They will migrate considerable distances, leaving their usual haunts to get to their breeding grounds. The nest site is usually selected on a beach having loose sand within reach of the waves. When the exact spot is selected, the loose sand is brushed away with the front flippers, but the actual digging is done with the hind ones. About 60 to 190 eggs may be laid at a time. Upon completion of laying, the turtle covers her nest completely with sand. She obliterates her tracks by throwing sand over her back with the front flippers as she moves away. The entire nesting process requires about 2 hours. The breeding season seems to be from July to November in Sri Lanka, but October to mid-February in Australia. This species is considered one of the more valuable turtles for use as food.

The hawksbill turtle *Eretmochelys* is generally found close to land in tropical and subtropical oceans. Seldom does it enter lagoons. Although usually considered carnivorous, it is omnivorous and at times may subsist entirely upon algae. The breeding range is between 25° north and 25° south of the equator. Eggs are laid on sandy beaches in a manner similar to that used by *Chelonia*. As many as 115 eggs or more are laid at a time. The egg-laying season extends from November to February in some areas, but seems to take place during April to June in others. *Eretmochelys* is of commercial importance because of its overlapping scutes which are utilized in the manufacturing of jewelry, etc.

Family DERMOCHELIDAE: The leather turtle *Dermochelys*, said to be the swiftest and the largest of living chelonians, usually inhabits relatively deep water near the edge of the Continental Shelf. Newly hatched leatherbacks head directly for the open ocean and do not return to shallow water until they are ready for egg laying. An adult may attain a weight of more than 780 kg. Their food consists of algae, crustaceans, and fishes. *Dermochelys* is believed to lay eggs three or four times a year, which in Sri Lanka takes place during May to June. The eggs are laid on sandy beaches at night. Often several females will deposit their eggs in close proximity to each other.

For additional information regarding the biology of sea turtles, see Deraniyagala (1939), Carr (1952), and Caldwell (1960).

BIOGENESIS OF CHELONITOXIN

The origin of turtle poison (chelonitoxin) is unknown, but most investigators who have studied the problem appear to be rather consistent in their opinion that the toxin is derived from poisonous marine algae eaten by turtles (Deraniyagala, 1939; Loveridge, 1945; Romeyn and Haneveld, 1956; Pillai *et al.*, 1962). Turtle poisoning has a remarkable resemblance to ciguatera in its sporadicity and spotty geographical distribution; a species of turtle may be

basking on reefs and beaches of islands uninhabited by man. They are omnivorous but primarily vegetarian, feeding upon *Cymodocea, Thalassia, Zostera, Halophila*, and other algae. When kept in captivity they seem to show a preference for a diet of meat and fish. Green turtles nest between the latitudes 30° north and 30° south of the equator. They will migrate considerable distances, leaving their usual haunts to get to their breeding grounds. The nest site is usually selected on a beach having loose sand within reach of the waves. When the exact spot is selected, the loose sand is brushed away with the front flippers, but the actual digging is done with the hind ones. About 60 to 190 eggs may be laid at a time. Upon completion of laying, the turtle covers her nest completely with sand. She obliterates her tracks by throwing sand over her back with the front flippers as she moves away. The entire nesting process requires about 2 hours. The breeding season seems to be from July to November in Sri Lanka, but October to mid-February in Australia. This species is considered one of the more valuable turtles for use as food.

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safe to eat in one locality but deadly in another. These observations lend support to the idea that turtle poison is derived from the food of the animal.

MECHANISM OF INTOXICATION

Chelonitoxications result from the ingestion of the flesh, fat, viscera, or blood of various species of tropical sea turtle. Toxicity in turtles is sporadic and may occur at any time of the year. The degree of freshness of the turtle meat has no bearing on the toxicity of the organism.

MEDICAL ASPECTS

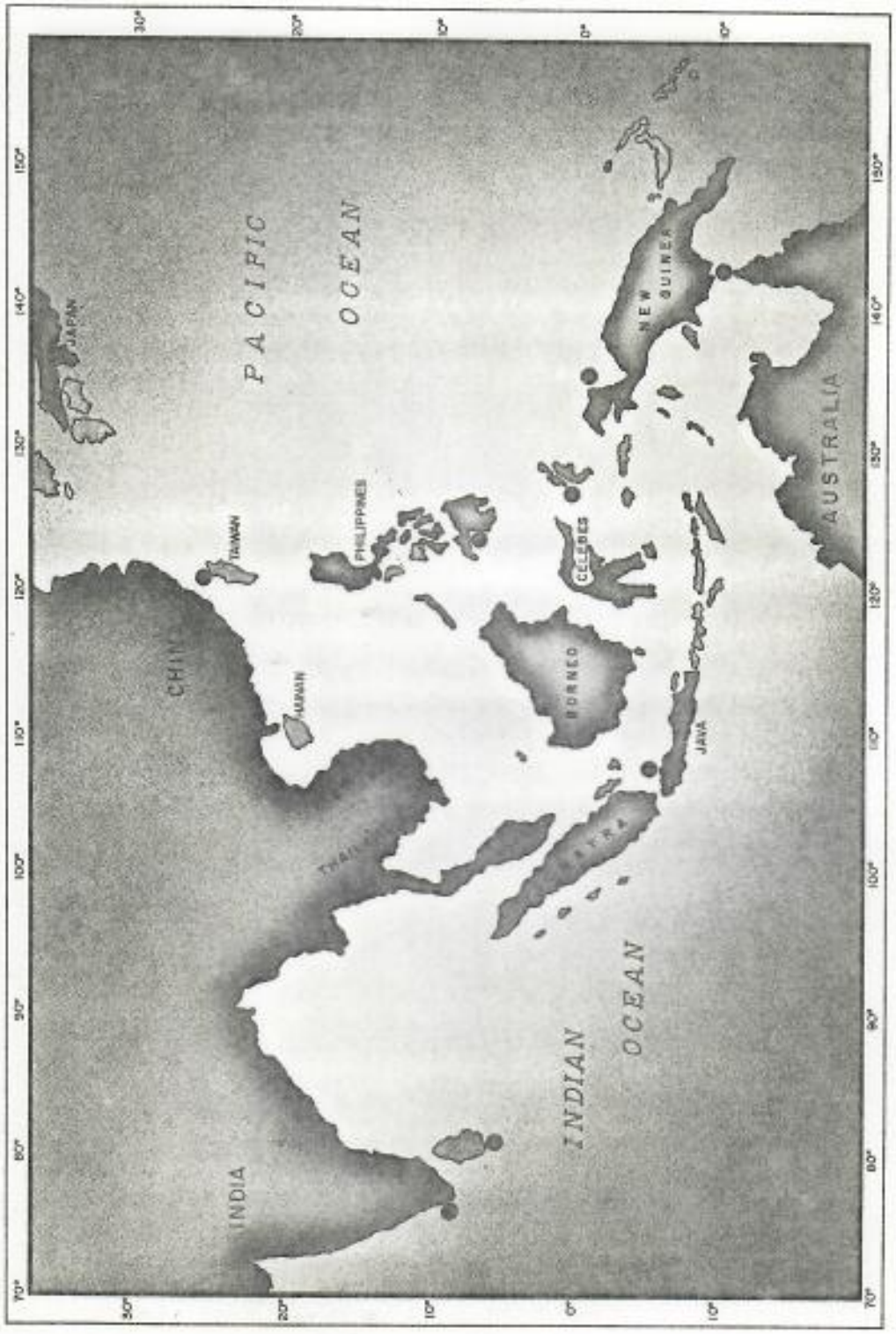
Clinical Characteristics

The symptoms of chelonitoxication vary with the amount of flesh ingested and the person. Symptoms generally develop within a few hours to several days after eating the turtle. In one large outbreak involving 100 persons, most of the victims developed symptoms about 12 hours after eating the turtle. The initial signs and symptoms usually consist of nausea, vomiting, diarrhea, facial tachycardia, pallor, severe epigastric pain, sweating, coldness of the extremities, and vertigo. There is frequently reported an acute stomatitis consisting of a dry, burning sensation of the lips, tongue, lining of the mouth, and throat. Some victims complained of a sensation of tightness of the chest. The victim frequently becomes lethargic and unresponsive. Swallowing become very difficult, and hypersalivation is pronounced. The oral symptoms may be slow to develop but become increasingly severe after several days. The tongue develops a white coating, the breath becomes foul, and later the tongue may become covered with multiple pinhead-size, reddened pustular papules. The pustules may persist for several months, whereas in some instances they break down into ulcers. Desquamation of the skin over most of the body has been reported (Cooper, 1964). Some victims develop a severe hepatomegaly with right upper quadrant tenderness. The conjunctivae become icteric. Headaches and a feeling of "heaviness of the head" are frequently reported. Deep reflexes may be diminished. Somnolence is one of the more pronounced symptoms present in severe intoxications and is usually indicative of an unfavorable prognosis. At first the victim is difficult to awaken and then gradually becomes comatose, which is followed rapidly by death. The symptoms presented are typical of a hepatorenal death. The overall case fatality rate on reported outbreaks is about 28 percent (Halstead, 1970).

The clinical characteristics of marine turtle poisoning have been discussed by Bierdrager (1936), Deraniyagala (1939), Kinugasa and Suzuki (1940), Romeyn and Haneveld (1956), Pillai *et al.* (1962), and others.

Pathology

The few autopsy reports that are available cite pathological findings consistent with an alimentary toxicosis. Aside from the oral findings described in the



MAP 1
Map showing areas where major outbreaks of sea turtle biotoxications have occurred.
(R. Kreuzinger, from various sources)

preceding clinical section, the esophagus may show ulceration of the mucosa. The serosal surface of the stomach in some cases has revealed dilatation of the blood vessels along the lesser curvature, with scattered ecchymoses along the vessels. The mucosa was generally rough and eroded, but without marked ulcer formation. Pathological changes are quite marked in the intestines. The small intestine showed dilated blood vessels, scattered regions of congestion, and irregular scattered areas of ecchymoses. Most of the changes appear in the proximal portion of the small intestine, but some are observed in the ileocecal region. Aside from dilatation of the coronary arteries, there are no remarkable changes reported for the heart. Liver pathology varies somewhat, but generally the liver is enlarged and the capsule irregular and tense, pale or yellow in color, or else soft and friable. The liver most commonly shows evidence of a massive fatty degeneration, centrilobular congestion, and necrosis of the adjacent liver cells. In some instances the fatty changes are so great as to give the appearance of solid fatty tissue. The kidneys may also vary in appearance from cloudy swelling, fatty degeneration, or necrosis of the tubules.

Autopsy reports on marine turtle intoxications have been published by Bierdrager (1936), Kinugasa and Suzuki (1940), Romeyn and Haneveld (1956), and Pillai *et al.* (1962).

Treatment

There are no known antidotes for chelonitoxin. The treatment is symptomatic.

Prevention

There are no reliable external characteristics which differentiate a poisonous sea turtle from a nonpoisonous one. According to Bierdrager (1936), the natives on the northern coast of west New Guinea claim that toxic turtles have a long neck, black tongue, and black under the chin. However, apparently these natives do not have much confidence in their ability to recognize the edible turtles from the poisonous ones because they test the turtle by feeding parts of the flesh to cats and dogs before they partake of it. If one is going to eat sea turtles in a region where they are reputed to be toxic, the safest procedure is first to feed a sample of the flesh to a dog or cat and then wait at least 24 hours before eating the turtle. Natives frequently succumb to turtle poisoning; nevertheless, one should always check with the local inhabitants concerning the safety of eating turtle meat. If there is the slightest suspicion regarding the edibility of a marine turtle, the turtle should be discarded.

PUBLIC HEALTH ASPECTS

Geographical Distribution

Aside from the somewhat vague report of two cases of marine turtle poisoning in the Windward Islands, West Indies, most of the outbreaks have taken place in the Indo-Pacific and particularly in the Malay Archipelago, southern

Sri Lanka, and southern India (*see* Map 1). It is noteworthy that the distribution of poisonous turtles does not coincide with the general zoogeographical distribution of sea turtles, but the toxic individuals appear to be confined to rather specific localities in the Indo-Pacific region. Areas that are considered to be especially dangerous are the Moluccas Islands; Japen Island, west New Guinea; Quilon, India; and Pantura, Sri Lanka. Numerous outbreaks of turtle poisoning have occurred over the years along the coasts of Mindanao, Philippine Islands, but there are no specific locality data available.

Incidence and Mortality Rate

The overall incidence of chelonitoxications is unknown, but it is quite apparent that the incidence is much higher than the few reports cited indicate. Like ciguatera, most outbreaks of turtle poisoning occur in areas where public health facilities are either minimal or nonexistent. In regions where poisonous sea turtles are endemic they do constitute a definite public health problem to the local populace and must be reckoned with by the transient visitor. The reported outbreaks show a total of 365 persons poisoned, of which 103 persons died, giving a case fatality of about 28 percent.

Seasonal Incidence

It is believed that sea turtle poisoning may occur during any season of the year, but the most dangerous months, apparently, are April through August.

Ecology

Most investigators who have studied the problem of sea turtle poisoning to any extent are convinced that the poison is derived from toxic marine algae. However, ecological and food web studies have not been conducted on this subject (*see* section on Biogenesis of Chelonitoxin, p. 919).

TOXICOLOGY

There appear to be only three toxicological reports in which attempts were made to test the toxic properties of turtle flesh, viscera, or blood experimentally on laboratory animals. Marcacci (1891) injected blood from a large sea turtle (species not given) intravenously and intraperitoneally into dogs. The injections resulted in a progressive paralysis unaccompanied by convulsions. Marcacci's report is too brief to be of much value.

Loisel (1904) prepared alcoholic extracts from the eggs of the European freshwater turtle *Testudo pusilla*. Intravenous injections of the extracts into the ear veins of rabbits resulted in tonic convulsions and death. Loisel did not investigate the toxicity of sea turtles.

Kinugasa and Suzuki (1940) prepared alcoholic extracts from the flesh of a large sea turtle of an unidentified species which had poisoned 57 persons at Koryu, Taiwan. Seven of the victims died. The extracts were injected subcutaneously into two guinea pigs, one mouse, and six frogs. Both the guinea pigs vomited several times about 15 minutes after injection of 20 ml

subcutaneously, but none of them died. The mouse was given 2 ml subcutaneously but showed no reaction. The following day the same mouse was given 10 ml of the extract. About 30 minutes after the injection the mouse became restless, rubbed its face with its forepaws, and then vomited a small amount of mucus and undigested food. The mouse showed no further signs but died 30 hours after the injection. Injections of the extracts in the frogs showed stimulation of the vagus nerve and slowing of heart action with small doses (0.5 ml), paralysis of the vagus nerve and increased heart rate with larger doses (1.0), and cardiac arrest with larger injections (10-15 ml).

PHARMACOLOGY

(Unknown)

CHEMISTRY

(Unknown)

Venomous Sea Snakes

Snakes are characterized by a body covering of horny epidermal scales or shields. The quadrate bone is movable with the skull. Vertebrae are usually of the procoelous type. Snakes may be either oviparous or ovoviviparous. They are further modified by the absence of limbs, feet, ear openings, sternum, or urinary bladder. The mandibles are joined anteriorly by ligaments. Their eyes are immobile, covered by transparent scales, and without lids. The tongue is slender, bifid, and protrusible.

The only serpents pertinent to this monograph are the sea snakes of the family Hydrophiidae. Sea snakes have a body more or less compressed posteriorly and a strongly compressed, paddle-shaped tail. Their nostrils, with the exception of *Laticauda*, are situated on the upper surface of the snout and have watertight, valvelike closures. The tongue in sea snakes is short, and only the cleft portion is protrusible. The loreal shield is usually absent. Venom fangs are present, and the maxillary teeth are grooved.

The biology and systematics of sea snakes have been discussed by such authors as Wall (1921), Smith (1926, 1935, 1943), Ditmars (1931), Volsøe (1939), Loveridge (1945), Kinghorn (1961), Taylor (1965), and Dunson (1975).

REPRESENTATIVE LIST OF SEA SNAKES REPORTED TO CAUSE HUMAN ENVENOMATIONS

Phylum CHORDATA

Class REPTILIA

Order SQUAMATA: Lizards and Snakes

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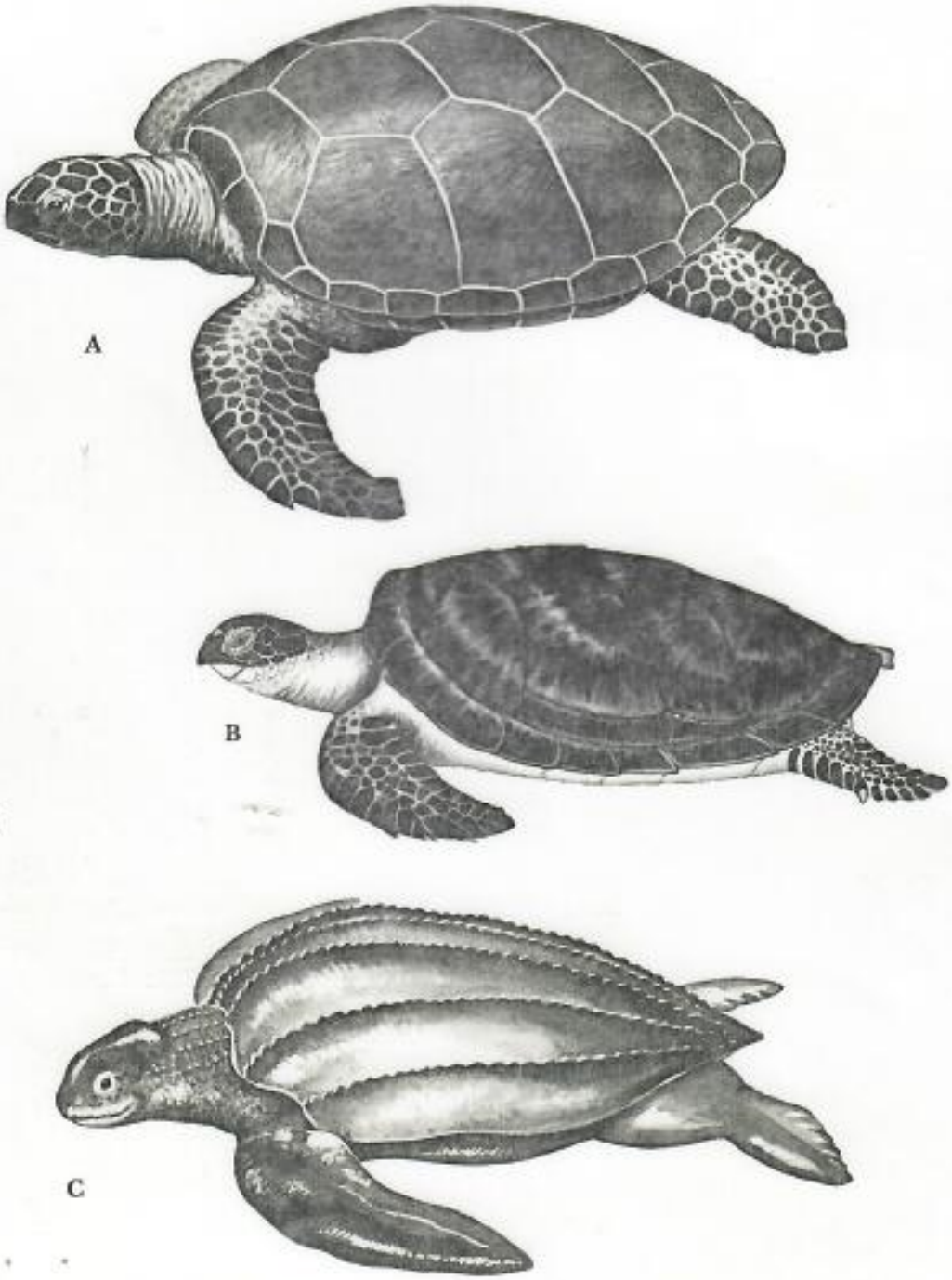
XXV: PLATE 1

FIGURE a.—*Chelonia mydas* (Linnaeus). May attain a weight of over 250 kg, and a carapace length of about 120 cm. The flesh of this turtle may be poisonous to eat in some localities. (H. Baerg)

FIGURE b.—*Eretmochelys imbricata* (Linnaeus). May attain a weight of about 125 kg, and a carapace length of about 85 cm. The flesh of this turtle may be poisonous to eat in some localities. (Mrs. R. Kreuzinger)

FIGURE c.—*Dermochelys coriacea* (Linnaeus). May attain a weight of over 250 kg and a carapace length of about 120 cm. The flesh of this turtle may be poisonous to eat in some localities. However, in most regions this turtle is highly esteemed for its fine flavor. The toxicity of marine turtles is believed to be due to their feeding on toxic plants. Heron Island, Queensland, Australia. (H. Baerg)



Chapter III—INVERTEBRATES

Phylum Porifera

SPONGES

Sponges are multicellular animals of simple and loose organization. Generally, they have spicules of silica or calcium carbonate imbedded in their bodies for support and fibrous skeletons made of a horny substance called spongin; however, either or both of these may be lacking. Regarded as plants for many centuries, sponges received animal status from zoologists in 1835.

Because sponges lack a distinct enteron and the germ layers are not well established, the phylum Porifera is sometimes classed in a separate subkingdom, the Parazoa, or the Metazoa.

Prior to the advent of synthetics, the marine sponge industry was quite substantial. The annual world commercial sponge crop during its peak years is said to have been valued at more than $5 million (Potter, 1938). Production in the United States is at present less than 6 percent of what it was in 1936 (Walford, 1958). Sponge fishing is one of man's most ancient industries, the finest sponges having come from the Mediterranean area. Moore (1908) has written an excellent and well-illustrated article on the former sponge fishing industry.

There are approximately 4,000 species of sponges: about 1 percent (all members of a single family) inhabit fresh water; 10 percent are intertidal; and the remainder are marine or benthic.

The brevity of this chapter merely reflects the lack of our knowledge in this vast uncharted biochemical sea.

The systematics and identifying characteristics of the sponges pertinent to this chapter have been discussed by De Laubenfels (1932, 1936, 1950, 1953), Dickinson (1945), Pratt (1948), and Miner (1950).

REPRESENTATIVE LIST OF MARINE SPONGES REPORTED AS TOXIC

Phylum PORIFERA

Class DEMOSPONGIAE: Sponges

Family DESMACIDONIDAE

Fibulia nolitangere (Duchassaing and Michelotti) (Pl. 1, fig. a). *8*

DISTRIBUTION: West Indies.

SOURCES: De Laubenfels (1953), Fraser-Brunner (1973).

OTHER NAME: *Amphimedon nolitangere*.

9 *Microciona prolifera* (Ellis and Solander) (Pl. 2). Red moss, red or red beard sponge, oyster sponge (USA).

DISTRIBUTION: Cape Cod to South Carolina.

SOURCES: Corson and Pratt (1943), Nigrelli, Jakowska, and Calventi (1959), Jakowska and Nigrelli (1960), Fraser-Brunner (1973).

Family HALICLONIDAE

8 *Haliclona viridis* (Duchassaing and Michelotti) (Pl. 1, fig. b). Green sponge (USA).

DISTRIBUTION: West Indies.

SOURCE: Jakowska and Nigrelli (1960).

Family TEDANIIDAE

8 *Tedania ignis* (Duchassaing and Michelotti) (Pl. 1, fig. c). Fire sponge, scarlet sponge (USA).

SOURCES: Duchassaing and Michelotti (1864), Verrill (1907), De Laubenfels (1936, 1953), Fraser-Brunner (1973).

BIOLOGY

Sponges obtain food by propelling water through tiny pores in the body wall, thus capturing microorganisms and organic detritus that may be present. It is believed by some that they may utilize dissolved nutrients. Since there is no digestive tract, digestion is intracellular and takes place in the specialized collar cells, in the choanocytes, and in the amebocytes. Some of the products of digestion that are stored in amebocytes as glycogen, fats, glycoproteins, and lipoproteins are known as thesocytes. Excretory products are usually complex nitrogen bases such as agmatine, a guanidine derivative.

Because of their sessile habits and porous structure, sponges are a veritable hotel of living sea creatures, including other sponges, bryozoans, mollusks, coelenterates, annelids, crustaceans, echinoderms, fishes, blue-green algae, and cryptomonads. With the limited exception of certain nudibranchs, sponges are not used for food to any great extent by other marine animals. Despite their complex ecological relationships with other marine organisms, sponges appear to do an excellent job of resisting the action of the multitudinous organisms that enter the labyrinthine canals and cavities, and, in some instances, become part of their diet. Sponges constitute a rich source of biologically-active substances, including antibiotic and toxic materials (Nigrelli, Jakowska, and Calventi, 1959; Jakowska and Nigrelli, 1960).

MECHANISM OF INTOXICATION

Sponge poisoning is contracted from handling the sponge. There is no knowledge of human intoxications resulting from the ingestion of sponges.

MEDICAL ASPECTS

Clinical Characteristics

Very little information is available regarding the toxic effects of sponge extracts on man. Verrill (1907) and De Laubenfels (1936, 1953) pointed out that contact with the surface of the West Indian sponges, *Tedania ignis* and *Fibulia nolitangere*, results in immediate swelling and smarting of the skin. The reaction resembles that produced by poison oak *(Rhus)*. Similar lesions are said to be caused by *Hemectyon ferox* (Duchassaing and Michelotti, 1864). The dermatitis is believed to be the result of a chemical rather than a mechanical irritant, i.e., the spicules. Corson and Pratt (1943) reported a contact dermatitis known as "red moss" or sponge poisoning, which occurs frequently among oyster fishermen in Northeastern United States from handling *Microciona prolifera*, the red sponge. The initial symptoms consist of redness, a feeling of stiffness of the finger joints, and swelling of the affected part. Within a few hours, variable-sized blisters containing clear or purulent fluid may develop over the affected area. This dermatitis gradually spreads until it involves an extensive skin area and, if inadequately treated, continues for several months. The diagnosis of the disease can be confirmed by a patch test using a small piece of the sponge. Corson and Pratt's experiments suggest that red moss poisoning is probably due to a mechanical irritation produced by the spicules of the sponge, but this has not been definitely established. Nothing is known about the effects of the oral administration of toxic sponge extracts to man.

In the Tisza Valley, Szeged, Hungary, fishermen who came in contact with certain freshwater sponges were reported to have a similar type of dermatitis (Szentkirályi, 1937).

The ailment known as "sponge fishermen's disease," or "la maladie des pêcheurs d'eponges nus" as it is termed in some parts of the Mediterranean region is caused by contact with the stinging tentacles of the small coelenterate *Sagartia rosea* which is attached to the base of the sponge. The disease is not due to a chemical irritant produced by the sponge itself (Zervos, 1903, 1934, 1938; De Laubenfels, 1936; White, 1934). This disease is discussed elsewhere (*see* p. 110). Cleland and Southcott (1965) have discussed injuries caused by sponges in the Australian region at length, and Southcott (1976) has incriminated *Neofibularia mordens* and *Lissodendoryx sp.* as capable of inflicting a contact dermatitis.

Treatment

Soothing lotions and antiseptic dressings may be applied to the affected areas. De Laubenfels (1936) found dilute solutions of acetic acid to be effective. Antibiotic therapy may also be indicated.

TOXICOLOGY

Data on the toxic effects of sponge extracts are meager. Richet (1906*a*) was not only the first, but also one of the few men to test sponge extracts on laboratory animals. He prepared alcoholic extracts from the marine sponge *Suberites domunculus* and obtained a crude toxic product that he called suberitine. Richet found that dogs or rabbits developed vomiting, diarrhea, prostration, evidence of abdominal pain, intestinal hemorrhages, and respiratory distress if suberitine were injected intravenously. The lethal dose of the crude extract was said to be 10 mg/kg of animal weight in dogs. Autopsies of the animals revealed pericardial and intraperitoneal hemorrhages. If suberitine was administered orally, no untoward effects were observed. Moreover, if the sponge extract was heated for a period of time at temperatures higher than 80° C, the toxic principle was destroyed. In another series of experiments, using a wider spectrum of test animals—dogs, rabbits, guinea pigs, frogs, turtles, pigeons, etc. —Richet (1906*b*) found that the lethal dose of suberitine varied from 6 to 16 mg/kg of animal weight, depending upon the species and the individual.

As a result of the autopsy finding observed by Richet (1906*b*) in animals that had been killed by sponge extracts, Lassablière (1906) attempted to determine the effect of suberitine on globular resistance and hemolysis production. He concluded that dog red blood cells became susceptible to disruption with dilutions of suberitine; however, hemolysis did not occur.

De Laubenfels (1932) has made the field observation that if the sponge *Tedania toxicalis* is placed in a bucket of seawater with fish, mollusks, crabs, and worms, the other animals expire in less than an hour. When placed under similar conditions, other species of sponges were observed to be nontoxic. De Laubenfels believed that this species of sponge exuded a chemical substance that was toxic to most species of marine animals.

Zahl (1953) found aqueous extracts prepared from *T. ignis*, *Ircinia fasciculata*, *Spheciospongia vesparia*, *Dysidea etheria*, and *Callyspongia vaginalis*, to be toxic if injected intraperitoneally into white mice.

Halstead and Habekost (1954) prepared aqueous extracts from *Pseudosuberites pseudos*, a marine sponge that was obtained from Guaymas, Sonora, Mexico. Crude extracts were prepared by adding 1 ml of distilled water for each gram of sponge tissue, homogenizing the mixture in a Waring Blender, and centrifuging for 20 minutes at 2,000 rpm. One ml samples of the clear supernatant were injected into each of eight Swiss Webster mice weighing between 15 and 20 g. All the mice died within from 52 minutes to 21½ hours. The extracts showed some decrease in toxicity after storage for 3 to 6 months at a temperature of -5° C. Preliminary studies indicated that this particular poison is not soluble in methanol.

The antibiotic properties of aqueous extracts from the green sponge *Haliclona viridis* were investigated and found to be quite toxic to a variety of laboratory animals (Jakowska and Nigrelli, 1960). The extracts appeared micro-

scopically as homogeneous cell populations. The active principle was heat-stable and could be extracted selectively from frozen and heat-dried sponges with a variety of organic solvents and chromatographic procedures. The authors did not indicate the exact nature of their methods. Aqueous extracts in dilutions from 10 to 100 ppm were found to be lethal to a variety of invertebrates, fish, amphibia, and mice. These same extracts were also observed to exhibit an antimicrobial effect on *Staphylococcus aureus* and *Escherichia coli*. There appeared to be a direct relationship between toxicity and antimicrobial activity.

No other reports have been found thus far on the toxic properties of marine sponges. However, Arndt (1928) has reported that aqueous extracts prepared from the freshwater sponges *Spongilla lacustris* (Linnaeus), *S. fragilis* Leidy, *Ephydatis fluviatilis* (Linnaeus), and *E. mulleri* (Lieberkuhn), are toxic when injected intraperitoneally, intracardially, or subcutaneously into white mice or guinea pigs.[1] Injections produced diarrhea, prostration, and respiratory distress. The extracts were observed to produce a weak hemolysis of sheep and guinea pig red blood cells. Desiccation for 2 years and temperatures up to 100° C did not destroy the toxic properties of the extract.

PHARMACOLOGY

Das, Lim, and Teh (1971) have reported the isolation of histamine and other histamine-like substances from the marine sponge *Suberites inconstans*, commonly found in the western tropical Pacific.

CHEMISTRY

(Unknown)

[1] According to De Laubenfels (1936), the sponges that Arndt called *Ephydatia* actually belong to the genus *Meyenia*.

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devoid of hemolytic activity for fish erythrocytes. The poison appeared to affect the nervous system of fish, frogs, or mice, particularly the respiratory centers, before voluntary muscles. Injection of the crude toxin seemed to produce a general paralysis. Symptoms observed in mice included increased activity and tremors. After 10 minutes, there was ataxia, decreased muscle tone, flaccid paralysis, slowed and labored breathing, defecation, aphradisia, marked myosis, cyanosis, anoxic convulsions, and death. Survival time in mice was 1 to 48 hours, depending on the dose administered. Post mortem examination revealed that the lungs were ischemic; the heart was contracted, especially the left ventricle; hemorrhagic edema appeared in the peritoneal cavity; the skin of the nose and ears was very white; the cornea was cloudy; the colon had no formed stools; and the urinary bladder was empty. Localized changes in cardiovascular tone were observed in some test animals. *Physalia* toxin in the isolated heart of the clam elicited responses similar to those caused by acetylcholine.

Burkholder and Burkholder (1958) demonstrated antimicrobial activity in extracts prepared from sea whips, sea fans, and plexurid corals. The active principle is said to be present in the outer gray-purple of the cortex of the coral. No antimicrobial activity could be detected in the species of stony corals that were tested. The researchers were unable to determine whether the coral polyps or their associated zooxanthellae produce the antibiotic materials.

Lane, Coursen, and Hines (1961) have found that the crude toxin of *Physalia* withstands lyophilization without significant loss in toxicity for prolonged periods of time. They were able to separate *Physalia* toxin into component peptides by one-dimensional chromatography in 80 percent of aqueous *n*-propanol. It was observed that each of the resultant peptides retained considerable toxicity for the fiddler crab *Uca pugilator*. Further studies indicated that the crude toxin of *Physalia* nematocysts had no effects on the growth of 14 species of marine yeasts and 10 species of marine bacteria, or the ciliate protozoans *Paramecium caudatum* and *Tetrahymena gelli*.

The loggerhead turtle *Caretta caretta* is able to eat *Physalia* with apparent immunity.* Wangersky and Lane (1960) thought that the immunity of the turtle to Physalia toxin might be the result of protective antibodies in the blood acquired by contact with *Physalia* early in life. However, subsequent turtle plasma-toxin precipitation tests failed to indicate that the loggerhead turtle possesses such blood immune bodies. The authors suggested that possibly the loggerhead turtle may possess localized tissue antibodies, or that the turtle is not susceptible to venoms of the type injected by *Physalia*.

The toxicity of the sea anemone *Rhodactis howesi* has been investigated by Martin (1960) and Farber and Lerke (1963). Martin prepared toxic aqueous extracts from homogenates of whole sea anemones and injected the material intraperitoneally into toads *Bufo marinus*. A maximum dose of 8 ml of homogenate was administered for each 100 g of toad. The toads showed no change in behavior or reaction for several hours after injection. Later they demonstrated a stuporous condition, respiratory distress, and most of them died

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chapter 68

HAZARDOUS MARINE ANIMALS

by Albert H. Banner

Hazardous marine animals can be divided into three categories: those that present such physical dangers to man as abrasions from coral and attacks by sharks; those that are venomous, passively or actively transferring a toxin to man through a simple to complex venom apparatus such as the stinging capsules of jellyfish or the spines on scorpionfishes; and those that are poisonous to eat because of toxins produced by the body or acquired through the environment, such as pufferfishes and those fishes that produce the disease ciguatera. The physically dangerous forms are mostly confined to large predatory sharks and fishes; the venomous and poisonous animals range from the protozoans to some of the largest fishes and marine mammals. In this chapter all are considered except venomous marine snakes, which are covered by Dr. Russell in Chapter 69, those forms such as shellfish that cause individual allergic responses, and those animals that have become toxic through biologic or chemical pollution.

Hazardous marine animals reach their greatest development in tropical seas, especially around coral reefs; only a few are found off temperate coasts, but these, too, may present serious threats to public health. As would be expected from the great development of scientific and medical research in the temperate zone and the emphasis that this research places upon locally important and geographically available problems, most tropical toxiphoric forms have been little studied. Some, however, if not many, of these tropical forms are of considerable importance to the public health of indigenous populations and will be of ever-increasing importance to the world population as tourism expands and as the marine tropics are used increasingly as a food resource.

In this presentation, the arrangement is that of biology and not of medicine and follows as closely as possible the biologic arrangement of phyla and classes and, at times, orders. Only where the toxin or the venom apparatus is widely spread through a phylum, as are the nematocysts in the coelenterates, is a broad discussion offered.

More detailed information is available in two broad reviews—those of Russell, "Marine Toxins and Venomous and Poisonous Marine Animals" and *Poisonous Marine Animals*, and especially the great and comprehensive work of Halstead, *Poisonous and Venomous Marine Animals*.[1-5] The earlier work of Fish and Cobb is now dated.[6] More specialized but comprehensive reviews are found in Cleland and Southcott's *Injuries to Man from Marine Invertebrates in the Australian Region;* Baslow's *Marine Pharmacology;* and Scheuer's *Chemistry of Marine Natural Products*.[7-9] The author wishes to express his indebtedness to all these authors but especially to Halstead whose review is cited again and again in this text.

PHYLUM PROTOZOA

No marine protozoan apparently causes any direct threat to man except the dinoflagellate *Gymnodinium breve* Davis, as this organism ecologically functions in the sea as a plant and is classified by the botanists in their phylum Pyrrophyta, the short discussion of this species is in the appendix. The several other dinoflagellates whose toxins are transmitted to man principally through shellfish are discussed in the section on poisonous mollusca.

PHYLUM PORIFERA

The sponges, all members of the phylum Porifera, are mostly marine animals and all are sessile as adults. Their "tissues" (not true tissues, but loosely-organized cells) are spread

that caused vomiting in kittens when administered by feeding or injection but no deaths, they named "ciguaterin."[255, 256] Kamiya and Hashimoto reported that this emetic, when purified and hydrolyzed, produced 14 amino acids; they suggested the toxin contained a peptide moiety.[257]

Yasumoto and associates found two or possibly three toxins in some herbivorous Tahitian surgeonfishes (*Ctenochaetus striatus*, shown in Figure 68–34, and *Acanthurus lineatus*). One, found in the flesh, viscera, and gut contents, was identical to carnivore ciguatoxin in solubility and pharmacologic action. A second toxin, found only in the viscera and gut contents, was water-soluble and nondialyzable. When the second toxin was injected at lethal dosage into mice, it caused a loss of activity, weakness, and convulsions. The extract also caused hemolysis, a characteristic not found in ciguatoxin. The possible third toxin appeared to be carried by some fishes, but was recovered in amounts insufficient for study.[258]

Clupeoid Poisoning

Various species of sardines, herrings, and anchovies (various genera, including *Clupea*, *Harengula*, *Sardinella*, *Engraulis*) have been implicated in illness and death in the Indo-Pacific from the Gilberts, Samoa, Tonga, and Fiji to Madagascar and in the Caribbean.[228, 259, 260] (It should be noted that fishes of this order, Clupeiformes, are difficult to identify, and there is considerable confusion about proper scientific names, even at the generic level; therefore, the identification of toxic species, especially of those of which no specimens causing the poisoning were preserved for ichthyologic examination, is most doubtful.)

Almost nothing is known about the intoxications. Sardines (or other similar fishes) apparently are eaten with impunity most of the year or even for years, then suddenly they may become lethally toxic. In Fiji the season of toxicity is when the bololo (palolo) worm (*Eunice viridis*) rises en masse to spawn in late October or early November; in 1955, for example, five villagers ate sardines during this season and all five died.[261]

Evidently it is only the viscera that are toxic. The author examined case reports from the island of Ovalau, Fiji. In one case a Fijian fisherman had divided his catch between a resident Gilbertese and an Asiatic Indian. The former grilled and ate the whole fish and was dead before midnight; the latter cleaned his fish before cooking it and was not afflicted, but threw the viscera to a dog, which died. Similar accounts were reported by Vincent-Cuaz from Madagascar where, following a serious outbreak of clupeotoxism, the Service de Santé decreed "l'étêtage et l'éviscérage des poisson avant consommation."[250]

Syndrome. Banner and Helfrich reported the signs and symptoms as related by Dr. A. Erasito of the Levuka Hospital, Ovalau: "severe vomiting and diarrhea, fever with profuse sweating, restlessness, pulse first rapid then gradually slowing, low blood pressure (90/60), rapid respiration; the skin was reported to itch, the eyes dilated, the reflexes of the biceps, knee and ankle absent." In two cases death occurred at 3 and 11 hours after eating the fish. Dr. Erasito stated, "I have not yet heard of one poisoned by *daniva* [the sardine] who did not die."[228]

Inasmuch as the sardines are only toxic at times and in limited areas, it would appear that the toxin originates in the food chain; the toxicity of the viscera and not the flesh substantiates this hypothesis. The fishes of this group are exclusively plankton feeders, so one should look to a bloom of some toxigenic plankter. Vincent-Cuaz reported that the outbreak at Tulear, Madagascar, seemed to be associated with an upwelling, a phenomenon that often causes marked shifts in the species population of the plankton community.[250] Perhaps this toxicity, too, may eventually be attributed to dinoflagellates as is paralytic shellfish poisoning. At present, however, nothing is known about the toxin, its pharmacology, or its origin, and the only laboratory study was that of Russell, who fed a cat some *Clupea venenosa* that were reported to be toxic. Four minutes after eating the flesh (it would not touch the viscera), it assumed "a most un-catlike stance." It vomited, lost its righting reflex, but recovered in about two hours.[167]

Miscellaneous Minor Toxicities in Fishes, Reptiles, and Mammals

Filefish Poisoning

Hashimoto, Fusetani, and Kimura reported that poisonings had occurred in the Ryukyu Islands from eating the viscera of the filefish *Alutera scripta*. Examination of the stomach contents showed that toxic fishes had been feeding on toxic zoanthids, and chemical tests showed the toxin could not be distinguished from the palytoxin studied by Moore and Scheuer.[32, 262] The authors reported that the ingestion of this

fish by man induced "vomiting, diarrhea and joint-ache," while by pigs, rapid death.

Hallucinatory Mullet Poisoning

There are a few reports in the literature of intoxications by mullet and surmullet, which induce hallucinations or mental depressions in persons awake and nightmares in those who sleep. The disease is so well known to those who fish in Hawaii that the fish that most commonly causes the disease, *Upeneus arge*, a surmullet, is called the "nightmare weke" (Fig. 68–42). Cases in Hawaii were reviewed by Helfrich and Banner, who pointed out that by reputation the toxic fishes in Hawaii are confined to restricted areas on the reefs of Molokai and Kauai, although Banner later reported a characteristic case from a fish from Oahu.[263, 264]

In local Hawaiian knowledge, the mental disturbances are attributed to eating the brains. In all Hawaiian cases reported there was complete recovery overnight. Van Pel has also reported similar hallucinations from a rudderfish, probably *Kyphosus vaigiensis*.[265] Smith said of the Mullidae (surmullets or goatfishes), "It is best, however, not to eat the heads as the brains are said to cause convulsions."[163] Cooper reported that the Gilbertese believe the mullets and surmullets may cause "madness," "forgetfulness," or "sleepiness," and she stated that the fishes were eaten with "full expectation and possible enjoyment of the hallucinations or dreams that followed." She also said that the Gilbertese believed that the grouper *Epinephelus corallicola* and the damselfish *Abudefduf septemfasciatus* were also supposed to cause madness or forgetfulness.[75]

The toxin causing the hallucinations is unknown. Attempts to induce reactions in cats were unsuccessful in the experiments by Helfrich and Banner, but according to Jordan and co-workers, Dr. Nils Larsen of Honolulu fed *Upeneus arge* heads to cats, which "at once went crazy, but recovered."[263, 266]

Marine Turtles

Most marine turtles are esteemed as food. While the green turtle *Chelonia mydas* and other species have been blamed in poisoning outbreaks, the most commonly indicted is the hawksbill, *Eretmochelys imbricata* (Fig. 68–43). Considering how often this circumtropical turtle must be eaten, outbreaks of poisoning are extremely rare and are mostly, if not entirely, confined to the Indo-Pacific. The range of toxic turtles given by Halstead must be extended to include the Southern Ryukyus.[3, 79] As the hawksbill turtle may reach a weight of 123 kg, and the other species are yet larger, when a turtle is toxic, the number of individuals who eat the food and may become intoxicated is usually relatively large.[247] Pillai and associates, for example, reported an outbreak in Kerala, India, in which an unspecified number of individuals ate a large hawksbill. It was one of five caught and sold on the same day (the other four turtles were nontoxic). The 130 cases resulting were studied at the district hospital; of these, only 32 had moderate to severe symptoms, but 18 died.[267] Similarly, Cooper reported that some years before a group of Gilbertese were reported to have eaten a large hawksbill from which all individuals partaking became ill, and five died. While the disease is known by vague reputation or folklore to most people who eat turtles, the cases are often dis-

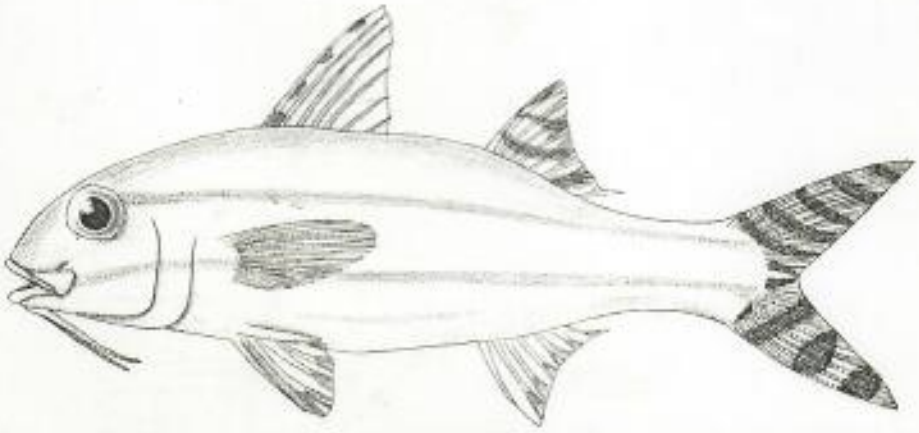

Figure 68–42. *Upeneus arge*, a goatfish, the "nightmare weke" of Hawaii. On occasion the ingestion of this fish, especially the brain or head region, will produce hallucinations or nightmares. Size ranges up to about 0.5 m. (Drawn from photograph by J. E. Randall.)

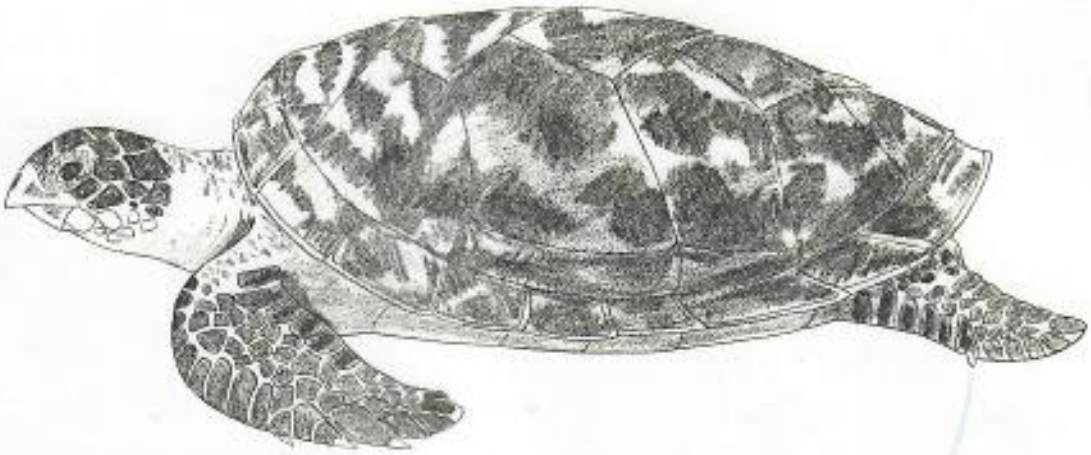

Figure 68–43. *Eretmochelys imbricata*, the hawksbill turtle. The hawksbill turtle is often eaten but may be highly poisonous and has caused many deaths. Size ranges up to almost 1 m in length and over 100 kg in weight. (Drawn from various sources.)

counted by physicians who are unfamiliar with it. Sometime in the late 1950's (my notes were lost in a fire) fishermen in a village in American Samoa captured a large hawksbill, and the entire village feasted and then became ill. When I interviewed the American doctor in Pago Pago, he dismissed the outbreak as an example of mass hysteria.

Pillai and co-workers gave a medical review of their 130 cases. They pointed out that those who had boiled the flesh and decanted the water before making curries developed at most only mild symptoms, but those who had used the cooking water became seriously ill or died. Those who ate the head of the turtle escaped with mild or no symptoms. The symptoms, which developed within 12 to 24 hours, were vomiting (with the vomitus bilious), severe constipation, general weakness, and pain in the throat. Six of the women who were pregnant aborted before death. These investigators reported that 32 of the patients were "drowsy with sunken and congested eyes and had moderate fever." The tongues were dry and coated, and the patients later developed severe glossitis; the throats were congested, but no other signs were noted in the alimentary system. There was slight tachycardia and a fall in blood pressure; the electrocardiograms were normal, even in one case in which it was taken 30 minutes before death. Nothing abnormal was noted about the respiratory system except in the 18 who died. These had pulmonary edema just before death and died of respiratory failure. In the 32 severe cases there was no pupillary response to light, deep reflexes were diminished, and the plantar response was flexor; two children had convulsions before death, while the adults died in a coma. The authors reported on findings from autopsies, and concluded, "The poison is both an irritant and a neurotoxin as evidenced by the ulcerations [of the alimentary canal] from the mouth downwards, the damage to the liver, congestion of the medulla of the brain, which produced coma and respiratory failure. The fact that the jerks were feeble with flaccid muscles with no evidence of spinal cord involvement shows that the poison had a curare-like action."[268] Hashimoto and co-workers reported that in one case an infant became intoxicated through breast milk.[79]

Halstead suggested that turtles become toxic through diet, perhaps from a toxic alga.[5] There have been no known laboratory studies on the toxin or its action.

Hypervitaminosis A

The livers of certain fishes and particularly marine mammals of the Arctic have been shown to contain so much vitamin A that a meal on the organ produces the classic symptoms of vitamin A poisoning. It is presumed the vitamin A comes from the diet and is stored. The initial symptoms include a throbbing headache, gastrointestinal disorders, dizziness, muscular weakness, and cramps. In severe cases desquamation of many parts of the body occurs.

The most infamous animal for causing this is the polar bear, *Thalarctos maritimus*. Russell reported from two specimens the concentrations of 34,600 and 30,700 international units (i.u.) per gram.[269] Ostwald and Briggs state that 1,000,000 i.u. at one time will cause toxic effects in adults; if so, a mere 30 g of Russell's sample would have caused intoxication.[270] A somewhat similar set of symptoms, including

desquamation, has been reported from eating whale liver in Japan, and Arctic seal and walrus livers.[5] Cleland and Southcott recorded probable cases caused by South Australian and Antarctic seals.[271, 272]

How extensive excess vitamin A is in fish livers is not known. Nater and Doeglas reported hypervitaminosis in 11 fishermen who ate halibut livers and received an estimated 2 to 30 million i.u. of the vitamin and had desquamation.[273] Lonie reported similar symptoms caused by livers of tropical Pacific sharks.[274] Finally, we have in our files a copy of a letter from an ichthyologist who was working in the South African Museum in which he complained of a "headache, most acute behind the eyes . . and a peeling of the skin" that resulted from eating meatballs made of the liver of an Indian Ocean tuna. It should be recalled that some fish livers, especially of cod, halibut, and soupfin shark, have been used as commercial sources of vitamin A, but that Russell reported that the concentration in polar bear liver was more than a magnitude greater than those found in fish livers.[269]

APPENDIX: TOXIC MARINE ALGAE

Various fresh-water blue-green algae and chrysophytes have been known to cause deaths of fish and farm animals and respiratory and gastrointestinal disorders in man.[275, 276] Only three types of algae in the marine environment are, however, of known importance as toxigenic agents to man, and one group of these only indirectly. The last group is the dinoflagellates that cause paralytic shellfish poisoning, which is discussed in the main text. The other two are *Gymnodinium breve* and *Microcoleus lyngbyaceus*.

Gymnodinium breve Davis, the dinoflagellate that was responsible for the "red tide" off the coast of Miami in 1947, evidently passed its toxin into the water, which was then carried downwind as an aerosol causing irritated mucous membranes. The symptoms were primarily respiratory and included coughing, sneezing, and respiratory distress.[277]

Microcoleus lyngbyaceus (previously *Lyngbya majuscula*) is a blue-green alga that is widespread and often very common on the coral reefs of the Indo-Pacific. It grows in olive-drab, hairlike masses that entangle themselves on fixed objects such as other plants or corals or, in calm water, lie in masses on the bottom. When disturbed by waves or currents, the alga is carried in semifloating masses near bathing beaches. If a bather goes through these masses, it is likely that the algal fragments will be picked up and held in close contact with the body by the swim trunks or brassiere. The areas in prolonged contact with the alga by this means may develop initial redness, followed by "blisters and deep desquamation, leaving moist, bright red and painful areas", especially "on the scrotum, perineum and perianal area."[278, 279]

It is notable that not all *Microcoleus lyngbyaceus* is toxic and that on the island of Oahu, Hawaii, from which the dermatitis was reported, had markedly differing toxicity in different geographic areas. Thus, of the algae from four beach and bay areas along 28 miles of the windward coast of the island, the two at the south were moderately toxic, the one at the north was highly toxic, and the alga from the bay in the middle was nontoxic.[232, 280]

The toxin has been isolated, but its structural formula has not been determined; it is insoluble in water but soluble in polar organic solvents.[281] The toxicity of a sample can be determined by patch tests on humans, mixed with gelatin and force-fed to mice, or extracted and injected intracutaneously into various laboratory animals.[278, 280, 287] The biologic effects of the toxin were explored by Moikeha and Chu.[282] Grauer has suggested that the dermatitis can be prevented, or at least decreased, by bathing the potentially afflicted parts as soon as possible with soap and water.

Acknowledgments: The drawings were made by Susan Monden, the invertebrates being taken from living and preserved specimens, or adapted and combined from a series of individual published sources. The fishes were drawn from the black and white or color photographs of Dr. John E. Randall. The author wishes to thank his colleagues who assisted him on a number of points and his wife for her editorial assistance.

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whereby experienced divers seek hollows under the coral where turtles frequently rest. A wooden stick with a line attached is wedged into the middle of the entrance. When a turtle enters the hollow, it dislodges the stick which floats to the surface and is seen by the fishermen from their canoes (Trust Territories of the Pacific Islands, 1957).

Various diving techniques have been used in the following locations:

Atlantic Ocean: True (1887), Marquez (1965, 1976), Nietschmann (1971, 1972a), Cardona and Rua (1972), Ramos (1974) Thurston and Wiewandt (1976), Carr (1977), Ross (1980).

Pacific Ocean: McCarthy (1955), Trust Territories of the Pacific Islands (1957), Japanese Tortoise Shell Association (1973), Pritchard (1977), McKeown (1977), McElroy and Alexander (1979), Onorio (1979), Pita (1979), Alcala (1980), Spring (1979, 1981a), Kowarsky (1981), Vaughan (1981).

Indian Ocean: Hughes (1976), Garnett (1978), Kar and Bhaskar (1981).

Remora : This is a very old method of turtle fishing practiced for centuries by natives around the world and was first described from Cuba by Columbus in 1494. The technique used is to fasten a line to the caudal peduncle of a remora (family Echeneidae) by a metal ring, band or simply a knot; the remora is then thrown overboard in the vicinity of a turtle. The fish then adheres to the turtle by the suction disc and is then pulled to the boat. The remoras are kept alive in pens or canoes filled with sea water, and they are well taken care of by the fishermen who fish them frequently and keep them for long periods of time. Presently, this method is rarely used, since more productive techniques have been introduced.

Remora turtling has been described in the following sources: De Sola (1932), Caribbean; Deraniyagala (1939), Sri Lanka; McCarthy (1955), Australia; Travis (1967), Somalia; Hughes (1973), Madagascar; Frazier (1981a), Kenya, Yemen.

Illustrations of remora turtling are found in Gudger (1919, a, b).

Grapnel: These devices are made from weighted fish hooks or curved, sharpened steel rods which are securely fastened to a central post, upon which a rope is fastened. The grapnel is dragged to the turtle, which is seen on the shallow ocean bottom, and is quickly jerked upwards when the hooks pass underneath it. The hooks thus, become embedded in the soft plastron or limb and the turtle is then pulled to the surface.

Grapnel turtling techniques have been described in the following sources: Nye (1887), Bahamas; Seale (1911), Philippines; Hughes (1973), Madagascar; Frazier (1981a), Kenya.

Illustrations of grapnel gear are found in the following source: Travis (1967), Somalia.

5.2 Fishing Areas

Hawksbills are fished throughout their tropical distribution (see section 2.1 and Figure 4), most being captured on reefs, banks and in lagoons adjacent to nesting beaches. Gillnets are fished in lagoon channels and passages between reefs, and harpooning for copulating turtles generally takes place in deeper water in front of nesting beaches. Diving, particularly at night for sleeping turtles, takes place on the shallow reef slopes facing deep oceanic depths.

5.3 Fishing Seasons

Hawksbill turtles are opportunistically harvested year-round wherever encountered. However, the majority of adult turtles are captured during the reproductive aggregations at the nesting beaches during the nesting season, the time of which varies with location (Table 2). At this time, copulating and nesting turtles are less wary, making them more susceptible to harvesting techniques.

5.4 Fishing Operations and Results

Hawksbill meat is generally eaten throughout its range, although the meat is often reported to be of poor quality, being dark and oily with a strong flavour. In spite of this, hawksbill meat is preferred over that of other sea turtles at Cayman Brac, San Andres and Old Providence Islands in the Caribbean Sea (Lewis, 1940; Thompson, 1947; Carr, 1956). The hawksbill is reportedly poisonous in many areas throughout its range, but often without documented cases. In many instances the hawksbill has been accused of being poisonous, possibly because of its strong flavour, which Carr (1956) believes to vary according to diet.

Hawksbills are not eaten, or are reluctantly eaten because of their reputed toxicity in the following locations: Gulf of Guinea (Greef, 1884), Australia (Musgrave and Whitley, 1926; Bustard, 1972), Sri Lanka (Deraniyagala, 1933), Gulf of Siam (Le Poulain, 1941), Colombia (Dunn, 1945), India (Acharji, 1950; Shantharam, 1975), Mauritius (Jones, 1956), New Guinea (Brongersma, 1958), Mexico (Del Torro, 1960), West Africa (Villiers, 1962), Seychelles (Frazier, 1971), Mauritius (Hughes, 1973, 1976), Senegal (Villiers, 1958; Maigret, 1977), Sudan (Hirth and Latif, 1980), Oman (Ross, 1981).

Documented cases of hawksbill turtle poisoning are relatively rare, having been reported from the following locations: Taiwan (Kinugasa and Suzuki, 1940), Gilbert Islands (Cooper, 1964; Onorio, 1979), Ryukyu Islands (Hashimoto et al., 1969; Hashimoto, 1979), Philippines (Ronquillo and Caces-Borja, 1960), Australia (Kowarsky, 1981), Solomon Islands (Vaughan, 1981), New Guinea (Romeyn and Haneveld, 1956; Spring, 1981, a), Sri Lanka (Deraniyagala, 1930, 1939), India (Valliappan and Pushparaj, 1973). Pillai et al., (1962) described in detail the clinical and pathological findings (including photomicrographs of affected epithelial, muscular, liver, kidney and heart tissues) of 130 cases of hawksbill poisonings in India.

Halstead (1978) reviewed the cases of turtle poisonings, summarizing all pertinent information. He found that poisoning was seasonally sporadic with a geographically intermittent distribution, and could result from eating either the meat, fat, viscera or blood. Symptoms generally appear from within a few hours to several days, depending upon amount ingested and the person. Major symptoms are: Nauses, vertigo, vomiting, diarrhea, severe gastric pain, sweating, coldness of the extremities, and a dry, burning sensation of lips, mouth and throat. Additionally, the tongue develops a white coating with tiny pustules, and victims may suffer from headaches and general lethargy. Halstead describes these clinical, as well as the pathological, characteristics in detail. There are no antidotes, consequently treatment is symptomatic. The overall case fatality rate is 28 percent, with the oldest and the youngest persons reportedly being more susceptible. Prevention is difficult, since toxic turtles are impossible to differentiate from non-poisonous turtles, the best method being to feed a sample to a dog or cat and wait at least 24 h. The origin of the toxin is unknown, but it is believed to be caused by ingestion of poisonous algae, jellyfish and even sea snakes (Deraniyagala, 1930, 1939; Le Poulain, 1941; Loveridge, 1946; Pillai et al., 1962; Villiers, 1962; Vaughan, 1981).

Indigenous people have attempted to identify toxic hawksbills by an interesting variety of methods. In Sri Lanka the fishermen offer bits of raw liver to crows - if rejected, the turtle is discarded; also, poisonous flesh reportedly turns greenish if mixed with slaked lime (Deraniyagala, 1930, 1939, 1953). Fishermen in India believe that the blood of toxic turtles thickens on a knife blade, and that a drop of blood on the skin will itch and become inflamed. They also believe, like the fishermen of Sri Lanka, that lime changes toxic meat greenish in colour (Valliappan and Pushparaj, 1973). Fishermen of the Suarkin Archipelago of Sudan choose a man who neither smokes nor drinks alcohol to taste the liver - if "evil-tasting", the meat is discarded (Hirth and Latif, 1980). Also, in the Solomon Islands, hawksbills that have unusually pale plastrons and short-straight intestines are believed to be poisonous and are not eaten (Vaughan, 1981).

Deraniyagala (1930, 1939) reported that if a toxic hawksbill should be eaten, the resulting sickness supposedly could be cured by dosing the patient with soup made from the carapace and plastron of Chelonia mydas.

Hawksbill eggs are eaten wherever and whenever found, apparently without any ill effects, being an important protein source (see section 1.3.3) in many lesser-developed villages. Calipee is also prepared for consumption in many parts of the world, and the oil and fat are commonly used for cooking.

The hawksbill turtle is most famous for the horny scutes called "tortoise shell", which are obtained from the carapace. There are 13 large and 25 small scutes on a turtle, and each scute has, when cleaned and polished, a rich brown mottling on a yellowish-translucent background. There are several different grades of shell depending on general colouration, the darker shell having the lowest demand and the lighter coloured or "blond" shells being the most often preferred. Articles such as dishes, knives, forks and spoons have been fashioned from hawksbill scutes since pre-historic man. However, the beauty of these scutes was quickly realized by affluent personages, and the manufacture of luxury items, such as the following, began: hair pins, combs, rings, nose rings, earrings, necklaces, brooches, fans, belts, miniature animals, inlayed furniture, and (more recently) eyeglass frames, cuff-links, tie tacks, buttons, snuff boxes, jewelery boxes, trays, model pagodas and model ships. The hawksbill turtle shell industry has thrived through the ages, dating back to ancient Greece, Rome and Egypt (Deraniyagala, 1939; Smith, 1958; Parsons, 1962; Schafer, 1962; Hughes, 1970; Hirth and Latif, 1980). European treasure ships returning from the New World were laden with turtle shell (Anon., 1980). Babcock (1940) presented a brief historical summary of the tortoise shell industry, as well as a state-of-the-arts profile, in the mid-twentieth century United States and Parsons (1972) gave an excellent historical account of the worldwide trade in hawksbill turtle shell.

Methods of scute removal are universally similar, the scutes being removed from the body carapace when the turtle is either dead or alive. More than one method may be used in any one location. The scutes on the carapace of a dead turtle are loosened when either placed in hot water (Seale, 1911; Hornell, 1927; Deraniyagala, 1939; Loveridge, 1946; México, Secretaría de Industria y Comercio, 1966; Hughes, 1971) or buried in the sand for a week or two (Seale, 1911; Deraniyagala, 1939; Loveridge, 1946; Travis, 1967; Ubeda, 1973). Removing scutes from

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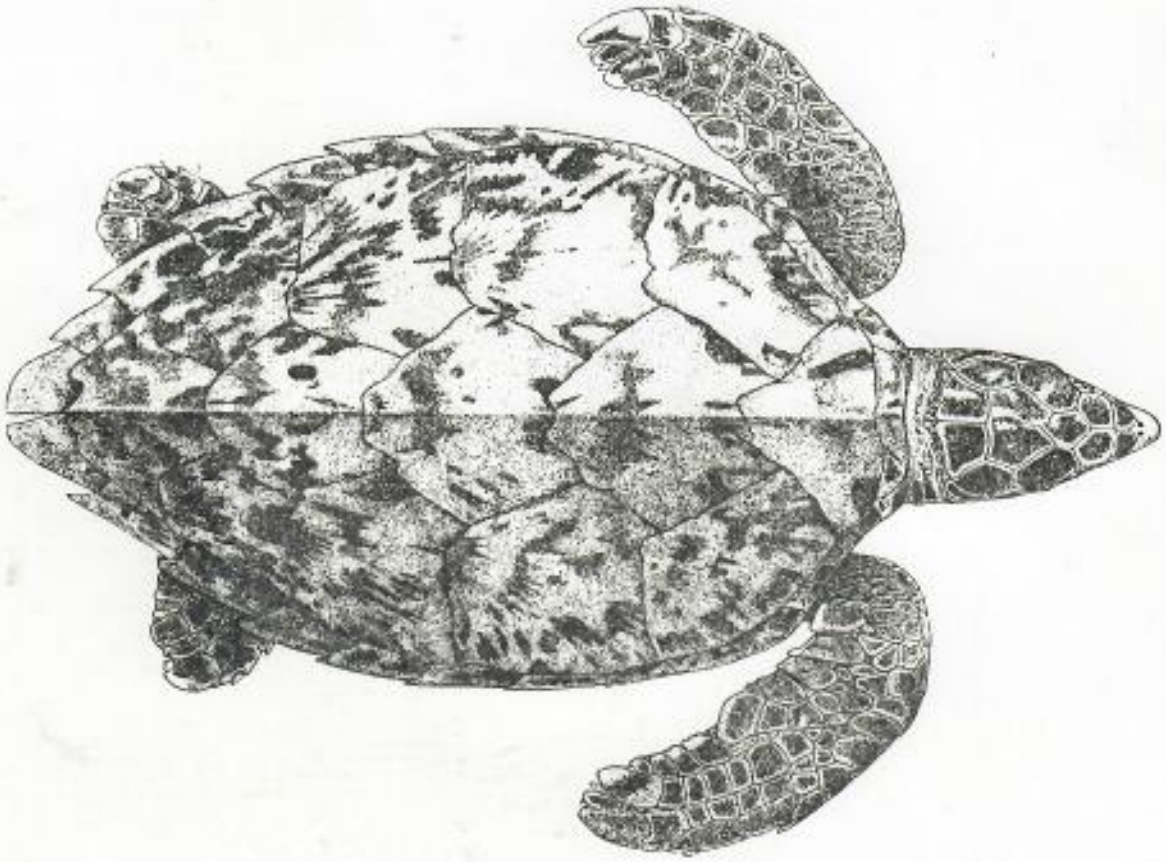
FIR/S137
SAST – Hawksbill turtle – 5,31(07)017,01

SYNOPSIS OF BIOLOGICAL DATA ON THE HAWKSBILL TURTLE *Eretmochelys imbricata* (Linnaeus, 1766)

Prepared by
W.N. Witzell



Turtle Poisoning File

Carr & Stanick 1975 Copeia

Because of the lack of information on the ecology of the bank and of volumetric data on the stomach contents, it is impossible to judge the degree of selectivity exerted by the turtles in their foraging. The combined diversity and infrequency of the molluscan remains suggests indiscriminate, opportunistic feeding, and the frequency and quantity of amorphous bottom material and detritus ingested—including a compacted ball of well-chewed sheet plastic—would seem to support that idea. Next to *Geodia* and *Styela*, bottom material was both the most frequent and the most voluminous item in eleven of the 29 digestive tracts.

All samples were taken between the middle of July and early October. This period includes a part of the nesting season (Carr *et al.*, 1966). A comparison of males with food in the gut (10 of 11 examined) and females with food (6 of 13 examined) showed no significant difference ($z = 1{\cdot}05$ in Mann-Whitney U test). Nevertheless, the trend shown in the Table suggests that a larger sample would reveal that males feed more actively at this season, and also that they are more inclined to select the sponge *Geodia* in their foraging.

It seems fairly clear that the hawksbill is a relatively indiscriminate feeder whose food consists mainly of benthic invertebrates. Since large numbers of reef-inhabiting invertebrates, particularly sponges, are toxic (Halstead, 1965; Bakus & Green, 1974), such a diet would be consistent with the occasionally poisonous character of hawksbill flesh that has been noted in various parts of the circumtropical range.

RENESTING

Multiple nesting

The Tortuguero hawksbill probably nests at least twice during a given season. Whether more nestings occur is not clear from our data. Table 2 shows every return of a hawksbill recorded at Tortuguero during a single season. Taking the small sample at face value, one would be inclined to say that only one or two nestings occur. However, the existence of the fishery on the bank introduces the possibility that renesting records were reduced by attrition of the tagged sample. The longer the stay of a turtle at Tortuguero the greater the likelihood that she would be killed.

A noteworthy feature of the tag-return records is the curiously comparable numbers of renesters and remigrants. In work with green turtles, both at Tortuguero and at Ascension Island, renestings have been found to outnumber remigrations by at least ten to one. Since the beginning of the Tortuguero project, eleven renestings and six remigrations of hawksbills have been recorded. The correspondence suggests that the low observed number of renestings may really mean that Tortuguero careys nest only once or twice, and then return to foraging grounds on the Outer Bank or elsewhere.

The homogenized sponge is centrifuged and the supernatant is evaporated. The crude extract is dissloved in 300 ml of fresh water in a finger bowl in which a goldfish is placed, and the behavior of the fish is timed and recorded. Control experiments are done simultaneously.
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2 July 1974

solated Brain Microvessels: A Purified, Metabolically Active Preparation from Bovine Cerebral Cortex

Abstract. *A purified, metabolically active preparation of brain microvessels was isolated from bovine cerebral cortex by using a simple procedure involving mild disruption of the tissue by homogenization and trapping of the vessels on nylon sieves. This preparation permits in vitro metabolic and structural studies of small blood vessels.*

Knowledge of the metabolism and structural composition of isolated blood vessels is important in studies of the biochemistry and pharmacology of this organ system, which is a primary site for a wide range of pathologic disturbances, including arteriosclerosis and diabetes mellitus. The microvessels, defined as those with diameters less than 300 μm, are frequently affected by such diseases to as great an extent as larger vessels like the aorta (*1*). However, only the latter have been the subject of extensive biochemical investigations (*2*). The difficulty of readily obtaining sufficient quantities of microvessels for experimentation is one reason for this disparity. The development in our laboratory of simple isolation procedures involving mild homogenization and sieving for obtaining morphologically intact and metabolically active preparations of kidney glomeruli and retinal blood vessels suggested that similar techniques might be useful for the isolation of microvessels from other tissues (*3*). The successful application of these techniques to the isolation of microvessels from cerebral cortex provides a readily obtained preparation of small blood vessels suitable for metabolic investigations of the microcirculatory system in general as well as those aspects peculiar to vessels of the central nervous system.

Figure 1 shows a typical preparation of brain microvessels at low (Fig. 1a) and higher (Fig. 1b) magnification. Gray matter is obtained from the cerebral cortex of bovine brain. For optimum preservation of metabolic activity, the vessels should be isolated promptly from brains removed from the animals immediately after slaughter, and the isolated vessels should be transported to the laboratory in cold oxygenated buffer to minimize the effects of anoxia on the tissue. Pieces of cortical tissue are homogenized in Earle's balanced salt solution buffered with HEPES (*4*) (1 : 1 by volume) with ten vertical strokes of a hand-held loosely fitting Teflon pestle in a smooth glass tube (*3*). The homogenate is poured over a 153-μm nylon sieve (*5*), and the material remaining on the sieve after washing with buffer is rehomogenized, resieved, and washed as before. A highly enriched preparation of microvessels with the appearance of fine threadlike strands is caught by the sieve, while the bulk of the nonvascular tissue of a fine granular nature is not retained. The vessels are freed of any adhering or accompanying nonvascular contamination either by homogenizing them with two or three additional strokes, resieving, and rewashing, or by "combing" through a suspension of the preparation in a petri dish with a piece of 210-μm nylon mesh. The vessel strands

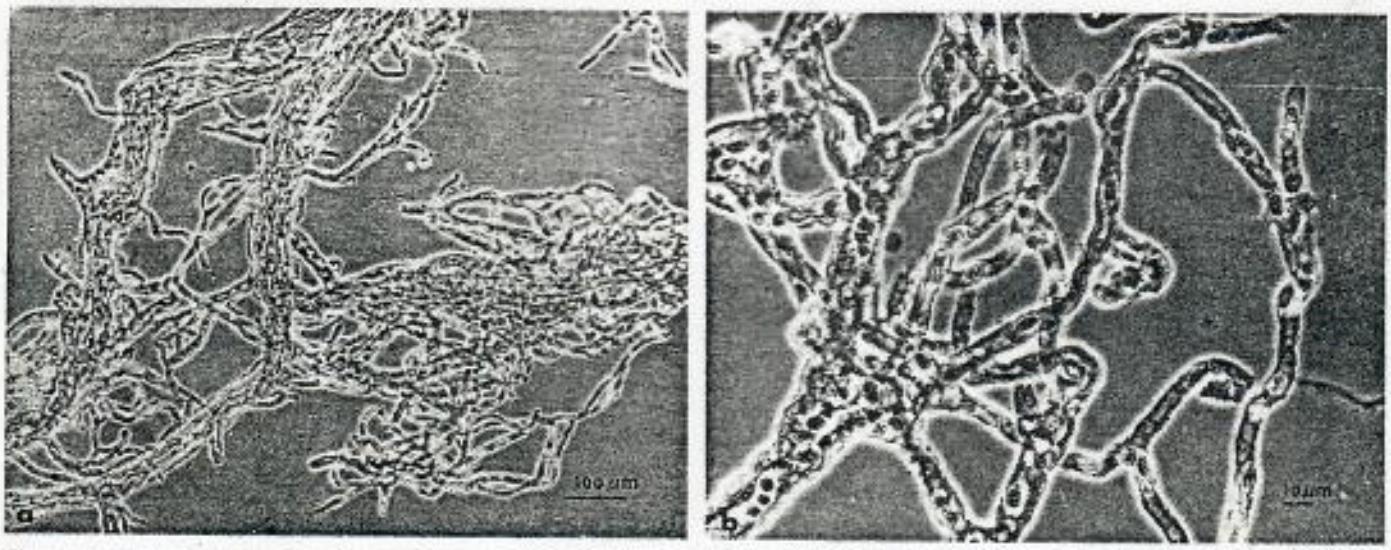

Fig. 1. (a) Phase contrast photomicrograph of isolated bovine brain vessels showing twisted plexuses of microvasculature. Numerous branches and bifurcations characterize these preparations. Nonvascular elements are not present. Vessel diameters range from 6 to 80 μm (× 85). (b) Phase contrast photomircograph at higher magnification showing vascular arborization in isolated bovine brain vessels. Clusters of red blood cells occupy vessel lumina. Endothelial cell nuclei are particularly evident at this magnification (× 350).

Disease. Surgeonfish sting.

Symptoms. Acute smarting pain may last several hours (13, 26, 96).

Treatment. See Scorpionfish sting.

Chemistry or Toxicology. Caudal peduncular spine inflicts wounds (92, 96, 123, 133).

SPECIES AND DISTRIBUTION. *Callionymus lyra* (Linnaeus), coast of France, tropic zones.

Disease. Dragonet sting.

Symptoms. Stinging pain, frequently followed by development of a felon (11, 26).

Treatment. See Scorpionfish sting.

Chemistry or Toxicology. Venom apparatus consists of venom glands in connection with spines, as in scorpionfish and weeverfish. Secretion has relatively little effect on man (11, 13, 41, 102).

CLASS REPTILIA: TURTLES

SPECIES AND DISTRIBUTION. *Eretmochelys imbricata* (Linnaeus), Arabia, Malay Peninsula, Malay Archipelago, Australia, Formosa, Samoa, Guiana, Bahamas, Guatemala. *Dermochelys coriacea* (Linnaeus), Cape of Good Hope, Indian Ocean, New Zealand, Solomon Islands.

Disease. Turtle poisoning.

Symptoms. Onset: almost immediately to a week. Reported symptoms are diarrhea, boils, fever, hallucinations, marked debility, nausea, vomiting, sore throat and lips, and irresistable somnolence. Coma and death may occur within 12 hours after ingestion; death may be delayed until 2 weeks after ingestion (16, 110, 124).

Treatment. Unknown.

Chemistry or Toxicology. Nothing known.

CLASS MAMMALIA: MAMMALS

SPECIES AND DISTRIBUTION. *Neophoca cinerea* (Peron and Le Sueur), South Australia. *Erignathus barbatus barbatus* (Erxleben), coasts of southern Greenland, Iceland.

Disease. Seal poisoning.

Symptoms. See *Thalarctos maritimus* Phipps.

Treatment. See *Thalarctos maritimus* Phipps.

Chemistry or Toxicology. See *Thalarctos maritimus* Phipps.

SPECIES AND DISTRIBUTION. *Thalarctos maritimus* Phipps, Arctic regions; Greenland.

Disease. Poisoning from bear liver.

Symptoms. Onset: 2 to 8 hours after eating. Drowsiness, sluggishness, irritability, vertigo, severe headache and vomiting, diarrhea. In 24 to 48

hours, the skin often begins to peel around the mouth. Peeling may be confined to the face or spread over the whole body. Flesh said to kill dogs in 20 minutes (23, 109).

Treatment. Unknown.

Chemistry or Toxicology. Bear and seal liver was found to be high in vitamin A. Toxicity is at least closely associated with the vitamin in its concentrates. In experiments, one rat succumbed with lesions specific for hypervitaminosis A (109).

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Am. Zool.

Hendrickson 1980

POISONING FILE

Eretmochelys imbricata *(Linnaeus), the hawksbill turtle*

The hawksbill is represented here as another specialized offshoot from the *Chelonia* line, probably separated from the main stem for a longer period than *C. depressa*, but still having a strong genetic similarity to *C. mydas*. Intergeneric mating can produce viable offspring. Earlier this year, I had the opportunity to examine a group of about 18 animals on the Cayman Turtle Farm which were clearly the results of a hybrid cross between *Chelonia mydas* and *Eretmochelys imbricata*. The turtles (something over 2 yr old when I saw them) had hatched from a batch of wild-laid eggs imported from Surinam in a routine shipment for farm stock during the period before the farm stopped all recruitment from the wild. The 5–8 kg individuals showed wide gradations and mixes of characters between *C. mydas* and *E. imbricata* (one wonders if they will be fertile when mature?!).

The hawksbill's strategy for survival seems to be that of a coral-reef-scrounging-omnivore tied closely to coral reef habitats which supply both feeding and nesting requirements within a small spatial range. Most of the adequately documented reports of human poisoning after consumption of sea turtle flesh relate back to this species, no doubt in correlation with its omnivorous food habits. Although there is a regrettable lack of quantitative information on hawksbills from tagging programs, there is general agreement that it is basically a solitary nester which does not engage in distant migration. Bustard (1979) reports hawksbill populations in the Torres Straits which nest on coral cays only a few miles apart, yet are "readily distinguishable on carapace morphology, coloration, and shell thickness." The strategy tying the species strongly to coral reef areas is adequate to explain why hawksbills are found nesting mainly, but not only, on islands. The general pattern of living where the reefs are would provide less consistent isolation from land predators than is common for the other species; this, in turn, conforms with the observations that hawksbills are largely solitary nesters which show exceptional alertness and freedom from stereotypy during the nesting process (Carr *et al.*, 1960).

Caretta caretta *(Linnaeus), the loggerhead turtle*

This species and the two species of *Lepidochelys* belong in Zangerl's (1958) Tribus Carettini and represent what I consider to be the third major strategic choice made by ancient sea turtles—neritic residence with adoption (continuation?) of carnivory (Fig. 1). Between the genera *Caretta* and *Lepidochelys* there seems to have been a two-fold partitioning of resources, dividing both habitat regions and principal food items. Both genera show less evidence of race-formation than do the chelonin genera.

The ecological strategy of the loggerhead appears to be one of antitropical distribution and mollusc-feeding. Although loggerheads range widely and appear to tolerate low temperatures better than any other species except the leathery turtle, their main concentrations are over productive sea bottoms on the eastern sides of continental masses where the general water movement is from the tropics toward cooler regions (exception: the Mediterranean populations). Loggerheads characteristically nest on mainland beaches (exception: the Japanese populations). While there do seem to be migrational movements in this species, they appear to be more a seasonal drift-and-return process than directed movement between distinctly different habitats as in the chelonin mode. The heavy head and jaw structure of the loggerhead are presumably direct adaptations to their molluscan diet, although there are numerous reports of *Caretta* feeding on other prey, including jellies at the sea surface and mangrove leaves in the shallows.

Lepidochelys olivacea *(Eschscholtz), the olive ridley turtle*

So little is known of the non-nesting life of the olive ridley that any attempt to describe its full ecological niche, or strategy, must necessarily resort to educated guess-

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NOTES ON A RARE CASE OF TURTLE POISONING

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ABSTRACT

A rare case of turtle poisoning which occurred in a Muslim community at Cotabato, Mindanao, is reported. The meat, eggs and entrails of a sea turtle normally found in the locality were boiled and eaten, after which 11 individuals, as well as dogs, who ate the entrails died. Description of the turtle thru letters showed that its possible species is *Eretmochelys imbricata*, the hawksbill turtle. Although a previous case of poisoning was reported from a nearby locality, yet no accurate data were taken for comparison.

The symptoms noted showed the neurotoxic action of the poison which was similar to other cases previously reported in the literature. The epithelium of the buccal cavity was severely affected.

Samples of the turtle meat were salted and sent to the Manila Health Office for analysis, but it failed to show the symptoms of poisoning when fed to rats.

A review of literature of cases of turtle poisoning was made. A previous case in Cebu, Philippines (1917) was cited although Taylor (1921) believed that it was the green turtle, *Chelonia mydas* L. which caused the poisoning. The symptoms exhibited in these two Philippine cases were more or less identical.

This paper is a report on a rare case of turtle poisoning which occurred in Rosa community, Dinaig, Cotabato, Philippines.

On February 5, 1954, there was a gathering of Tirrurays, a Moslem community in Barrio Rosa, Dinaig, Cotabato, in connection with spiritual rites for a deceased. In this celebration, the guests, including their dogs, were served with meat, eggs and entrails of a sea-turtle, commonly eaten in said locality and known as *payukan*. The local name for this turtle is hawkbill turtle, *Eretmochelys imbricata* Pennant. The turtle's meat was prepared in the usual way as used by the natives wherein pieces of meat were boiled. Of the people who partook of this boiled turtle's meat, a total of 11 individuals, eight males and three females, died. Likewise, many dogs, which ate the entrails of the turtle, died also.

The approximate carapace size of the turtle was about one meter in length and about 61 cm. in width. As described (per comm.), this turtle had thin scutes which indicated that it had been previously caught while still young, its scutes having been removed and then thrown back into the sea, because it is locally believed that it would develop another set of scutes. The scutes of this turtle are commonly known as the *tortoise shells of commerce*, and it is the common practice in the locality to return the tortoise to the sea after removing the scutes by heating the carapace over the fire until the shell is loosened.

In another adjacent community in Kimini, Cotabato, the same kind of turtle, with a new growth of scutes, had caused a similar poisoning during the same season. Inasmuch as this is the only turtle wherein scutes are removed for commercial purposes, we believe that the identity of the species is, more or less, accurate. Furthermore, the egg diameter (2.5 to 3 cm.) is the normal size of this turtle.

Symptoms.—A few hours after partaking of the cooked meat, the victims complained of hot sensations at the region of the abdomen, accompanied by cold sensations at the extremities. A strong feeling of nausea ensued which caused some of the victims to vomit a portion of the food they ate. This condition persisted until the following day when dizziness then set in, accompanied by degeneration of the sense of balance, together with blurred vision and a feeling of sleepiness. To some of the victims, vomiting and diarrhea had been very severe. After 3 to 4 days in this condition, the mucous membrane of the mouth and throat of the victims became red and swollen; becoming sore later. These membranes assumed the appearance of having been scalded with hot water. The tongue, particularly, became heavily coated with a whitish membrane. Because of this condition of the mouth, throat, and tongue, the victims had extreme difficulty in eating and even in drinking. Some patients complained of frequent urination of highly colored urine. Of the victims who partook of the feast, six children, ranging from 1½ to 4 years old, one young man, aged 35, and three older people, 50 to 60 years old, died.

Eight days after the poisoning, the District Health Officer of Cotabato, who was called to attend to the cases, advised the patients to gargle warm water with normal saline solution to prevent further infections of the mouth.

Those who were still alive at the time were very weak and were given a liquid diet of boiled rice, and tea or coffee. Those

who were seriously ill were given muscular injections of caffein and sodium benzoate.

The Health Officer sent samples of the turtle meat to the Manila Health Office for analysis. Furthermore, he gave the clinical data for which we are greatly indebted. The meat was fed to laboratory rats, but these did not show any symptoms of poisoning. The meat was heavily salted and dried. However, it was washed to remove most of the salt before boiling in water. Both meat and water were given to the animals. Reports from the Public Health Research Laboratories, Manila, indicated negative results as far as active ingredients were concerned. Rats fed with the meat, which was boiled after washing, were not affected at all. It is possible that the salt used to preserve the meat might have destroyed the poisonous nature of the flesh.

This information was verified by the Municipal Health Officer of Cotabato.

Inquiries made in Zamboanga City indicated that there is no known case of turtle poisoning in that City where green turtles (*Chelonia mydas*, L.) are butchered regularly throughout the year. The Muslim population there relish the meat of this reptile without any mental reservation. That no reported case of turtle poisoning in that city where a lot of marine turtles are being butchered more than in any other part of the Philippines is therefore interesting.

Review of Literature:

The earliest record of turtle poisoning was from the New World. It took place at Saint Jacques, Windward Islands, North America, way back in 1697, as reported by the monks, Damplier and R. P. Labat, in 1724. Labat stated that Jean Montididier, also a monk, bought a tortoise plastron of probably *Eretmochelys imbricata* Pennant, and in spite of his warning, ate as much as he could. As a result, after four days, he was covered with boils. These were accompanied by a terrible diarrhea and high fever for which he suffered for eighteen to twenty days. Father Labat himself, who took caution in partaking of the turtle's meat, had a little diarrhea for six days, accompanied by three boils.

Cleland, sometime in 1845–47, cited from Banfield's "Confession of a Beachcomber" that in some localities in northern Queensland, Australia, the flesh of the hawksbill turtle, *Eretmochelys imbricata* is said to be imbued with a deadly poison,

so that care should be exercised in the killing and butchering of this animal, lest a certain gland, located in the neck, be opened. The poison is so toxic that flesh cut with a knife, which has touched the critical part, becomes impregnated with the poison. One old seafarer acknowledged that he nearly "pegged out" after a hearty meal of the liver of the hawksbill.

"Banfield also states that the flesh of the luth or leathery turtle, *Dermochelys coriacea*, also causes symptoms of poisoning.

Chevellier and Duchesne (1851) reported that the hawksbill turtle, *Eretmochelys imbricata*, is not good to eat; that it has a special purgative quality according to Damplier and Labat, (*op. cit.*), and that, when eaten, one may be "certain of being covered with boils," if there are some impurities in the body. But if eaten sparingly, the flesh can cure some diseases. When the flesh of this turtle is salted, it loses its purgative effect.

Tennent (1861) reported that the flesh of sea turtles caught in the southwest coast of Ceylon during certain seasons is avoided because they are poisonous. Tennent cited a case of poisoning at Pantura, near Colombo, Ceylon, where in October, 1840, twenty eight persons who partook of a sea turtle were seized with sickness immediately after which a coma supervened and 18 died during the night. Those who survived related later that the flesh of the turtle was fatter than that of the ordinary. He also stated that similar fatal occurrences had been attributed to turtle curry, although there was room for believing that the poison might have been contained in some other ingredients.

The first Philippine report on turtle poisoning was made by Taylor (1921) and cited that the turtle alluded to in Sir Tennent's report (*op. cit.*) was a *Chelónia virgata* [*Chelonia japonica* (Thunb.)]=*Chelonia mydas* (Linnaeus). Taylor (*op. cit.*) cited a case of turtle poisoning in the Philippines, presumably by *Chelonia mydas*. It occured in November, 1917, in Bantayan Island near Cebu. Fourteen deaths were reported out of 33 cases. The victims suffered from pain in the throat and lips vomiting, and drowsiness. Two of those who died had their symptoms very much delayed, coming out after 8 days of partaking of the turtle's meat. Nevertheless, they had symptoms of poisoning similar to those who died earlier and they died after 6 days. It was also reported that relapse in this case occurred even after cure had set in. The

tendency to drowsiness from the moment symptoms appeared was also manifested, and even if improvement under treatment appeared later, still the victims died. It was noted that there is a great similarity of symptoms between the poisoning of 1917 in Bantayan Island and that of the present case. Read (1917) had scattered references to turtle poisoning but without proper identification of the real species.

The similarity of these two recorded cases of turtle poisoning in the Philippines indicates a strong credence that there is really a poisonous substance at times in the meat of some sea turtles which are caught in the Philippines that causes death, although the particular species is not clearly verified in all cases.

It should be noted that like the report of Chevellier and Duchesne in 1851, the flesh of the poisonous turtle, when salted, loses the purgative effect. The flesh of the turtle in the present case showed the same findings when a piece of its meat was sent to the Public Health Research Laboratories in Manila for analysis, just as a sample of meat from the 1917 poisoning was also sent in for examination. In both cases, the meat was heavily salted and dried, and the examination of the flesh failed to reveal the presence of any known poison.

It was noted that, inasmuch as the diarrhea following the eating of turtle's meat had a very dehydrating effect, most of the victims who died in the present case were both young and old. Although one of the dead was 25 years old, this supported the observations of Chevellier and Duchesne: "It is necessary to be of strong, robust nature to resist this evacuation." That the flesh samples delivered at the Government Laboratory in Manila did not have any trace of poison or any effect on experiment rats may be answered from the report of Chevellier and Duchesne (1851) that, if the flesh of the hawksbill turtle has been salted, the purgative effects of poison are lost.

Deraniyagala (1939) noted that the flesh of the leather back turtle, *Dermochelys sp.*, is also reported to possess faintly toxic properties during some seasons when taken on the east coast.

From the review of the literature, it is more or less, concluded that the sea turtle which caused the mass poisoning in Barrio Rosa, Municipality of Dinaig, province of Cotabato, is of the spicies *Eretmochelys imbricata*.

ACKNOWLEDGMENT

We are indebted to Mr. Amado B. Diaz, Municipal Health Officer, Cotabato, Cotabato, for supplying some pertinent information, leading to the indentification of the species of the turtle, and also the report on Sitio Kimini. We are also indebted to Dr. Brune W. Halstead, M.D., for sending us copies of the rare articles on turtle poisoning.

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TURTLE MEAT (*ERETMOCHELYS IMBRICATA*) POISONING IN NETHERLANDS NEW-GUINEA *

by

T. ROMEYN & G. T. HANEVELD

Public Health Service and Royal Netherlands Navy Medical Corps, Hollandia, Netherlands New-Guinea

INTRODUCTION

The Papuans are keen on hunting turtles in the seas around New-Guinea as their meat is considered a delicacy. LOVERIDGE (1948) described four species of turtles in these waters. Publications from various countries reported on poisoning—usually mass poisoning—by this meat; cases, often ending fatally, occurred in India, Ceylon and the Philippine Islands. As regards the East-Indian archipelago, VAN HASSELT (1922) mentioned poisonous turtles around New-Guinea. The Public Health Service of the Netherlands East-Indies instituted an enquiry into the matter (*Mededelingen*, 1933) and received several positive answers; some doctors reported their personal experience, others from hearsay. The symptoms were dizziness, nausea and vomiting, in a few cases with fatal outcome. BIERDRAGER (1936) recorded an instance of mass poisoning in about 52 people, five of whom died, after eating the meat of a large turtle (of the Cheloniidae species) on the Isle of Japen north of New-Guinea. The pathologist, BONNE (cited by BIERDRAGER, 1936), found fatty degeneration of the liver and kidneys. SIEGENBEEK VAN HEUKELOM (1936) also described degeneration of the liver, pancreas and kidneys.

PERSONAL EXPERIENCE

On August 24, 1954, a number of people in Kaipuri (Schouten Islands) ate turtle-meat. The government doctor arrived a week later; in the meantime one man had died after two days and another after six days. Four others suffered from vomiting, cramps and unconsciousness one week after the meal; they took a long time to recover.

All cases of turtle-poisoning in Netherlands New-Guinea and Indonesia were caused by *Eretmochelys imbricata* (hawk's bill turtle, vernacular name *tuturuga sisir*). This turtle may reach a length of 85 cm and a weight of about 20 kg; the jaws are curved like a bill or beak—hawk's bill—and the head has four prefrontal horn plates. The carapace has four overlapping horn plates, in contrast to *Chelonia mydas* (green turtle or soup turtle).

SYMPTOMS

(1) *General:* The symptoms usually begin about twelve hours after the meal: a feeling of distress often coupled with vomiting, dizziness and a burning sensation in the throat, sometimes also of the tongue, gums and lips. Headache and abdominal

* Originally published in Dutch in *Nederlandsch Tijdschrift voor Geneeskunde*.

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pain, sometimes diarrhoea. Mild cases may show only a few of these general symptoms.

(2) *Mouth and throat* affection becomes manifest after about two days; the tongue is white-furred, the mucous membranes of the mouth and throat are swollen and sometimes swallowing becomes difficult. From two to ten days afterwards red elevations, the size of a pin's head, appear on the tongue; these are the swollen papillae, especially near the point and along the edges. The papillae may still be visible after two months.

(3) *Nervous system:* somnolence occurs at an early stage. The patients may react when spoken to, but they fall asleep again immediately. This somnolence may lead rapidly to death. Occasionally the patients are restless. There is thermolability; some authors mentioned hypotony and hyporeflexia, but others did not observe these symptoms.

Course and prognosis. The severity of the affection is proportionate to the amount of meat eaten, but even in serious cases the prodromal symptoms do not start until 12 hours after the meal, though they are more intense then. Vomiting stops after a few days.

The light cases show the mouth and throat symptoms only; in severe cases the clinical picture is dominated by disturbed consciousness. None of the nine somnolent patients seen by BIERDRAGER (1936) recovered. One of the Kaipuri patients was unconscious for a week and then recovered slowly. In cases of prolonged somnolence the development of ulcerating stomatitis is possible, with intense *foetor ex ore*. KARIADI (*Mededelingen*, 1933) reported such a case; it ended fatally.

Treatment. In the first stage of the disease the stomach and intestinal tract should be emptied by emetics, gastric lavage, clysters and/or laxatives. In the later stages treatment can only be symptomatic, by administration of excitants. The symptoms of the mouth and throat may be alleviated by boracic glycerin and rinsing with permanganate of potassium.

What is the cause of the toxicity of the hawk's bill's meat?

Some authors assume that the turtle's meat is poisonous in certain seasons only; this is probably incorrect as cases have occurred in nearly every month of the year. Possibly only a certain variety of the species is poisonous, as is the case in many kinds of fishes. For the West-Indian *ciguatera*, a well known type of fish poisoning, one of the theories is that the toxin is the result of the dietetic habits of the fish (ARCISZ, 1950).

The hawk's bill is a carnivore; with its sharp beak it tears crabs, molluscs, perhaps also seaweed from the coral-reefs. Several toxic and irritating species exist among the coral fauna and vegetation, as evidenced *e.g.* by coral dermatitis manifesting itself by redness, oedema and cutaneous paraesthesia. Both fish and turtle poisoning might derive its origin from coral fauna or vegetation; extensive coral formations exist near all localities where turtle poisoning has been reported.

Neither a bacterial origin of the toxin nor a relationship with toxic products from the generative organs of the turtle have as yet been proved. Thus the problem of poisoning by turtle meat is still unsolved.

SUMMARY

In Netherlands New-Guinea, two patients died two to six days after eating turtle

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meat; a week after the meal four other persons developed symptoms, including vomiting, cramps and unconsciousness; recovery was very slow.

Comparison with cases described in the literature reveals that there are two important groups of symptoms, *viz.*, symptoms of the mouth and throat (swelling, redness of the buccal mucous membranes, white coating, and protracted swelling of the papillae of the tongue) and nervous disturbances, especially somnolence.

In the case of nervous disturbances the prognosis is less favourable. All cases were attributable to the carnivorous hawk's bill turtle (*Eretmochelys imbricata*). The toxicity of its meat is probably due to the poisonous coral vegetation on which the turtle feeds.

Treatment is chiefly symptomatic.

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FAX MESSAGE

FDG: 7/57

17 September, 1991

George H. Balazs
Marine Turtle Research
National Marine Fisheries Service
Honolulu Laboratory
Hawaii.

Fax No: (808) 943 - 1290

Dear Balazs,

Ms Tekinaiti has asked me to enquire from the family that were affected by turtle poisoning late in 1986. The report is from the woman who handled the turtle so the information should be very precise.

To ease the identification of the turtle the pamphlet that you sent was shown to them and were advised that it was much bigger than the hawksbill, with both fore and hind flippers appear to be reddish in colour.

The turtle was sighted when members of this household went diving using harpoons on the reef. They grabed the turtle from the head and they were shocked to hear it crying. They took it ashore and it died the next morning. The pancreas (which appear to be orange in colour) was cooked that afternoon with soy sauce and onions and they ate it.

Two days later the children had severe stomach aches at different time intervals. 4 children died : a 2 year old, a 3 year old, a 4 year old and one infant (less than one year who was breastfed). More than ten people were greatly affected.

The family claimed to have thrown away the shell or the skull straight after killing it. Do not hesitate to contact me should you require further information.

Regards,

Taniera
T.Taniera (Ms)
for Chief Fisheries Officer.

Would appreciate if you could [illegible] the pamphlet as per para 2 above.

Turtle poisoning p.435 1964

Ciguatera and Other Marine Poisoning in the Gilbert Islands[1]

M. J. COOPER[2]

AMONG THE ANIMALS that live in the sea are many that may be poisonous to eat; these animals include fish, sharks, crabs, molluscs, and turtles. Of all marine animals the most important are fish, which are for so many people an essential source of food. There are a number of different ways in which teleost fish may be poisonous. Some fish are naturally poisonous; puffers for instance are always toxic. Some species of fish can be poisonous at certain seasons; in Fiji there is a species of sardine which may be deadly poisonous in the later months of the year. A third type of poisoning is found where some fish are poisonous to eat when they are caught on certain reefs or parts of a reef, and yet when caught on other parts of the same reef, or on nearby reefs, are perfectly safe to eat. This type of poisoning, known as ciguatera, is common throughout the tropical Pacific, usually on oceanic islands and isolated reefs.

Ciguatera is not, as many people think, a recent development. Captain Cook, in the journal of his second voyage to the Pacific in 1772–1775, relates how all of his officers who ate "two reddish fish, about the size of bream and not unlike them" were poisoned and the pigs, that were given the offal, died. These fish were taken in the New Hebrides, and Cook refers to an earlier record of poisonous fish in those waters when he remarks that these reddish fish must be the same kind as those mentioned by Quiros, and called by him "pargos." Pedro de Quiros was in the New Hebrides in 1606. However, prior to World War II there were few reports of ciguatera poisoning in the Pacific; cases of poisoning did occur, but unless a stranger to the Pacific was involved little notice was taken. During and after World War II attention was drawn to the problem, as there were many more people in the Pacific who were poisoned by supposedly good food fish, often in areas where toxic fish had been previously unknown.

Although the symptoms of ciguatera poisoning, the species of fish likely to cause it, and many of the areas harboring toxic species have been recorded, several aspects of the problem still remain to be solved. In spite of recent research into ciguatera poisoning an antidote to the poison, a field test for distinguishing a toxic fish from a nontoxic one, the true nature of the toxin, and the cause of the development of ciguatera among fishes have not yet been discovered.

This paper is a review of the history and location of ciguatera poisoning in the Gilbert Archipelago and of the various Gilbertese beliefs about marine poisoning, together with identifications of the species considered toxic by the Gilbertese, and some of the author's opinions on the development, cause, and spread of toxicity.

The Gilbert Islands are a group of 16 atolls lying north and south of the equator; latitude 3° N passes through the most northerly island and latitude 3° S passes a few miles south of the most southerly island. The group lies between longitude 172° and 173° E of Greenwich. From north to south the 16 atolls are Makin, Butaritari, Marakei, Abaiang, Tarawa, Maiana, Abemama, Kuria, Arunuka, Nonouti, Tabiteuea, Beru, Nikunau, Onotoa, Tamana, and Arorae. Atolls are of two distinct kinds, lagoon islands and reef islands. A simple lagoon island consists of a lagoon, a body of fairly shallow water set off from the ocean, according to tradition, by a ring of small islets; in fact, the islets are usually in a chain lying on the weather side of the lagoon, with submerged barrier reefs on the lee side. A simple reef island is a small island with a fringing reef round it and no enclosed body of water. Many islands appear to be a mixture of both types. The total land area was estimated

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by Dr. Rene Catala at 114 square miles. The population in 1947, the time of the last census, was 27,000; in 1958 the Gilbertese population was estimated to be 32,652 (Doran, 1960).

The information presented here was collected during the period 1953–1962 while the author was resident in the Gilbert and Ellice Islands Colony with her husband, who was an administrative officer with the Gilbert and Ellice Islands Colony Government. Residence was maintained for varying periods of time on Tarawa, Christmas Island (in the Line Islands), and Ocean Island; personal visits were made to all the Line and Phoenix islands, including Washington Island, and to almost all of those in the Gilbert group. During this time the author learned the Gilbertese language, which permitted her to gather information directly from the islanders.

In the course of a study of the scientific equivalents of the Gilbertese names for fish it was found that while some names would encompass all members of a whole family of fish, other names were restricted to a single species, and some names defined the development stages of a generic group. Through the initial study of Gilbertese names for fish, the author became interested in Gilbertese traditions and customs associated with fish, and finally in an investigation of fish toxicity in the archipelago.

The intimate association of the Gilbertese with the sea, almost their only source of dietary protein and fat, makes them reliable givers of factual information about fish poisoning. This dependence upon the sea means that every adult member of a community must have a basic knowledge about the reefs and the fish species around his island, particularly in the area of his village. Although in recent years the traditional dependence on fish as a major source of food has been lessened to some extent by introduction of imported foodstuffs, the detailed knowledge of environment has been preserved and is still known by the elders of the Gilbertese community who are the traditional custodians of natural lore. These "old men"—the term in Gilbertese is traditionally one of respect—have proved to be the most fruitful source of information when dealing with the history of fish toxicity. Younger men, active fishermen, have provided information on the species caught and the areas fished.

Due to the restricted nature of the Gilbertese diet, there are very marked preferences for certain species of fish. Fish considered to be very fatty or greasy are greatly sought after, because the Gilbertese at times develop a craving for animal fats. These sought-after species include *Lutianus bohar*, *Lethrinus variegatus*, *Acanthurus xanthopterus*, *Epinephelus fuscoguttatus*, *Cephalopholis mineatus*, *Myripristis* spp., *Chanos chanos*, and Muraenidae spp. The larger these fish, the more tasty they are considered to be. Some of these species have been found to be toxic, even dangerously so, in certain areas in the Gilberts. But even if a species is known to be toxic, there comes a time when the Gilbertese find it impossible to resist the temptation of a good fatty meal. This craving for animal fats is not restricted to the Gilbertese. Harry (1953) relates that the islanders of Raroia Atoll, in the Tuamotus, were unable to resist eating certain species of fat fish even when they knew that these species were toxic, and that as a result there were frequent cases of poisoning. Population pressure, together with particular food preferences, forces the Gilbertese to continue sampling a known toxic area. On account of this, a fairly accurate picture of the evolution of toxicity in an area may be obtained.

Considerable information was collected from Gilbertese visiting Tarawa, from assistant medical officers (graduates of the Fiji School of Medicine), and from officers and crews of the various ships operating in the colony. This information was later checked by the author, who was able to visit all the "toxic islands" with the exception of Tabiteuea and Arunuka, and by her husband, whose duties took him to all the Gilbert Islands. A special visit was made by the author to Marakei to obtain a more detailed picture of a toxic area than was possible when surveying the group as a whole.

ACKNOWLEDGMENTS

It is impossible to list by name all the very many people who have helped me and given me the information contained in this paper, but I should like to express my thanks to all of them. In particular I should like to thank the staff of the Colony Medical Department; Captain E. V. Ward, acting marine superintendent, for infor-

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and Valenciennes) and *Abudefduf septemfasciatus* (Kendall and Goldsborough). These fish are customarily eaten only by the old people—who are forgetful anyway. It was not possible to find out if these fish were at times genuinely "toxic," or merely considered so on account of their habits.

Scombroid Poisoning

There is no evidence of any scombroid poisoning in the Gilbert or Ellice islands. This type of poisoning appears to be caused by a bacterium (Kawabata et al., 1956), which may be found in the flesh of certain scombroid fishes. This microbe reacts on certain chemicals in the flesh of the fish when too long a time is allowed to elapse between catching and cooking the fish. The reaction is quickened by tropical temperatures. In the Gilberts scombroid fishes of various species are frequently caught early in the morning and left in the sun, and later the flesh is salted for consumption the next day. No poisoning has been reported, and it is thought that the scombroid fishes inhabiting this part of the Pacific are not infected with the specific bacterium (Banner, personal communication).

"Castor Oil" Fish Poisoning

On a few islands where the sea is very deep, close to the shore is found the castor oil fish, *Ruvettus* sp. Although this is a favorite food, it has the reputation of causing poisoning from the purgative properties of the oil in its flesh (Fish and Cobb, 1954). The choicest part of the fish is considered to be the roe, which is boiled whole, but the flesh is eaten as well. If the fish is cooked soon after catching, no "poisoning" results. However, the Gilbertese, and in particular the Ellice people, are well aware of its purgative properties; indeed, if there is a prolonged shortage of them, perhaps due to rough seas, the amount of epsom salts sold by the stores increases to quite staggering proportions.

Clupeoid Poisoning

During the time that the author was in the Gilberts, clupeoid poisoning was unreported. However, in November 1962 two children are reported to have died and other people have been taken ill after eating "sardines" (*te tarabuti*) caught off Betio, Tarawa. No details are known, except that there were two separate catches involved.

Some years ago at Bairiki, Tarawa, a woman died after eating what was described as "sardines" (possibly *Harengula* sp.). This woman was the only person taken ill among a number of people who ate the catch. At the time she was blamed for her own death, as she threaded her fish on an old piece of corroded brass wire before cooking them, instead of using a piece of coconut midrib: it was considered that she had died from copper poisoning.

Turtle Poisoning

The hawksbill turtle, *Eretmochelys imbricata* (Linnaeus), is considered to be deadly poisonous throughout the Gilbert and Ellice islands. It is not generally eaten, but occasionally one will be eaten in error, either in mistake for the green turtle or by someone who does not know the hawksbill's reputation.

The poisoning caused by the hawksbill is very severe, and the Gilbertese describe it as being similar to ciguatera but very much worse. It is so rare for anyone to eat this turtle, and so to be poisoned, that none of the assistant medical officers who were consulted had ever seen a case. The details of the following cases were supplied by eye witnesses on whom the severity of the poisoning had made an everlasting impression.

On Arorae, about 15 years ago, a group of people ate a hawksbill turtle. All of them became very ill and five of them died. Their symptoms were described as follows: vomiting; very severe stomach ache, and diarrhea; their skin was "very hot to touch; they were very thirsty, but something was wrong with their mouths and they were unable to drink; they were unable to move their arms and legs; finally, their skin peeled off as if they had been cooked." One man was so severely poisoned that he is said to have died less than a day after he ate the turtle, but even in that short time he peeled. The others died at various intervals, the longest surviving about a week.

The symptoms in a more recent incident on Tabiteuea involving an unknown number of people were described as follows: vomiting; severe stomach ache, and diarrhea; gradual paralysis; flaking skin, leaving great sores, especially

Summary of Toxic Conditions in the Gilbert Islands

ISLAND TYPE		BUTARITARI Lagoon		MARAKEI Lagoon		TARAWA Lagoon		ABEMAMA N. Lagoon		ABEMAMA S. Lagoon		ARUNUKA Lagoon		NONOUTI Lagoon	TABITEUEA Lagoon		BERU Lagoon		NIKUNAU Reef		ONOTOA Lagoon	
ANNUAL AVERAGE RAINFALL (IN INCHES)		125		79		70		57		57		53		50	47		49		44		44	
YEAR TOXICITY COMMENCED		About 1948		1946		Unknown; resurgence 1944–		Unknown (1917?)		Unknown (more than 50 years)		Unknown; but resurgence 1929–37		Unknown (1890?)	Unknown (1919?)		Unknown (more than 40 years ago)		Unknown (more than 40 years ago)		Unknown (more than 40 years ago)	
SPECIES TOXIC IN YEAR STATED	COMMONEST VERNACULAR NAME	1958	1961	1958	1962	1958	1962	1958	1962	1958	1962	1958	1962	1962	1958	1962	1958	1962	1959	1962	1958	1962
Holocentrus spp. (Gunther)	Ku	+		+																		
Myripristis spp. (Cuvier)	Mon			+																		
Epinephelus merra (Bloch), and allied species	Kuau			+											?							
E. corallicola (Cuv. & Val.)	Kuaurang			+		+		+							?							
E. fuscoguttatus (Forskål)	Maneku	+	L+	+	+	+	L+	+	+			+	L+	L+	+	?	+	L+	+	?	L+	?
Cephalopholis argus (Bloch & Schneider)	Nimanang			+	+	+		+	?						+	?	+					
C. miniatus (Forskål)	Nrekereke	+		+	+	+		+	?						+	?	+	+				
Plectropomus truncatus (Fowler)	Rekimoa	+		+	+	+		?	?			?	?	?	+	?	?	?	?			
Variola louti (Forskål)	Bukitakeiau	?		+	+	+		+							+							
Promicrops lanceolatus (Bloch)	Bakati	L+	L+	+	+	L+	L+	L+	L+	?	?	?	?	?	?	+	+	L+	?	L+	?	?
Caranx spp. (undetermined species)	Tauman, Aurua	?		+	L+	L+		L+						?	L+		?					
C. lugubris (Poey)	Aongo	L+		+	+	L+		L+						?	L+	?	?					
Acanthurus spp. (undetermined species)	Riba	?		+	+	+		+	+			?	?	?	?	?	+					
A. triostegus (Linnaeus)	Koinawa			+											?	?						

(undetermined species)																							
A. triostegus (Linnaeus)	Koinawa			+												?	?						

ISLAND TYPE		BUTARITARI Lagoon		MARAKEI Lagoon		TARAWA Lagoon		ABEMAMA N. Lagoon		ABEMAMA S. Lagoon		ARUNUKA Lagoon		NONOUTI Lagoon	TABITEUEA Lagoon		BERU Lagoon		NIKUNAU Reef		ONOTOA Lagoon	
ANNUAL AVERAGE RAINFALL (IN INCHES)		125		79		70		57		57		53		50	47		49		44		44	
YEAR TOXICITY COMMENCED		About 1948		1946		Unknown; resurgence 1944–		Unknown (1917?)		Unknown (more than 50 years)		Unknown; but resurgence 1929–37		Unknown (1890?)	Unknown (1919?)		Unknown (more than 40 years ago)		Unknown (more than 40 years ago)		Unknown (more than 40 years ago)	
SPECIES TOXIC IN YEAR STATED	COMMONEST VERNACULAR NAME	1958	1961	1958	1962	1958	1962	1958	1962	1958	1962	1958	1962	1962	1958	1962	1958	1962	1959	1962	1958	1962
A. lineatus (Linnaeus)	Katawa			+	+	+									?	?						
A. xanthopterus (Val.)	Mako	?		+	+	+		+	+								+					
Ctenochaetus spp. (*striatus* and *strigosus*)	Ribabui	?		+	+	+		?	?						?		?					
Aprion virescens (Val.)	Awai	?		+	+	+									+							
Lutianus raigiensis (Quoy & Gaimard)	Bawe			+																		
L. monostigma (Cuvier)	Baweina			+											+							
L. bohar (Forskål)	Ingo	L+	L+	+	+	+	L+	+	+	L+	L+	+	L+	L+	+	L+	+	L+	L+	L+	L+	L+
L. gibbus (Forskål)	Ikanibong			+	+												+					
L. semicinctus (Quoy & Gaimard)	Tinaemea	?		+	+	+		+	?						?	?	?					
Lethrinus spp. (undetermined species)	Roa			+	?	?									?	?						
Scarus spp.	Inai	?		+	+	?		+	?						+	?	?					
Sphyraena sp. (unidentified species)	Nunua	+	L+	+	+	+	L+	L+	L+	L+	L+	L+	L+	L+	L+	L+	L+	L+	L+	L+	L+	L+
Mugil spp. and other mullets	Aua			+	+																	
Muraenidae	Rabono	+	L+	+	+	L+	L+	L+	?	L+	?	L+	L+	L+	L+	L+	L+	L+	L+	L+	L+	L+

+ Denotes a species which may be toxic.
L+ Denotes a species in which large specimens only are potentially toxic.
? Denotes a species about which there is insufficient information on its toxicity.

The author would like to point out that the above list refers to the more common toxic species in the Gilberts. To discuss the degree of toxicity of all possible species is beyond the scope of this paper.

on the mouth, lips, and in the armpits; intense thirst, but due to the condition of the mouth, inability to drink; finally, the victims died, described as being unable to breathe.

The green turtle, *Chelonia mydas* (Linnaeus), is eaten throughout the Gilbert Islands and has not been implicated in any poisoning.

It should be noted that the hawksbill turtle is primarily a carnivore (Loveridge, 1946), preferring crabs and molluscs, although in captivity they will eat fish as well as seaweeds. On the other hand, the green turtle is primarily a herbivore, grazing many hours a day on beds of *Thalassia* (Loveridge, 1946, citing Deraniyagala, 1939). In captivity the green turtle may prefer animal food (Loveridge, 1946). In the Gilberts young green turtles are sometimes kept until they are large enough to eat, being fed almost exclusively on fish.

The hawksbill and green turtles were and still are Gilbertese family totems. Members of the families concerned will often maintain that all turtles are poisonous.

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The Gilbertese, surprisingly enough, do not make as much use of the various seafoods on their reefs as do many islanders in the Pacific. Sea urchins, again reported by Randall (1958) as causing a ciguatera-like poisoning, are not eaten by the Gilbertese.

During the Japanese occupation some varieties of seaweeds were eaten by the Gilbertese, but as soon as food supplies returned to normal

ISLANDS THAT ARE FREE FROM TOXICITY

ISLAND	MAKIN	ABAIANG	MAIANA	KURIA	TAMANA	AROAE	OCEAN ISLAND
TYPE	Reef	Lagoon	Lagoon	Reef	Reef	Reef	High, reef
RAINFALL	107"	83"	62"	55"	48"	56"	63"
ANCHORAGE (outside lagoon)	Very Poor	Poor	Nil	Poor	Very Poor or Nil	Poor	Moorings only

this ceased. No information is available, therefore, as to the toxicity of any alga in the Gilbert Islands.

SUMMARY

The Gilbert Islands were surveyed for fish poisoning, using local Gilbertese as informants. The following observations were made:

1. Ten out of 16 Gilbert Islands have fish that cause ciguatera.

2. The toxic areas are all found on the western lee sides, and are usually confined to open sea reefs and anchorages in this area, seldom penetrating into lagoons.

3. There is a definite evolution of toxic conditions over the years, with a few species being initially toxic, almost all reef fish being toxic at maximum, and, in the final stages, only large eels, certain snappers, and groupers remaining toxic. This cycle appears to take at least 8 years.

4. Of the other fish in the archipelago, the liver of some sharks, the heads of some mullets and surmullets, the "castor oil fish," and some sardines all have reputations for varying degrees of toxicity. In addition, the hawksbill turtle is also reported to be toxic. At least the shark's liver and the hawksbill turtle produce symptoms somewhat similar to those of ciguatera.

5. Of the invertebrates, only two species of crab and one species of gastropod have been said to be toxic; no other crustaceans or molluscs were considered as being toxic.

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PACIFIC SCIENCE OCT. 1964

and Valenciennes) and *Abudefduf septemfasciatus* (Kendall and Goldsborough). These fish are customarily eaten only by the old people—who are forgetful anyway. It was not possible to find out if these fish were at times genuinely "toxic," or merely considered so on account of their habits.

Scombroid Poisoning

There is no evidence of any scombroid poisoning in the Gilbert or Ellice islands. This type of poisoning appears to be caused by a bacterium (Kawabata et al., 1956), which may be found in the flesh of certain scombroid fishes. This microbe reacts on certain chemicals in the flesh of the fish when too long a time is allowed to elapse between catching and cooking the fish. The reaction is quickened by tropical temperatures. In the Gilberts scombroid fishes of various species are frequently caught early in the morning and left in the sun, and later the flesh is salted for consumption the next day. No poisoning has been reported, and it is thought that the scombroid fishes inhabiting this part of the Pacific are not infected with the specific bacterium (Banner, personal communication).

"Castor Oil" Fish Poisoning

On a few islands where the sea is very deep, close to the shore is found the castor oil fish, *Ruvettus* sp. Although this is a favorite food, it has the reputation of causing poisoning from the purgative properties of the oil in its flesh (Fish and Cobb, 1954). The choicest part of the fish is considered to be the roe, which is boiled whole, but the flesh is eaten as well. If the fish is cooked soon after catching, no "poisoning" results. However, the Gilbertese, and in particular the Ellice people, are well aware of its purgative properties; indeed, if there is a prolonged shortage of them, perhaps due to rough seas, the amount of epsom salts sold by the stores increases to quite staggering proportions.

Clupeoid Poisoning

During the time that the author was in the Gilberts, clupeoid poisoning was unreported. However, in November 1962 two children are reported to have died and other people have been taken ill after eating "sardines" (*te tarabuti*) caught off Betio, Tarawa. No details are known, except that there were two separate catches involved.

Some years ago at Bairiki, Tarawa, a woman died after eating what was described as "sardines" (possibly *Harengula* sp.). This woman was the only person taken ill among a number of people who ate the catch. At the time she was blamed for her own death, as she threaded her fish on an old piece of corroded brass wire before cooking them, instead of using a piece of coconut midrib: it was considered that she had died from copper poisoning.

Turtle Poisoning

The hawksbill turtle, *Eretmochelys imbricata* (Linnaeus), is considered to be deadly poisonous throughout the Gilbert and Ellice islands. It is not generally eaten, but occasionally one will be eaten in error, either in mistake for the green turtle or by someone who does not know the hawksbill's reputation.

The poisoning caused by the hawksbill is very severe, and the Gilbertese describe it as being similar to ciguatera but very much worse. It is so rare for anyone to eat this turtle, and so to be poisoned, that none of the assistant medical officers who were consulted had ever seen a case. The details of the following cases were supplied by eye witnesses on whom the severity of the poisoning had made an everlasting impression.

On Arorae, about 15 years ago, a group of people ate a hawksbill turtle. All of them became very ill and five of them died. Their symptoms were described as follows: vomiting; very severe stomach ache, and diarrhea; their skin was "very hot to touch; they were very thirsty, but something was wrong with their mouths and they were unable to drink; they were unable to move their arms and legs; finally, their skin peeled off as if they had been cooked." One man was so severely poisoned that he is said to have died less than a day after he ate the turtle, but even in that short time he peeled. The others died at various intervals, the longest surviving about a week.

The symptoms in a more recent incident on Tabiteuea involving an unknown number of people were described as follows: vomiting; severe stomach ache, and diarrhea; gradual paralysis; flaking skin, leaving great sores, especially

on the mouth, lips, and in the armpits; intense thirst, but due to the condition of the mouth, inability to drink; finally, the victims died, described as being unable to breathe.

The green turtle, *Chelonia mydas* (Linnaeus), is eaten throughout the Gilbert Islands and has not been implicated in any poisoning.

It should be noted that the hawksbill turtle is primarily a carnivore (Loveridge, 1946), preferring crabs and molluscs, although in captivity they will eat fish as well as seaweeds. On the other hand, the green turtle is primarily a herbivore, grazing many hours a day on beds of *Thalassia* (Loveridge, 1946, citing Deraniyagala, 1939). In captivity the green turtle may prefer animal food (Loveridge, 1946). In the Gilberts young green turtles are sometimes kept until they are large enough to eat, being fed almost exclusively on fish.

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MINISTRY OF NATURAL RESOURCES DEVELOPMENT
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Date: 7/30/91

George H. Balazs
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National Marine Fisheries Service
Honolulu Laboratory
Hawaii.

Fax No: (808) 943 - 1290

Dear Mr G.Balazs,

Thank you for your fax with photos received yesterday and today.

Today we went back to that family at Ambo for the shell or the skull and were advised that they threw away the shell straight after killing it. We also enquired to their neighbours for the shell but couldn't find it.

During our first visit to that family we brought with us the pamphlet that you sent with your previous correspondence and were advised that it was not the common turtles (green turtle or hawsbill turtle) that they normally encounter but a different only and they pointed to the logger head turtle. They described it as the one with the very long neck, its middle scutes forms a characteristic uniform hump on the back and with the sides scutes flattens out.

An investigation was carried out in 1979 by Fisheries Divison with questionnaire and identification sheet provided by USP with the dual purpose of collecting information from the fishermen the type of turtle species found and utilization in Kiribati. It was reported from that survey that four turtle species were found in Kiribati namely:

	Kiribati Names
(i) Green turtle	Te On
(ii) Hawksbill turtle	Te Tabakea or Te Borauea
(iii) logger head	Te On n ae
and (iv) Pacific Ridley	Te On mron

Tomorrow will try and obtain information from the family that were affected by the turtle poisoning late last year and report back.

Yours sincerely,

T.Tekinaiti (Ms)

1979

CIQUATERA CASES

Two cases were told from the islands of Nikunau and Arorae where after eating of the fatty tissues of the turtle, most liked by the people, these people vomited with bowels, frothing at mouth, stiffenning of limbs, skin peeling as if after burning with eventuall death.
The Hawksbill turtle was noted as the ciquatera cause. Many people abandoned the catching and consumption of turtles after these cases. Cooper (1964) also reported of the Arorae Island case with details of ciquatoxic symptoms.

It is interesting to note though that hawksbills (Te Tabakea) are not usually taken by turtle divers and those who follow the advice of the elderly people, as 'Te Tabakea' is considered sacred, (a totem creature) in connection with Gilbertese history. It has served its purpose as well as a deterent for a potentially ciquatoxic animal.

USE OF TURTLE

Turtles are taken for food where the meat and intestines are used. Ways of cooking these, (outline in Temaribo, 31/12/1975) plus other factors shows others who do not normally eat these, to consider catching the turtles. The meat sells at $0.50¢ per lb. at the Local Produce Division Very little is sold as the turtle is usually kept alive until required by each family. However, turtle meat is taken relatively less than fish.

The turtle shield is used primarily for decorations and ornaments. The 40-50 cm shield now sells at $8.00 at the Local Produce Division. Two of the divers in Tarawa go out catching turtles mainly to sell the shield. Kuria island is the main area beside Tarawa where the local produce obtains turtle shields for sale. However, supply is as when demanded.

PREDATION AND LEGISLATION

Quite efficient methods of catching the turtle by man is one of the major predator on the eggs and turtle itself. However, knowledge of catching of nesting turtle is not so widely known. Also peo;e normally prefer fish, only fiarly recently with shift of population to Tarawa, other foreign boats exploiting it, more efficient methods of catching and the growing tendency for export of shield has the increase in effort for turtle hunting been noticed.

Natural predation is reported by (Cross, 1978) where a tiger shark

MARSHALL ISLANDS JOURNAL Volume 21, Number 39, Friday, September 28, 1990

Four dead after eating turtle at party

Tarawa — Turtle meat, a Kiribati delicacy, is an enemy sometimes. Four people, three of them children, died after eating turtle meat at a family party at Abemama Island in the Central Kiribati.

Medical reports said the meat was poisoned. The kids died on the island immediately and the fourth, a man named Tetaua Atanibeia who was flown to Tarawa for medical treatment, died a few days later.

It's never been established what type of turtle they had for the meal. But a few years back on South Tarawa a similar accident had taken place. A family was also enjoying their meal of turtle meat when two of the eaters, small children, started vomitting and later died at the Central Hospital.

In this case the turtle was caught a few days before it was cooked, and some people said the blood could have poisoned the meat because the sea mammal was about to die before it was cooked. Still many people are still puzzled and would like to be advised before they dig into their favorite again.

Eight battle for Christmas Island seat

Tarawa — Eight candidates will contest a vacant seat in parliament for Christmas Island later this month.

Among them are two former MPs from the island- Moiaua Toariri and Tabwi Teatata. The other six are new faces in the Christmas Island political arena.

Polling day is this week Thursday, and the newly elected MP would be able to participate in the next sitting of parliament before the end od the year.

The next sitting includes a budget address. The new general election is next year.

Former MP for Christmas Island, Bwebwe Manikaoti, resigned before the August sitting of parliament. There were no official explanations for his resignation.

Catholics promote good marriages

Tarawa — The Catholic Church in Kiribati has established a Marriage Encounter Association, that will promote happy marriage to members of the

New FADs for outer island fishermen

Tarawa — It has been reported that people in the outer islands would like to see more FADs, fisheries aggregating devices, anchored off their islands so they don't have to go further out in the ocean for catching fish.

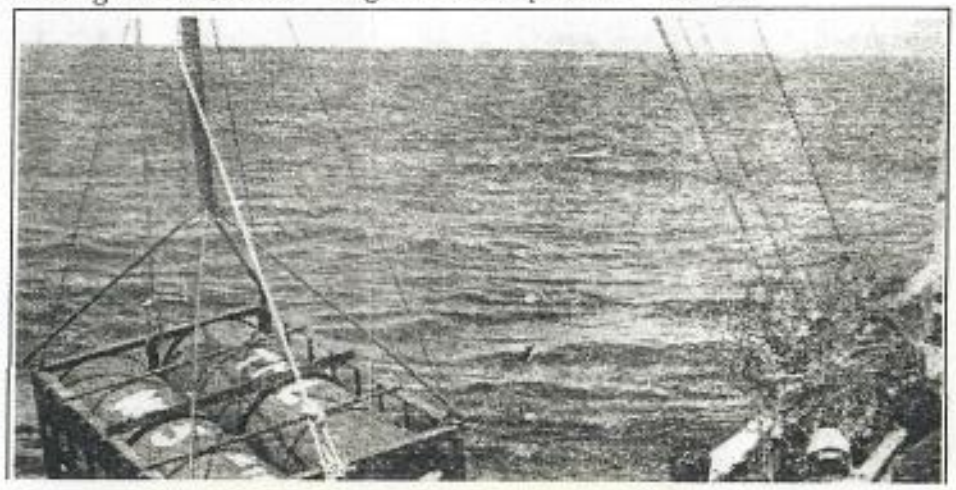
Outer island fishermen experienced the importance of these devices when Temautani and Fishing Division embarked on a mission to supply some of these floats to fishermen on the outer islands.

But most of the floats did not last long. They went mission somehow, and a fisheries officer ashore where it was located.

They caught many types of fish, including some they rarely see around their island waters. But the floats went missing after two weeks or so, and never have been replaced.

The Fisheries division saw and understands how much these FADs are needed but the cost of one alone is quite high and it's impossible to plant each island with one immediately.

The Fisheries officer said, depending on money available, one by one will be planted but all in one go because of its cost.



AMERICAN SAMOA GOVERNMENT
PAGO PAGO, AMERICAN SAMOA 96799

LBJ TROPICAL MEDICAL CENTER

In reply refer to:

PER 4-2
Serial:0215
JPT/jon

November 24, 1982

George H. Balaz
Fishery Biologist
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Honolulu, Hawaii 96812

Dear Mr. Balazs:

Dr. Edward Le'iato, one of our Samoan Medical Officers remembers the occasion when people on Aunu'u were poisoned from eating turtle flesh. He was one of the Physicians that took care of them at the old Hospital in Utulei.

1. He does not remember the month or the year, but thinks it was in the late 1950s or early 1960s.

2. About 100 patients received treatment. All of the patients recorded. Recovered.

3. Symptoms were nausea, vomiting, anxiety. Some had diarrhea and some were in shock with lowered blood pressure.

4,5&6. He has no memory or knowledge as to the kind of turtle eaten or under what circumstances the turtle was captured.

I regret I am not able to obtain more definite information about the incident.

Sincerely,

James P. Turner, M.D.

JAMES P. TURNER, M.D.
Deputy Director
Dept. of Health Services

American
Museum of
Natural
History

31 January 1986

Mr. George Balazs
NMFS
Honolulu Laboratory
Honolulu, Hawaii

Dear George:

Thanks very much for sending the list of references on turtle poisoning. There were several I hadn't heard of. I enclose a copy and translation of the Kinugasa and Suzuki (1940) paper. It is the one about Taiwan - if that wasn't clear in my letter (I didn't keep a copy). I am still waiting on the one from Tahiti. I think the guy is being a creep. About Tonga, I don't think that you sent me a copy of the SPC Fisheries Newsletter, at least I can't find it anywhere.

By coincidence, your list arrived the day before I met Anders Rhodin. We worked together on post-mortem exams on the Long Island ridleys. The autopsies weren't very revealing, but the organization that picked up the turtles was very keen on doing this from their experience with marine mammals. The specimens are now at the AMNH and I think I will be working up the data on the cold-stunning episode for Okeanos Research Foundation. We'll see.

I may be able to get the Bierdrager paper translated by a museum volunteer who speaks Dutch and used to live in Indonesia. If I succeed, I'll send you a translation.

I really find myself scrambling for time these days. After not working for a while, I now have two teaching jobs, on opposite ends of Manhatten. They keep me hopping. One is a lecture course on the evolutionary history of the earth at an arts college, the other a genetics (ugh) lab at Barnard College, the women's part of Columbia. The students are as different as night and day--punkers and preppies! What an experience. The rest of the time I'm at the AMNH working on sea turtles. New York is really a fun place to be for a while, but I'm sure I'll enjoy more space when we move on.

Thanks, again, for sending me your poisoning references. They will be a big help. I'm keeping my fingers crossed on a post-doc at Scripps that I applied for over Christmas. It would make getting the research funds much easier for the work with the natural products chemist.

Best wishes,

Anne

Anne Meylan

P.S. Have you heard about the poisoning event in Tanzania in early January 1983?(Over)

Central Park West at 79th Street New York, New York 10024, U.S.A. Telephone (212) 873-1300

I heard about it from people who saw it in the newspapers, and then I never ran it down. I thought I'd mention it, in case you have some clippings, or want to have a try at finding it in the AP news around that time.

P.P.S. Thanks for the info on poisoning from Johannes. As it turns out, I have extracts from that particular "poison gland" and several other organs that were sent to Clive Wilkinson. I intend to analyze them for the presence of secondary metabolites from sponges, but am waiting to get my connections with Scripps. Interesting info Johannes mentioned. I want to read his book someday, if I can ever get around to it.

Kinugasa, M. and W. Suzuki, 1940. Über untersuchungen der ursache der massenhaften vergiftung nach dem genuss von fleisch einer an der küste von koryo in der prafektus sintiku gefangenen seeschildkrote. Taiwan Igokkai Zassi 39(74): 66-74.

[illegible]

Check Ref. (title)

original attached

Kinugasa
& Suzuki

A study on the cause of the outbreak of poisoning by sea turtle meat in Taiwan

Masaru Kinugasa health technician
Wasaburo Suzuki

Forward

In 1939, 4.26, about 1:00 AM. 2 fishermen caught a sea turtle at an off-shore of Gorju beach, Shinchiku state in Taiwan and served it among friend.

As soon as the news of poisoning was informed we rushed there and did the investigation.

Out of 57 poisoned, 7 died, 9 serious 41 minor.

We found a certain poison. Thus it seems possible that some sea turtle has a certain poison.

1. The clinical symptoms of the patient

a) The symptoms of the not serious patient

Many of them felt sick and heavy head only. The mildest cases were heavy head and sick feeling. But some of them vomited. A less mild cases were dryness inside the mouth, few diarrhoea.

The pulse was generally slow. None had fever.

On the nerve systems, a few cases of enlarged pupils. Eyes, ears, nose and throat were not disturbed. no jaundice.

b) The symptoms of the serious patients

Very slight fever generally and only a few patient noticed it. Almost every patient had heavy head, headache, dry and hot feeling at the mouth, tongue and throat. Also sick feeling, vomitting, though the degree varied by the individuals. The pulse were fast and was contrary to that of the non-serious patients. Quite a few patients had temperature around 37°C and pulse 150. some had diarrhoe, chilly on all over the body, arms and legs were cold. the mucous membranes were generally conjested, enlarged pupils and the reaction of the pupils were weak, The entire body felt very sick. Missed the urine examination.

2) The beginning of the poisoning and the hours required to death

Some patients felt the subjective symptoms in 8 hours after eating, some were in 50~60 hours, and hard to get the average time. But many felt the symptoms in 16 hours.

It depended upon the difference of the physical

constitution, the difference of the amount eaten and ages. Heart medicine and antidote were given but with 48 hours average, patients died.

This disease seemed to have symptoms such as heavy head, hot (burning) feeling in the mouth and throat. pressure in the chest but did not have any particular objective symptoms, pale face and sweat. sirious patient had cold hands and legs.

The unusual thing was, generally no fever or very little rise, yet many had fast pulse. It seemed the circular systems were affected. In spite of the low fever, the heart beated faster. The poison seemed to stimulated the vagus nerves in the beginning and paralize them later.

As a conclusion, the poison seemed to stimulate the vegus nerves with a very little amount and later paralize them.

3. Observation in pathological anatomy

3 examples: 1. 3 year old. 23 hours after death.
2. 9 year old, 16 " "
3. 11 year old. 12 " "

The followings was the summary of the 3.

Stomache

At the small curved area many blood vessels

were quite swollen and scattered spots of effusion of blood along the vessels generally. The mucous membrane were rough and inflamed but no ulcer were found. There were some blood in the food in the stomache, some had bile in it. The blood vessels on the mucous membrane were considerably swollen.

Intestines

The most obvious change were observed in this area. In the small intestines, the blood vessels were considerably swollen everywhere, scattered spots of conjestion, and in every case there were some spots of effusion of blood. Some were like lines, circles or oval shape, from 2 cm diameter to the size of a hat pin, all the kinds of sizes and shapes. Many were seen at the upper part of the small intestines. The duodenum were filled with bile.

Heart

The surface had slightly swollen coronary arteries. Inside had some fluid blood. The valve and the mucous membrane had spots of conjestion, but no unusual change.

Liver

The surface and inside had map-like or tree branch-like conjestion.

Kidney

not unusual

4. With all the circumstances, the sea turtle was believed to be not poisoned

5. The sea turtle was found not rotten.

6. The bacteriological examinations were negative.

7. The chemical examination
There were no salmonella family.
But a certain toxin was admitted, however it was not clear what it was.

8. Animal experiment
The toxin stimulates vagus nerves with a small amount, and slows down the heart beat. With large amount, the vagus nerves will be paralized and heart beat gets faster.
With extreme amount, the heart stops, probably the heart muscle become paralized.

over.

臺灣新竹州後龍海岸ニ於テ捕獲セル海龜肉ノ食用ニ因ル多數中毒發生ノ原因檢索ニ就イテ

新竹州醫務部衛生課(課長下村博士)

衛生技師 衣笠 勝

鈴木和三郎

(昭和14年10月10日受付)

目錄

緒言

昭和14年4月26日午前1時頃臺灣新竹州竹南郡後龍庄外埔海岸沖ニ於テ、同所居住者漁師許大炮及朱某兩名ノ捕獲セル或種海龜肉ヲ食用セルガ、多數(57例、內死亡7、重症9、輕症41)ノ中毒者ヲ出セリト報セラレタルヲ以テ、余等ハ時ヲ移サズ現地ニ急行シ、其中毒原因ニ就キ本論ノ如キ調査ヲ遂ゲタルニ、一種ノ固有毒ヲ認メタリ。仍テ或ル種ノ海龜中ニハ一種ノ固有毒ヲ保有シ居ル可キコトヲ考察シ得タルヲ以テ茲ニ報告セントス。

第1章 中毒患者ノ臨床症狀ニ就イテ

(a) 輕症者ノ症狀

輕症者ノ症狀ハ單ニ惡心並ニ頭重ヲ訴フルニ過ギザルモノ多ク、特ニ神經質ノ患者ニアリテハ多數ノ重症者並ニ死者アルヲ見聞シテ、精神不安ヲ感ズルモノ少カラザルガ、之等ノ者ヲ除キテ思考スルニ、極輕度ノ場合ニハ頭重、惡心ノミナルモノ及嘔吐ヲ發セル者アリ。更ニ稍々症狀ノ强キ者ハ、口腔內ノ乾燥感アルモ下痢ヲ來セルモノ少シ。脈ハ一般ニ遲脈ヲ呈スル者多キモ、發熱セル者1例モナク、肝、脾ヲ觸レズ、神經系統ハ僅ニ瞳孔ノ散大セル者少數ニ認メタルニ過ギス。其他眼瞼及耳鼻咽喉科領域ノ變化及炎症ノ狀並ニ皮膚發血等ヲ認メズ。又黃疸ヲ認メタル者ナシ(第一表參照)。

第一表の一　中毒者ノ臨床的症狀表（輕症者）

氏名	性	年齢	自覺的症狀	他覺的症狀	備考
呂氏来好	女	61	昭和14年4月26日午後6時食同28日朝發病、頭重、口腔咽頭乾燥感時ニ嚥下時咽喉痛覺ノ月部苦悶	熱ナシ Mils (−) Lebel (−) 口腔、咽頭舌ハ帶發赤ス	前掲ノ外陳氏圭 11 歳♀
張氏淵	女	13	臨床的ニ同樣但シ稍々重症ニテ一時ハ意識不明ノ時アリシモ兩次回復ス	熱 36.9 脈搏ノ時ハ速脈 140 回復後遲ニ遲脈 62 ヲ算ス	朱粗波♂ 18 歳
鄭氏素	女	13	4月26日正午食28日早朝發病顯著ノ口腔咽頭乾燥感頭痛胸內苦悶	熱ナシ 脈 60 Mils (−) Lebel (−)	黃氏香♀ 3 歳
朱永成	男	15	頭痛惡心アルモ他ニ異常感ナシ	熱 35.5 脈 60 Mils (−) Lebel (−)	林氏淑♀ 14 歳
陳氏玉霞	女	12	前記症狀ニ一致ス	熱 36.5 脈 58	陳　乾♂ 22 歳
陳氏球	女	9	前上ノ他口腔、咽頭ニ熱感アリ	熱 36.6 脈 56 Mils (−) Lebel (−)	陳夫得♂ 26 歳
吳　氏	女	11	同　前	熱 36.5 脈 58 Mils (−) Lebel (−)	陳氏霞♀ 15 歳
洪氏粉	女	14	同　前	熱 35.8 脈 50	朱　木♂ 6 歳
朱氏素琴	女	7	前上ノ他惡心嘔吐、四肢厥冷、頭重、鼻汁、眩暈、一時起坐不能	熱 37.0 脈 74 Mils (−) Lebel (−)	陳氏敦早♀ 7 歳
朱登山	男	10	同　前	熱 37.0 脈 78 Mils (−) Lebel (−)	黃氏朱♀ 9 歳
					陳有得♂ 66 歳
					ハ惡心頭痛、鼻咽頭ニ乾燥感アリ、他覺的ニハ熱ナク脈ハ遲脈時ニ緩滯ス

第一表の二　中毒者ノ臨床的症狀表（重症者）

氏名	性	年齢	自覺的症狀	他覺的症狀
朱氏玉蓮	女	17	4月26日正午食同28日發病惡心、嘔吐、鼻汁流出、眩暈、舌咽頭、口腔ニ著シキ灼熱感及咽喉痛感アリ、四肢末端ノ厥冷感加チシビレ感強ク一時起坐不能	顏面蒼白、一般皮膚軟弱、顏面冷汗前額ヨリ流下ス 熱 37 度脈ハ 140 內外ヲ算スルモ緊張良不正ハ 3—7—10 ノ間隔ニ來ル
趙氏倫	女	32	前記同樣特ニ四肢末端ノシビレ感強シ	同　前
黃氏好	女	38	前記ノ外胸內苦悶強シ	同　前
朱氏香	女	11	多量ニ食ス前記ノ外一時意識不明ニ陷リ四肢末端麻痺ノ感覺アリ	顏面蒼白、口唇チアノーゼ顏面特ニ前額ノ發汗著シ 熱 37.4 脈頻數 150 微弱
朱氏蘭	女	18	同　前	熱 37.2 脈頻數 190
趙氏足	女	20	同　前	

(b) 重症者ノ症狀

各例ニ就イテ多少ノ差アルモ、大體ニ於テ熱發ハ存在スルモ其ノ程度ハ極ク輕度ニシテ、熱發

ヲ自覺スル者少ナシ。頭重並ニ頭痛、口腔、舌帶、咽頭ノ乾燥感灼熱感ハ共ニ殆ンド全例ニ於テ認メラレ、又惡心、嘔吐モ强弱ノ差ヲ有スルモ、殆ンド全例ニ之ヲ有セリ。脈搏ハ本症候群中特有ナルモノニシテ、發熱ニ比シ遲脈ヲ呈スル事、恰モ腸チフス」ノ症候ニ正反スルノミナラズ、興味アルコトハ輕症者ノソレト全ク相反スル作用ヲ見ルコトナリ。即チ體溫 37°C 内外ニシテ脈搏 150 ヲ算スル者少カラズ、季肋下部ニ於テハ膨滿感アリ、下腹部ニ於テハ腸蠕動運動ヲ喚起スルモノヽ如ク、雷鳴ヲ聞カザルモ下痢ヲ有スル者アリ。口腔内ノ乾燥感、鼻咽頭ノ乾燥感、全身冷感、四肢厥冷ヲ併有スル者少カラズ。又他覺的ニモ之等ハ粘膜面ニハ溢血斑ヲ伴ハザルモ、一般ニ充血セリ。神經症狀トシテ瞳孔散大ヲ來シ、又瞳孔反應微弱、其ノ他全身違和感、重感感强シ、脾ハ觸知セザルモ肝ハ一横指乃至二横指ヲ呈セル例アルモ、一般ニ必發的ニ非ザルガ如シ。同部ノ壓痛ハ各例トモ陰膜共ニ明瞭ナラズ。尿ノ檢査ヲ爲ス機會ヲ逸シタルハ甚ダ遺憾トスル所ナリ。大便モ入手セラレザリシガ、本中毒死ニ因ル解剖死體ヨリ小腸内ノ内容物ニ就テ後述ノ細菌學的檢査ヲ施行セリ。尚腎内蓄溜尿ヲ採リ蛋白ヲ檢シタルモ陰性ナリキ。

第 2 章　中毒症狀ノ發生及其ノ死亡ニ至ル迄ノ時間

該海鼠肉食用ノ時ヨリ初發症狀、即チ自覺症狀ヲ來ス時間ハ各例一樣ナラズ。早キハ 8 時間、遲キハ數10時間ヲ經テ發スル例アリテ平均値ヲ求メ難シ。然レドモ大體ニ於テ平均 16 時間ヲ要スルモノ多シ、是レ個人的體質或ハ食用ノ多少及性、年齢ノ相違ニ因スルハ勿論ナリ。尚死亡ニ至ル時間ニ就テハ、更ニ區々ニシテ發病ニ至ル期間ハ 24 時間ヲ經過セルモ、發病後僅ニ 3 時間 30 分ニシテ死亡セル例アリ。或ハ 80 時間ヲ經過セルモノ或ハ夫レ以上ニ及ブ者アリテ一樣ナラザルモ、一般ニ死亡ニ至ル時間ハ短カラザルガ如シ。勿論强心、解毒等ノ適當ナル醫治ヲ加ヘタルニモ拘ラズ、死亡セル者ノ平均時間ハ 48 時ヲ要セリ。之ヲ要スルニ本疾患ハ自覺的ニハ頭重、口腔咽頭ノ灼熱感、胸部壓迫感ヲ有スレドモ、他覺的ニハ著ルシキ症狀ヲ缺除シ、只僅ニ一般ニ顔面蒼白、發汗ヲ見ルノミナルガ、時ニ重症者ニアリテハ四肢厥冷セリ。而シテ最モ特異トスル處ハ、一般ニ熱ハ缺除スルカ、若クハ極片度ノ上昇ヲ示スニ過ギザルニ反シ、脈ハ速脈ヲ呈スル者多キコトナリ。如斯血行系ニ於テ著名ナル所見ヲ呈スルモノヽ如クニシテ、即チ發熱ニ比較シ心臟頻數ナルコトナリ。恐ラクハ著ルシク本毒作用ノ發現ヲ見ンカ、遂ニハ全心筋麻痺ノ經過ヲ取ルニ非ズヤト信ゼラル。即チ本臨床的所見ヨリシテ本毒ノ作用ハ其ノ始メ迷走神經ヲ鼓舞シ後之レヲ麻痺スルカ、或ハ交感神經ヲ鼓舞シ後之ヲ麻痺スルカニアルベシ、或ハ又心筋自體ニ前記ノ如キ作用ヲ呈スルモノナラン。而シテ前記ノ臨床的症狀並ニ既知動物毒ノ性質ヨリ推理スルニ恐ラクハ其ノ微量ニ於テ迷走神經ヲ鼓舞シ、後之ヲ麻痺スル性ヲ有スルモノニ非ラザルカ。本論ニ就キテハ動物實驗ノ條下ニ之ヲ說ル。

第 3 章 中毒死體ノ病理解剖學的所見

3 例（其ノ一、朱壹英當 3 年、死後 23 時間、其ノ二、朱氏蛮稅當 9 年、死後16時間、其ノ三、許氏緞當 11 年死後 12 時間）ニ就テ之ガ總括的所見ヲ記ルニ左ノ如シ。

胃、表面ハ一般ニ小彎部ニ於テ怒張セル血管多數ニシテ而モ其ノ血管ニ沿ヒ周圍ニ溢血斑散在セリ。一般ニ胃粘膜面粗糙ニシテ且ツ糜爛セルガ潰瘍ヲ認メタルモノ 1 例モナク溢血斑及胃內容物ニ血塊ヲ認メ、又膽汁ヲ滿タセルモノアリ。胃粘膜面ニ配布セル血管ハ著シク怒張セリ。

腸、本臟器ニ於テ病的變化最モ著明ニシテ、特ニ小腸ニ於テ至ル所配布血管ノ怒張、充血斑ヲ散在性ニ認ムルノ他各例トモ溢血斑スラ存在セル箇所ヲ發見ス。其ノ形狀及大サハ或ハ線狀ニ或ハ圓型ニ或ハ橢圓型ニ。大ナルモノハ直徑 2cm ニ及ブモノアリ、小ナルモノハ帽針頭大ニ至ル迄デ千差萬別ナリ。特ニ小腸ニ於テハ上部ニ多ク認メラルヽモ盲部ニ於テモ認メラル。尙十二指腸部ニハ膽汁ヲ以テ充滿シ腸壁切開ヲ行フヤ、多量ノ膽汁流出セリ。（寫眞參照）

心臟、表面ハ冠狀動脈稍々怒張ス其他所見少ク、內面ハ尙少量ノ流動性血液ヲ保有ス。水洗後心內膜面ヲ診ルニ著ルシキ所見ヲ認ザルモ、心筋及心膜面ニ充血斑ヲ認ムモ割面ニ著變ナシ。

肝、表面及內面共ニ地圖狀或ハ樹枝狀ノ充血斑ヲ認ム、割面ハ溢血斑ト思ハル、箇所アリタ。

腎、一般ニハ腫脹ヲ認メザルモノ多シ。表面、割面、內面共ニ異常ヲ認メズ。

第 4 章 海龜肉內ニ他ノ毒物ノ混在アリヤ

何等カ怨恨關係ニヨリ他殺ノ目的ヲ以テ他ノ毒物ヲ混在セシヤ否ヤニ就イテハ、先ヅ實情調査ヲ論理的ニ行フニ午前 1 時頃地曳網ニカヽリシ該海龜ヲ許、朱兩名共同ニテ捕獲シ得タルガ兩名ニ於テ等分シ（目前ニ於テ）許、朱ハ其ノ家族ニ食用セシメタル事確實ニシテ、特ニ許大炮ハ自身之ヲ食用セリ。朱某ハ自ラ捕ヘタル大ナル動物ハ之ヲ食セザル習慣ヲ持シテ食セザリシモ、然カモ自己ノ妻、娘其ノ他家族全部ニ之ヲ食用セシメタル外、近在ノ者特ニ親シキ者ニ之ヲ分讓セリ。其ノ他怨恨關係ナキ事實並ニ今日迄同樣或種無毒ノ龜ノ食用ニ就イテモ、略今回同樣ニ行セシ事數度ニ及ビ、且ツ彼等ノ自宅及附近ニ斯ル毒物ノ存在ナシ。仍テ當時ノ實情ハ推理的ニ斯ル毒物ノ混在ヲ否定シ得ルモノナリ。

第 5 章 海龜肉ノ罕ナル腐敗ニヨル中毒ニ起因スルヤ

該龜ヲ捕獲（4 月 26 日午前 1 時）シ屠殺（午前 9 時）後食用ニ供セル時間ハ前後ヲ通ジ、僅ニ 3 時間ニシテ、然カモ當時北臺灣ノ 4 月 26 日ナルヲ以テ氣溫低ク推定溫度 18 度內外ナリ。且ツ余等 4 月 27 日午後 7 時頃龜肉料理ノ殘品ニ付キ檢査スルニ未ダ腐敗シ居ラザリキ。

第 6 章　細菌學的感染ノ有無ニ就イテ

ハ食用ニ供セル海鼠肉ノ尚殘存セルモノ並ニ重症者ノ血液及該中毒死體ノ胃、腸、腎ノ內容物ヲ檢體トシ、先ヅ遠藤氏培養基並ニ普通寒天培地ニ移シ、之ヲ孵箱器內ニ(攝氏 37 度)納メルコト 24 時ニシテ生ジタル「コロニー」ヲ見ルニ、Salmonella 族ト思ハルヽモノ認メザリシモ、確實ニ大腸菌ト思惟セラルヽ「コロニー」ヲ除キ、全「コロニー」ニ就キ綿密ニ既知血清及重症者朱氏蕊 18 歳、趙氏足 20 歳ナル者(何レモ女性)ノ血清トノ間ニ就キ凝集反應ヲ試ミシニ、悉ク陰性ナリシガ、更ニ中毒死體ノ腸內容物タル檢體ヨリ生ゼル「コロニー」ニハ假性凝集(腸チフス)ヲ呈セルモノアリタルヲ以テ、更ニ嚴重ニ型ノ如ク鑑別培養ニ附シタルニ、之ヲ否定シ得タリ(第 2 表參照)。

第 2 表 ノ 一

氏名	朱玉蕊	趙氏足	朱氏雲祝		朱登英		許氏綾				備考
檢査物	血液	血液	胃內容物	腸內容物	胃內容物	腸內容物	胃內容物	腸內容物	腎內容物	料理セル海鼠肉	(−)ハ「コロニー」ノ發生ナキモノ
培養基ノ種類 遠藤氏寒天	−	−	+	+	+	+	+	+	+	+	(+)ハ「コロニー」ノ發生ヲ示ス
血液寒天	−	−	+	+	+	+	+	+	+	+	ブイヨン及肉汁ハ遠藤、普通寒天、血液寒天ノ各種ニ移植檢査セリ
普通寒天	−	−	+	+	+	+	+	+	+	+	
ブイヨン	−	−	+	+	+	+	+	+	+	+	
牛肉汁	−	−	+	+	+	+	+	+	+	+	

第 2 表 ノ 二

氏名	菌種	血清																
		朱氏蕊	趙氏足	チフス	パラA	パラB	パラC	パラK	赤痢混合	ユニポート	ロンドン	シュタット	馬流感菌	プレスラウ	スタンレー	ゲルトネル合	豚コレラ合	鼠チフス
朱氏雲祝	胃檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	腸檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
朱登英	胃檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	腸檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
許氏綾	胃檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	腸檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	腎檢出菌	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
	料理セル海鼠肉	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−

備考　試驗管內 50 倍ヨリ 800 倍迄稀釋反應ヲ行ヘリ

第7章 化學的檢索

1) 中毒ノ原因ナリト認メラル、食物。

午前時ニ屠殺シ、許、朱、兩家ニテ分チ肉ト内臓トヲ一緒ニ少量ノ落花生油ト共ニ熱シ鹽及醤油ヲ以テ調味セルモノナリ。

2) 殘食物ノ形態。

多量ノ黄色脂肪様物質ノ下ニ黒褐色ノ少量ノ液ヲ有スレ共他ハ鶏卵大ニ切リタル肉片ニシテ味覺ヲ刺戟スル臭ヒヲ有シ腐敗臭ナク弱酸性ヲ呈ス。

3) 食物殘品ニ對スル毒物檢索。

A. 本品ハ微ニ酸性ヲ呈スルヲ以テ「アルカリ」ヲ以テ中和シタル後酒石酸々性トナシ水ヲ加ヘテ重量煎上ニ致シ後濾過シタル酸性ノ液ニ就キ、「スタース、オット法ニヨリ次ノ操作ヲ遂グ。

第一　酒石酸々性液ニ「エーテル」ヲ加ヘ浸出シ若クハ抽出ス。

第二「エーテル」ヲ以テ浸出シ盡シタル酸性ノ水液（第一）ニ「ナトロン溶液ノ過剰ヲ加ヘ再ビ「エーテル」ヲ以テ浸出シ抽出ス。

第三「エーテル」ヲ以テ浸出シ盡シタル「アルカリ性ノ（第二液）ニ「クロールアンモン」ヲ加ヘテ後、アミールアルコール」ヲ以テ浸出ス。

第四　第三ノ殘液ニ炭酸瓦斯ヲ通シ砂浴ニ乾固シ其ノ殘渣ヲ「ソックスレット浸出裝置ニ納メ、「アルコール及クロ、ホルム兩液ヲ以テ抽出ス。

以上四種ノ抽出シタルモノヲ夫々通法ニヨリ精製シタル液、順ヲ追ヒ精密ニ夫々常法ニ從ヒ檢索セルニ、何レモ反應陰性ナリキ。

B. 可檢物 200 g ヲ同量ノ稀酒精ヲ以テ 24 時間抽出セル酸性ノ液ヲ重量煎上ニ蒸發シ數回通法ニヨリ、精製セルモノヲ 500.00 cc ノ水ニ溶解シタル液ニ就キ次ノ反應ヲ試ム。

I) 本液ノ 10.0 cc ヲ「フエリング溶液中ニ入レ熱スルニ之ヲ還元シテ赤褐色沈渣ヲ生ズ。

II. 本液 20.0 cc ニ「アンモニア性硝酸銀溶液ヲ加ヘ熱スルニ黒褐色ヲ呈ス

C. 更ニ可檢物ヲ常法ニ從ヒ有機物質ヲ破壞シタル液ニ無機毒ノ檢索ヲ試ムルモ何レモ之ヲ認メズ。

以上化學的操作ノ結果ニ徴スルニ、本食物中ニハ「アルカロイド及プトマイン並ニ無機的毒物ヲモ之ヲ證明セサルモ、酒精ニヨル抽出物中 B ノ試驗ニ對スル反應ハ一種ノ「トキシン」ノ存スルコトヲ認メ得タリ。

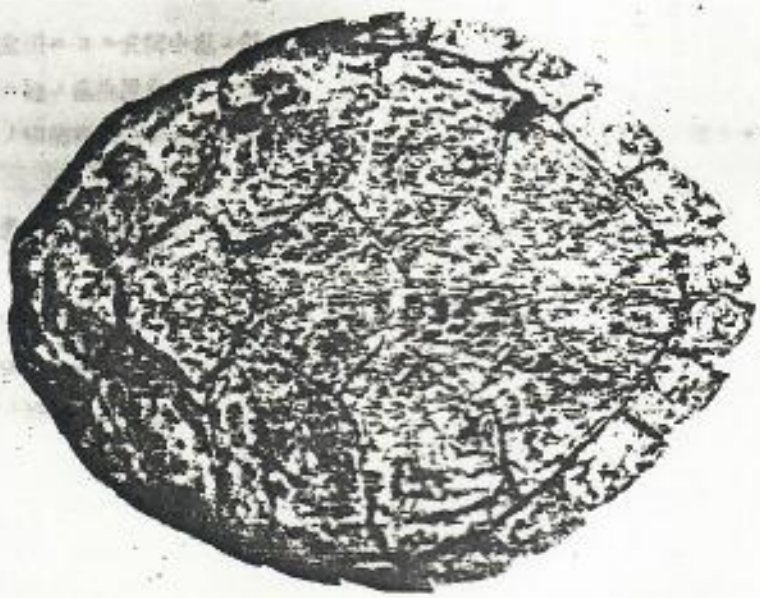
第8章 動物實驗

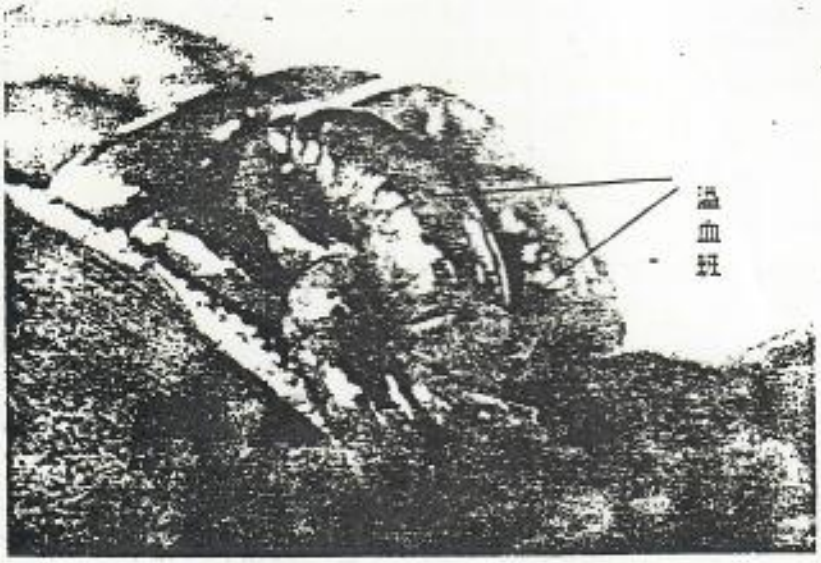
可檢物ヲ試驗液トシ、動物實驗ニ移セリ。使用セル實驗動物ハ海豚體重一頭ハ 1.5 瓩、他ノ一頭ハ 1.3 瓩ニテ都合二頭及ビ一頭ノ「マウス並ニ蛙六匹ニ就キ實驗セリ。可檢毒試驗液ハ浸出毒微量（1/100）ナルヲ以テ（中毒者自身及家族ノモノ、言ヲ以テ推理スルニ恐ラクハ魚肉ヨリ寄

ロ内臓器中ニ多ク含有セラレ居ルモノナランカ）可及的可檢毒試驗ヲ多量ニ使用セザルベカラズ。從ツテ小動物マウス等ヲ可トスルモ前章ニ於テ既述セルガ如ク、本中毒例ノ臨床的所見ヨリ考察スル時ハ、特ニ本毒ハ其ノ血行系ニ作用スルガ如ク、思考セラルヽニ就キ、蛙ノ心臓ニ就イテ本可檢毒試驗液ノ作用ヲ觀察セリ。而シテ可檢毒液ハ料理ヲ施セル該海鼠肉ヨリ製セルB液ヲ以テセリ。該液ニ20.0ccヲ海猿ノ皮下ニ注入シ、對照トシテ他ニ1.45瓩ノ海猿及1.3瓩ノ海猿ニ生理的食鹽水ヲ同様20.0ccヲ皮下ニ注入セリ。注入後可檢毒液ヲ注入セル一號及2號ハ共ニ殆ンド時ヲ同ジウシ、約15分ニ於テ數回ノ嘔吐ヲ來セルモ、尚斃死ニ至ラズ。然ルニ對照海猿3號及4號ニ於テ何等カヽル症狀ヲ見ザリキ。「マウス」ニ於テハ可檢液2.0ccヲ注入セルガ見ル可キ變化ナシ。仍ツテ翌日更ニ同液10.0ccヲ1時ニ皮下ニ注射シタルニ30分後ヨリ不快症狀アルガ如ク、前後左右ニ運動スルノミナラズ、口唇ヲ前手ヲ以テ恰モ拭フガ如キ運動ヲナセルガ約20分ニシテ數回ノ嘔吐ヲ來セリ。(但シ内容物ニ乏シク粘液様水様液及胃液ノ他少量ノ顆粒狀食物殘渣ヲ吐出セリ。)其ノ後著シキ變化ナク30時間後ニ於テ斃死セリ。剖檢セルニ海猿ニ於テハ2頭トモ内臓ニ變化ヲ認メズ。「マウス」ニ於テハ脾、心臓、小腸ニ充血斑ヲ認メタルモ著明ナラズ。然カモ對照マウス無キヲ以テ變化ノ程度ヲ云々シ難ク、且ツ組織標本ヲ作成セザリシヲ以テ詳細不明ナリシハ遺憾トスルトコロナリ。又蛙6匹ニ夫々1號ヨリ6號ノ番號ヲ附シ1號、2號、3號、4號ハ之ヲ抽出セル毒液（B液）ヲ注射シ、5號、6號ハ之ヲ對照トシテ使用セリ。而シテ該蛙ヲ1號及2號ハ大腦ヲ切除シ、他ノ3號、4號ハ「クロロホルム麻酸ヲ行ヒテ腹壁ヲ切開シ心臓ヲ體ニ附着ノマヽ、見易キ様露出シ實驗セルガ、始メ可檢毒液ヲ0.5瓦筋肉内ニ注入後直チニ心臓運動稍々緩徐トナリ、更ニ心臓附近ニ倍量ヲ注入スル時ハ、更ニ緩徐トナルガ如ク更ニ同量ヲ心囊内ニ注入スル時ハ、逆ニ心運動ハ速トナリ脈搏數ハ注射前ニ比シ多數ヲ算スルガ如シ。然レドモ之ヲ心筋内ニ前記實驗量ノ10倍以上ヲ注射スル時ハ、又緩徐トナル。更ニ可檢毒液10.0或ハ15.0ヲ與フル時ハ遂ニ心臓ハ其ノ運動ヲ1時停止ス。而シテ時間ヲ經ルニ從ヒ再ビ搏動ヲ始ルモノアリ。此ノ場合漸次搏動强クナリテ、遂ニ逆ニ搏動ハ注射前ヨリモ速ニナルニ至ル。然レドモ甚ダシキ多數ヲ與フル時ハ、遂ニ心臓ヲ麻痺スルガ如シ。此ノ時ハ内臓器特ニ肝表面及ビ腸壁ニ出血斑ヲ生ゼリ。

以上ノ事實ヨリ考察スルニ該トキシン」ハ矢張リ先ノ推定通リ、其ノ少量ニ於テ迷走神經ヲ刺戟ス。從ツテ心動緩徐トナリ更ニ其ノ毒量多量ニシテ其ノ刺戟强キトキハ迷走神經ヲ麻痺シ、反ツテ心動ヲ速カナラシムガ如シ。然レドモ實驗ノ示スガ如ク超多量ヲ注射スル時ハ、又再ビ心動停止スルニ至ルハ恐ラク心筋自體ノ麻痺ヲ招來スルモノヽ如シ。而シテ實驗ニ使用セル1號、2號、3號、4號ハ共ニ其ノ大腦切除ヲ行ヘタルト又「クロロホルム麻酔ニヨリタルモノモ其ノ毒物ノ實驗的成績ハ完全ニ相一致ス。

衣笠.鈴木論文附圖





總　括

1) 或種海鼈食用ニヨル食餌中毒ニ就キ科學的原因探索ヲ行ヘリ。

2) 該海鼈内ニ他ノ毒物混在ノ有無ヲ檢シ、其ノ然ラザルヲ確ノ得タリ。

3) 該海鼈内ノ細菌學的感染ノ有無、特ニ Salmonella 類ヲ求メタルモ、其ノ然ラザルヲ確認セリ。

4) 該内中毒患者ノ臨床的症狀ヲ或程度ニ明瞭ニシ得タル結果ハ其ノ原因ノ中毒ニヨルヲ思ハシメタリ。

5) 中毒死體ノ剖檢的所見ヲ求メ其ノ中毒ニヨルモノトノ信念ヲ高メタリ。

6) 化學的ニ該鼈内内ヨリ詳細不明ナルモ「トキシン」樣毒物ヲ檢出シ得タリ。

7) 動物實驗ノ結果ハ前項化學的試驗成績ニ一致セルガ如キ作用ヲ認メタリ。

結　論

以上ノ檢索ノ結果ヲ綜合スルニ本中毒原因ハ海鼈體内ニ存在セシ毒物ニ基因スルモノニシテ其ノ毒物ハ恐ラク一種ノ「トキシン」ナル可シト判定セラル。仍テ或種海鼈中ニ「トキシン」樣物質ヲ保有シ居ルカ又ハ或ル期間之ヲ保有スルモノナル可シト考察シ得ラルヽ處ナリ。尙本調査研究ハ何分事件發生地ガ遠隔地ナル事、急ヲ要スルヲ以テ尙研究ニ充分ナラズシテ、誠ニ粗下粗率ノ感アルヲ遺憾トスル處ナルガ、他日更ニ之ガ詳細ナル調査研究ヲナサン。

文　献

1) 下條久馬一，今日ノ「バクチフス論、日本傳染病學會雜誌 第11卷 第10號及第12號（昭和11年7月—同13年9月） 2) ロベルトヲツトー，衛生檢査法 3) 弁波敬三，數理化學 4) 目野五七郎，和漢薬物學 5) 森島庫太，藥物學 6) 原 三郎，實驗藥理學本草綱目 7) 竹内松次郎，細菌學及免疫學 8) 食餌中毒トプロトイス菌ノ相互的關係ニ就イテ日本傳染病學會雜誌第10卷 田村主五郎 9) 杉田慶介，食餌中毒例ヨリ檢出セル「プロテウス菌類以一新菌種ニ關スル研究 藥學雜誌 第37卷 第2號 昭和13年2月

Über Untersuchungen der Ursache der massenhaften Vergiftung nach dem Genusz von Fleisch einer an der Küste von Koryo in der Präfektur Sintiku gefangenen Seeschildkröte.

Von

Masaru Kinugasa und Wasaburo Suzuki.

Aus der Hygienischen Unterabteilung, die Abteilung für Polizeiangelegenheiten.

Es trug sich in der letzten Zeit zu, dasz viele Vergiftungsfälle (57 Fälle, von denen 7 starben, 9 schwer und 41 leicht erkrankten) nach dem Genusz von Fleisch einer in der Nähe an der Küstengegend Koryo (Kreis Tikunan) in der Präfektur Sintiku gefangenen Seeschildkröte (Karette?) entstanden, und dasz wir bald nach dem Empfangen der Nachricht über dieses Ereignis nach dem Entstehungsorte der Vergiftungsfälle eilten, um von verschiedenen Seiten die Fälle zu untersuchen und folgende Ergebnisse zu erhalten:

1. Wir machten chemische, in solchen Fällen erforderliche Untersuchungen.

2. Die Untersuchung der Reste des gekochten, in Betracht kommenden Fleisches auf die anderen, etwaigen, absichtlich gemischten Gifte und das Verhör der damaligen Sachlage von den Patienten ergaben, dasz kein Gift darin gemischt war.

3. Wir untersuchten das Restfleisch der betreffenden Schildkröte, das Blut von damit vergifteten Patienten und den Inhalt von Magen, Darm und Nieren der Todesfälle auf etwaige Bazillen, besonders auf Salmonellagruppe, und konnten feststellen, dasz die Folge der Untersuchungen negativ ausfiel.

4. Wir konnten auch die klinischen Symptome der betreffenden Patienten bis zu einem bestimmten Grade klar stellen; sie deuteten uns an, dasz die Ätiologie der Erkrankung in Vergiftung zu suchen ist.

5. Obduktionsbefunde lieszen unser Glauben an Vergiftung immer fester machen.

6. Chemisch konnten wir eine Art von einem Toxin ähnlicher Giftsubstanz, deren nähere Eigenschaften noch nicht zu erklären sind, in dem Restfleisch herausfinden.

7. Die Ergebnisse des Tierversuches lieszen die Wirkung erkennen, welche mit der Folge der obengenannten chemischen Untersuchung übereinstimmte.

Schluszfolge:

Aus den obenerwähnten Ergebnissen läszt es sich schlieszen, dasz die Ursache der betreffenden Vergiftung auf das Gift im Fleisch der Seeschildkröte zurückzuführen ist, welches vielleicht als Art Toxin angenommen wird. Daher wird es auch vermutet, dasz das Fleisch einer Art Seeschildkröte entweder immer oder in einem gewissen Stadium einen Toxin ähnlichen Stoff enthalten werde.

Unsere Untersuchungen sind leider nicht vollständig angestellt worden, weil die Sache in einem abgelegenen, im Verkehr nicht begünstigten Orte geschah, und der Bericht darüber Eile beanspruchte, um allgemeine Aufmerksamkeit auf diese Sache richten zu lassen. Wir werden künftig Gelegenheit haben, von neuem dieselbe Sache noch näher zu untersuchen.

Lis Sedlak-Weinstein,
11 Glenaplin Ave,
TARRAGINDI QLD 4121

Dr. George Balaz,
National Marine Fisheries Service,
P.O. Box 3830,
HONOLULU HAWAII 96812

3.6.87

Dear Dr. Balaz,

G'day from Australia, hope you still remember us. Do you still have any contact with Cris Frieberg, we'll probably see him when we go to Micronesia at the end of this year (if he is still Fisheries officer there). We're going to Fiji next month and on reading newspapers from there, hoping to get some background to the political situation we came across some articles on turtle poisoning you might be interested in. Is it ciguatera or something related? Would be interested in finding out more about it.

Also since I'm doing post-grad. work (here it comes!) in Cetacean parasitology, I was wondering who would be a good contact in Hi. I know Dr. Townsley has retired (I think the year I graduated), but I don't know who replaced him. In particular I'm interested in crustacean ectoparasites of the smaller odontocetes.

Aloha,

Steve + Lis
Weinstein

FORECAST: Cloudy. Details and map on Page 22.

FIJI
Sunday Times

APRIL 19, 1987 | 28 PAGES | 25¢ | No. 456

LATE NEWS

Boat found

SYDNEY — The crew of a RAAF Hercules yesterday sighted a boat which had been missing for more than a week off the coast of Papua New Guinea with 20 people on board.

Equipment including basic medical supplies and signalling gear was dropped to the 16m mission ferry.

Earlier report Page 5.

Doctors baffled by turtle poisoning

MYSTERY OF TWO DEATHS

By SATENDRA SHANDIL

Doctors at Labasa Hospital are baffled by the deaths of two Udu villagers who died after eating turtle meat at their village on Saturday.

Eight others have been admitted to the hospital.

A woman, Pulu Rasole, 19, died, while being taken to the Tawake nursing station on the southern coast of Udu Point while a man, Navitalai Setoki, 33, died later on Wednesday after arriving at Labasa Hospital.

The Medical Superintendent at the hospital, Dr Mary Schramm, said poisoning could only have occurred if the turtle meat was cooked and eaten sometime later without refrigeration to preserve the food.

Nakudamu villager Rapoama Nabiri, 17, told the Sunday Times that he was among a group of villagers who caught three turtles near their village on Saturday afternoon.

He said two of the turtles were "familiar ones" while a third one was an "irregular one".

Nabiri said about 12 of the villagers ate the turtles for dinner at Nakudamu Village the same night.

His father, Savenaia Senibase, 60, was also admitted to Labasa Hospital with six others.

They are Etuate Lokasi, Pita Kinadavura Qaqa, twin sisters Ruci Manisaqanitavua, 15, and Milinkere Baleisavusavu, 15, and Mary Dunn, 14.

The three girls were admitted to the intensive care unit of the hospital but were reported to be in stable condition.

Almost all of the patients were put on intravenous drips.

Medical staff at the hospital said these were the first cases of turtle poisoning they had come across.

They said deaths from eating turtle meat were unknown.

Senior Sister Wati Zoing said medical staff were baffled by the cases.

One of the patients, Mary Dunn, 14, told the Sunday Times she had only taken a "small piece" of the meat.

She said she suffered from pains and vomiting.

Two elderly villagers from Udu Point who came to visit the patients, Mrs Meresaini Kelevi, 56, and Mr Solomoni Begidrau, 54, said this was the first time they had seen deaths caused by eating turtles.

Mrs Kelevi said they had heard of deaths being caused by eating turtles in the past but this was from stories passed down by ancestors.

According to the stories, some villagers ate turtle meat after a giant turtle surfaced three times during a period when sea food was banned due to the death of a chief.

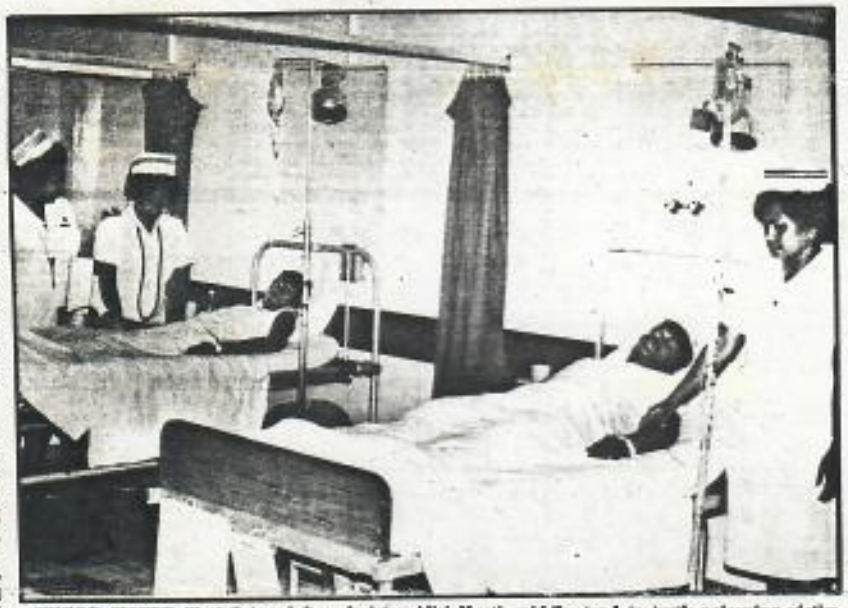
The story is also recounted in a old song, according to the villagers.

Dr Schramm said she believed that the poisoning could have occurred because of the cooked food not being preserved before being eaten.

Doctors at Labasa Hospital were still waiting for a detailed report yesterday.

The Divisional Medical Officer Northern, Dr Asinate Beladuadua, was still out at Udu Point yesterday with a team of health personnel.

Dr Beladuadua was expected back in Labasa late yesterday.



SENIOR SISTER Wati Zoing, left and sister Alisi Vuati, middle, tend to turtle-poisoning victim, Rapoama Nabiri, 17, while Sister Vasiti Batireregn tends to victim Etuate Lasei. The two were among eight people from Nakudamu Village in Udu Point at Labasa Hospital yesterday after eating turtle meat last Saturday night.

Sikh faces 'war' charge

NEW DELHI — A powerful Sikh leader is to stand trial on charges of waging war against the Indian state in a case filed after the 1984 army assault on the Golden Temple, the Press Trust of India (PTI) said yesterday.

Gurcharan Singh Tohra, president of the influential Shiromani Gurdwara Prabandhak Committee, was named as the principal accused in the case to be heard by a special court in Jodhpur in northern Rajasthan State, PTI said.

It said Tohra, who was arrested in December, was to face trial along with 366 suspected Sikh militants rounded up during the army assault on the temple, the holiest of Sikh shrines, in the Punjab city of Amritsar.

No date for the trial was announced.

The June 1984 operation at the heavily-fortified temple to flush out Sikh militants fighting for a separate homeland officially left 650 people dead. But witnesses have said that nearly 1000 people died.

Prosecutors said that Tohra and the other accused had waged war against the Indian Government by conspiring among themselves and confronting military and paramilitary forces between June 5 and 10 of 1984.

The migrants' plight: Page 7

TWO DEAD AFTER FEAST OF TURTLE

Two people are dead and eight others have been admitted to Labasa Hospital after eating turtle meat last Saturday.

All the victims are from Nakunadamu Village near Udu Point on the Northern tip of Vanua Levu.

Doctors at Labasa Hospital are puzzled about what caused the two deaths.

"It is very puzzling," the medical superintendent at the hospital, Dr Mary Schramm, told The Fiji Times.

Doctors say there are no known cases of humana dying after eating turtle meat.

Reports reaching Labasa from Udu Point say that the villagers fell sick late at night last Saturday.

One woman, who has not yet been identified, died while being rushed to the Tawake Health Centre on Sunday.

A man from the same village was admitted to Labasa Hospital but died on Monday afternoon.

Eight other people from the same village were admitted to Labasa Hospital on Wednesday.

Dr Schramm said they were in stable condition.

"The village is so far away that the villagers don't come to Labasa for treatment unless they are very ill," Dr Schramm said.

She said no one knew how many more people were suffering in a similar manner in the village of Nakunadamu.

The symptoms were not that of food poisoning, she said.

"The man who died was vomiting and had diarrhoea, then he became drowsy, fell unconscious and died," Dr Schramm said.

She said the other villagers were also suffering from vomiting, diarrhoea and spells of drowsiness.

She said a medical team, led by subdivisional medical officer Dr Asinate Boloduadua, was on its way to Nakunadamu Village yesterday to try to find out what really happened there last Saturday.

"We don't know whether they had a big *magiti* with a lot of other food like fish apart from turtle meat," Dr Schramm said.

Some people said they had one turtle while others reported that there were two turtles.

She said once the medical team reported back, the doctors might get a clue as to what caused the illness.

"Right now, we don't know," she said.

FLOTSAM AND JETSAM

By Beachcomber

SERVICE at service stations is not what it used to be

A Beachcomber was kept waiting for 10 minutes at a Flagstaff Service Station yesterday afternoon before a grumpy-looking attendant finally turned out to put petrol in his car.

When the Beachcomber asked him to please check the oil, the grumpy attendant replied: "Do it yourself."

Maybe the "service station" should be called a "do it yourself station".

* * *

IT MAY NOT be much consolation to the kid having his head banged against the playground wall but the school bully according to new research in Britain, is condemned to a life in which he victimises himself with a pattern of self-defeating belligerence and failure.

Feared by other children, disliked by teachers and often ignored by parents. He tends to drop out of school and is three times more likely to run foul of the law.

'Bullies see the world with a paranoid's eye,' says one psychologist. 'They feel justified in retaliating for what are actually imaginary harms.'

FACES OF DESPAIR

THESE faces have despair written on them. These are some of the men, women and children who have been stranded at Nadi for the past eight days. They say they are Sri Lankan refugees, fleeing their homes in Jaffna, in Northern Sri Lanka, because of the civil strife there. These pictures were taken at a guest house where the 16 have been given temporary shelter while airlines and immigration officials of Fiji and other countries decide what to do with them.

WOMEN shy away from the camera of The Fiji Times photographer. This picture of Sri Lankan nationals was taken in the living room of a guest house near the Nadi Airport on Thursday evening.

A 29-YEAR-OLD single woman who has ambitions of joining her two brothers in Montreal, Canada. She left her farmer parents behind in Jaffna, Sri Lanka when she caught a flight to Madras, India almost a year ago.

Pictures by SUNIL CHAND SUNNY.

AN 18-YEAR-OLD girl, a student, claims that her 25-year-old brother

Man faces murder charge

A 26-year-old Ba fisherman will appear in Ba Court next week

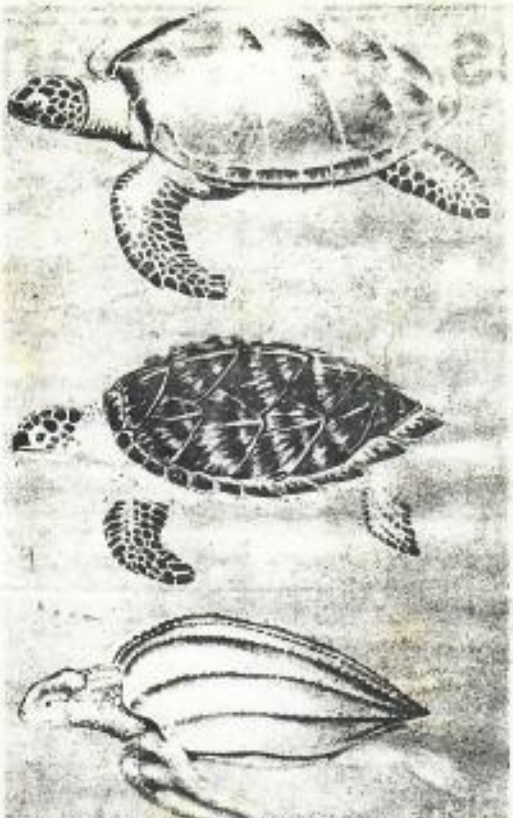
Coconut wireless

High hopes with Labour

IT could be a genuine hope and plea . . . a man who appeared in Suva Court recently on a charge of larceny made an unusual mitigation.

The man, in his early 20s, pleaded guilty to a charge of stealing a wrist watch and cash from a shop in Pier Street.

In his mitigation, the man said he was unemployed but now that the " Labour Government was in power " he had hopes getting employed.



TOP: Green Sea Turtle, *Chelonia mydas* (Linnaeus). Centre: Hawksbill Turtle, *Eretmochelys imbricata* (Linnaeus). Bottom: Leatherback Turtle, *Dermochelys coriacea* (Linnaeus).

How poisonous are turtles?

POISONING from marine turtles is one of the lesser known types of intoxications produced by marine organisms.

The cases that have been reported are sufficiently severe to be impressive.

As in the case of fishes, most of these species are commonly eaten with impunity.

For some unknown reason, certain species of marine turtles in the vicinity of the Philippine Islands, Ceylon, and Indonesia, under certain circumstances, may become extremely poisonous to eat.

Species of Marine Turtles reported as poisonous to eat:

GREEN SEA TURTLE, *Chelonia-mydas* (Linnaeus). Inhabits all tropical and subtropical seas.

HAWKSBILL TURTLE, *Eretmochelys imbricata* (Linnaeus). Inhabits all tropical and subtropical seas.

LEATHERBACK TURTLE, *Dermochelys coriacea* (Linnaeus). Largely circumtropical, but occasionally taken in temperate waters.

MEDICAL ASPECTS. Symptoms generally develop within a few hours to several days after ingestion of the flesh.

The initial symptoms are usually nausea, vomiting, diarrhoea, severe upper abdominal pain, dizziness, dry burning sensation of the lips, tongue, lining of the mouth and throat.

Swallowing becomes very difficult, and excessive salivation is pronounced.

The disturbances of the mouth may take several days to develop, but become progressively severe as time goes on.

The tongue develops a white coating, and the breath becomes very foul.

Later, the tongue may become covered with multiple pinhead-sized, reddened papules, which may later break down into ulcers.

If the victim has been severely poisoned, he tends to become very sleepy, and is difficult to keep awake.

If this symptom develops, it is usually a bad sign, and death soon follows.

Death is believed to be due to liver and kidney damage. About 44 percent of the victims poisoned by marine turtles die.

TREATMENT. There is no specific treatment. Some of the recommendation presented in fish poisoning are pertinent here.

PREVENTION. Marine turtles in the tropical Indo-Pacific region should be eaten with caution.

If in doubt, check with local native groups and find out if they are safe to eat in that locality. Turtle liver is especially dangerous to eat.

Turtle poisoning is complicated but not uncommon

TURTLE poisoning is " complicated " but not uncommon, a Suva bussinessman, Peter Fray, said yesterday.

He said it was likely that the turtle meat eaten by Udu villagers, which caused the death of two of them, was of the species not commonly found in Fiji — the leatherback turtles.

Speices commonly found here are Hawksbill turtle (eretmochelys imbricata) known as taku in Fijian, and Green turtle (chelonia mydas), known here as vonudina, ikadina.

A 19-year-old woman, Pulu Rasele, and 33-year-old Navitalai Setoki, died after eating turtle meat.

One of the eight admitted in Labasa hospital, Savenaia Senibase, 60, is still in a serious condition, a hospital doctor said yesterday. Seven others admitted after the incident, are recovering.

Mr Fray said he has seen a lot of turtle poisoning in Indonesia and Malaysia, mostly in the Christain community. He said the Muslims do not eat turtle meat.

Turtles eat seagrass, sea sponges and shell fish which are at times poisonous, he said. Parts of the turtle particularly poisonous are the skin, liver, bones and flesh near the stomach.

Mr Fray, originally from Bavaria, Germany, worked in Malaysia for 13 years on a project of breeding leatherback turtles which were almost extinct.

He said they managed to build a good stock and leatherbacks are known to be far travellers — some turtles taged (for identity) in Malaysia were later found in Tahiti.

U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
NATIONAL MARINE FISHERIES SERVICE
Southwest Fisheries Center Honolulu Laboratory
2570 Dole St. • Honolulu, Hawaii 96822-2396

May 1, 1987 F/SWC2

Dr. Gopal Krishnan
Labasa Hospital
Fiji

Dear Dr. Krishnan:

I recently read an article in the Fiji Times (copy attached) about poisoning of people at Nukudamu Village after eating a turtle or turtles. Poisonings possibly of the same nature are known to have occurred at other locations, and usually involve the hawksbill turtle, *Eretmochelys imbricata*. A short summary on this subject from an FAO publication has been enclosed for your information

As part of my research with the National Marine Fisheries Service, I have been compiling information on cases of poisoning by sea turtles throughout the Pacific. In this regard it would be greatly appreciated if you would answer the following questions to the best of your knowledge.

1. What kind of sea turtle was eaten? Was more than one turtle served at the feast?

2. Has sea turtle poisoning ever occurred before at Nukudamu Village, or anywhere else in Fiji?

3. How many people total ate the turtle? I understand that 11 sick people have been admitted to the hospital and 4 others have died. Were there people that ate portions of the turtle but did not become sick? Could the sickness or deaths be attributed to eating a particular part of the turtle, such as the viscera?

4. What were the approximate ages of the four people that died? Were they male or female?

5. What were the symptoms of the poisoning?

6. How soon after eating the turtle did the people become sick? How soon did the deaths occur?

7. How was the turtle cooked?

8. Approximately how many turtles are captured and eaten each year by the people of Nukudamu Village?

9. Do sea turtles lay their eggs near Nukudamu Village?

Thank you for any help that you can give to this inquiry. I have enclosed a color poster showing the different kinds of sea turtles. This may be of use to you in your work. I look forward to hearing from you at your earliest convenience.

Sincerely,

George H. Balazs
Zoologist

Enclosure

FIJI TIMES — APRIL 23, 1987

Turtle meal puts another three in hospital

THREE other people from Nukudamu village, near Udu Point, were admitted to the Labasa Hospital from suspected food poisoning after eating turtle meat yesterday.

Admitted are Tomasi Tuinavadra, 24; Merewalesi Sese, 33; Uate Saviri, 22.

They ate the meat at a feast two weeks ago.

Last night the hospital's consultant physician Dr Gopal Krishnan said their condition was satisfactory.

Meanwhile seven others who were admitted to the hospital this week from the same incident were discharged on Tuesday.

Four people have died from the incident. They are 64-year-old Osea Naceva, Sevanaia Senibusi, Navitalai Setoki and Tuinavadra's wife Rinieta Pulu.

Reports from Nukudamu village said more people who ate the turtle meat have begun to show signs of the sickness.

A medical team which returned from Nukudamu on Monday were to make another visit yesterday but they were held back because there was no vessel to take them across.

Sub-divisional medical officer northern, Dr Asinate Boladuadua, who was to head the team said they will probably make a visit today.

THE MINISTRY OF HEALTH
GOVERNMENT BUILDINGS
SUVA, FIJI
LABASA HOSPITAL
FIJI

TEL. No. 81444

REF. NO.

DATE:

9th June, 1987

Mr George H. Balazs,
Southwest Fisheries Center,
Honolulu Laboratory,
2570 Dole Street,
Honolulu, Hawai 96822-2396.

Dear Mr Balazs,

Thank you for your letter dated 1st May, 1987 and also for the referal reprints. Sorry for the delay in replying. As you required I am giving you below the details of the turtle poisoning happened in Fiji in April 1987.

Three turtles were speared off shore by a villager on 11.4.87 at Nukudamu in Fiji. Two of them were Green Sea Turtles and one Hawkesbill type. One of the Green turtles was eaten by a family in another village with no incidence of poisoning. The other Green Turtle was found decomposed after cooking and was discarded.

All the poisoning happened in Nukudamu village was after eating the Hawksbill turtle. 43 peple ate the turtle meat; from children of 5 years to adults of 78 years. 24 of them had symptoms of poisoning. The shoulder portion of the turtle was eaten by a family of 11, of which 3 of them developed only mild symptoms. The visceral and the rest of the turtle was eaten by 32 people of which 4 died, 4 severely ill, 5 moderately ill and another 8 mildly ill; none under the age of 14 was ill.

The 4 fatal cases were of 19, 33, 70 and 78 years old. The 19 years old was a female and the other males. The deaths occurred on the days 3,5 and 10.

The intervals between the time of eating the meat and the beginning of symptoms were from four hours to 10 days.

The main symptoms were nausea, vomiting, abdominal pain, muscular and joint pains, weakness, drowsiness with tendency to sleep and sore throat and mouth.

The turtle was cooked by boiling with minimum water added.

An average of about 200 - 300 turtles are caught and eaten per year by the villagers of Nukudamu and around. Sea turtles were not found to have laid eggs in that area this year.

The turtle poisoning was never reported in Fiji before; but according to the elderly villagers of Nukudamu, 56 people died in a neighbouring village after turtle poisoning in 1905.

I may be able to send you more particulars later.

Yours sincerely

(Dr K.E.G. Krishnan)
Consultant Physician, Labasa Hospital

Letters to the Editor–

THE MALARIA ERADICATION PROGRAMME IN THE BRITISH SOLOMON ISLANDS

Sir–

Further to my review 'covering the years 1970-72,' information is now available on progress during the years 1973-74. This is shown in Table 1.

During the period 1973-74 cyclical spraying operations with DDT continued regularly every 6 months throughout the malarious area. In North Choiseul, North Guadalcanal and Nggela this was carried out every 4 months due to the high vector densities. Surveillance also continued in all areas, though some difficulty was experienced in getting this fully established in bush Malaita and on the Weather Coast of Makira (San Cristobal). In May 1974 most of the New Georgia Group of Western District formally entered into the Consolidation Phase (i.e. withdrawal of DDT spraying).

In all four Districts good progress was made. The most notable progress was made in Western and Eastern Districts in both of which *P. falciparum* infections had virtually disappeared by the end of 1974. Western District continued to have a small problem with *P. vivax* infections in the Gilbertese and a rather large problem with the importation of malaria (including *P. falciparum*) from other parts of the Solomons and from Papua New Guinea (3 proven and 2 probable cases in 1974). Eastern District, after some resumption of transmission in Makira and Ndendi (Santa Cruz) during 1973 and early 1974, went on to dramatically reduce the parasite load in the community. This was aided partly by a mass drug administration trial which was carried out on the north-east coast of Makira.

Central District experienced a (not entirely unexpected) set back during mid 1973 with widespread resumption of transmission in Guadalcanal including Central Bush and southern (weather) coast area. Recovery from this devastating epidemic started in late 1973 and has progressed steadily ever since. The north coast of Guadalcanal remains the major problem area in the Eradication Programme with high vector densities, rapid commercial/agricultural development and a highly mobile population. In the remaining islands of Central District such excellent progress has been made that Ysabel and Russels may well be able to go into Consolidation towards the end of 1975. (Rennell went into Consolidation in 1972). In the formerly holoendemic Nggela group where trans-

Table 1. British Solomon Islands Protectorate

District	Year	No. of slides examined by ACD/PCD	No. Positives by ACD/PCD	% Positive
Central:	1970	33961	4523	13.3
Population	1971	28838	1923	6.7
69039	1972	32437	1656	5.1
	1973	35830	4082	11.4
	1974	34827	2215	6.4
Western:	1970	12726	520	4.1
Population	1971	16655	422	2.5
37123	1972	22275	378	1.7
	1973	22165	258	1.2
	1974	24836	86	0.34
Malaita:	1970	[illegible]	1451	39.6
Population	1971	[illegible]	1070	15.3
57761	1972	18943	1016	5.5
	1973	23691	993	4.2
	1974	27843	414	1.5
Eastern:	1971	2053	928	36.7
Population	1972	5510	918	16.6
24633	1973	8787	1084	12.4
	1974	10516	340	3.2

Papua New Guinea Medical Journal 1975 18(2) pp: 125-126

mission has been difficult to interrupt since spraying started in 1970, there was an average of only four malaria cases per month during the latter half of 1974.

Malaita District has experienced minor outbreaks of transmission in scattered areas every year since spraying started in 1970. This was repeated during 1973 and more seriously in 1974. During mid 1974 East Kwaio, an operationally difficult area, showed signs of resolving, only to break out again in November and spread up and down the coast and also across the mountain barrier to west central Malaita. This outbreak of mainly *P. falciparum* malaria, which was resolving by April 1975, was an object lesson in the need for very speedy action once the parasite gets the upper hand in a semi immune population (as a result of spraying).

During 1973-74 further efforts were directed towards improving the standards of field operations, but delays in supplies and transport problems continued, as always, to threaten the success of the programme. Great efforts were made to improve on the supervision of treatment of all proven malaria cases to avoid the well known relapsing propensity of S.W. Pacific *P. vivax* strains. Remedial measures were intensified around all foci. Mass Drug Administration was extensively used with good results in the Gilbertese communities in Western District, Nggela and N.E. Makira but equivocal results in North Guadalcanal.

Objectives and plans for 1975 include the elimination of all *P. falciparum* Parasitaemia from the Solomons; a special effort in Malaita; an improvement in the detection of imported cases into Western District and an all out onslaught on the north coast of Guadalcanal. Additional supplementary measures will include the use of ultra low volume peridomestic space spraying with malathion and the use of Abate Larvicide. These will be directed at specific foci with the objective of reducing vector densities to a very low level during the low transmission season.

The prospects for the eventual eradication of malaria from the Solomon Islands remain good. It cannot be emphasized too strongly however, that the task is not an easy one and that it requires the wholehearted backing of all agencies in the Ministry of Health, full support by other agencies and last but by no means least, full acceptance and co-operation by the people.

I am grateful to His Excellency, the Governor of the British Solomon Islands, for permission to publish this Progress Report.

Dr. J. G. Avery,
Chief Medical Officer (Community Health), Ministry of Health and Welfare, HONIARA. British Solomon Islands.

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TURTLE MEAT AND CONE SHELL POISONING

Sir

Two unusual cases which presented at Kavieng hospital during 1974 illustrate an interesting and disturbing lack of local knowledge about dangers from marine animals.

1. Turtle Meat Poisoning

A meal of turtle was eaten in Panapai village, close to Kavieng, on the afternoon of Sunday 11th August. During the night several children were sick. At about 6.00 a.m. the next day a child of 6 years was brought to the hospital, after having become unconscious following vomiting. There was no response to pain, a flaccid paralysis and dilated pupils. Lumbar puncture and a malaria parasite slide were performed to exclude more common diagnoses, but in spite of supportive measures including intravenous fluids, hydrocortisone and phenergan, the child died quite suddenly at midday. This child was alleged to have eaten the unlaid eggs of the turtle.

During the night 2 more children died in the village. The next morning the teacher brought 18 other children who had eaten the turtle. Fifteen were symptom free; 3 had had attacks of vomiting, and were kept

under observation for 24 hours. In the absence of further symptoms, they were discharged.

Despite careful inquiries we were unable to ascertain the details of the catching and cooking of the turtle, but the shell was recovered and identified as the ERETMOCHELYS IMBRICATA or Hawksbill Turtle. It was not possible to discover whether the poisoning was due to staphylococcal or clostridial contamination, or a toxin in the meat itself.

Several persons associated with this incident recalled the poisonings at Namatanai in December 1965 which resulted in 5 deaths, and on New Hanover in 1957 and isolated incidents had occured at various other places in the New Ireland District over many years. Nobody however knew definitely which species of turtle were dangerous or when. The only positive comment was that "If the turtle comes to you, do not eat it. If you go after it and catch it, it is good for eating." It is by no means clear what was meant by this.

Reviewing the literature (Dewdney 1967, Halstead 1959, Bell 1972.) it appears that several species of turtle, notably the Hawksbill, the Green Sea Turtle (CHELONIA MYDAS) and the Leatherback Turtle (DERMOCHELYS CORIACEA) do became poisonous to eat at times, possibly by a process analogous to ciguatera poisoning in fish. The presence of neurological symptoms generally favours this diagnosis rather than a bacterial intoxication, although this undoubtedly can occur. In spite of the risks and local uncertainty about the nature of turtle meat poisoning, turtles continue to be eaten in large number here, and so presumably intoxication will continue to occur from time to time.

2. Cone Shell Poisoning

A 35 year old man was admitted unconscious after having been diving on a reef in the Kavieng harbour the previous night with the aid of a flashlight he had picked up a coneshell, which he had placed in his trouser pocket. The shell was later identified as CONUS GEOGRAPHICUS. A short while later he felt a sharp prick in his thigh, but thought little of it. While cycling home he felt weak, and went straight to bed. His family later noticed that he was not moving his chest when he breathed, and was not rousable, so they brought him to hospital. On examination he had generalized flaccid paralysis, and was breathing satisfactory but only with his diaphragm. Other observations were normal. No mark could be seen on his thigh.

No treatment was given beyond careful observation. The following morning he was fully fully conscious, although drowsy, and could move his legs. He was out of bed the next day and discharged two days later.

I subsequently showed the shell to every local member of the hospital staff. Only two said they were aware that it was dangerous; one of these was from Manus District, the other, a mixed race person. This suggests poisoning by Cone Shells is very rare since otherwise one would expect local folk lore to have stored the information.

In the majority of cases of stings or bites which present to hospital (3-4 a month in Kavieng), no positive identification is made, because the victim is injured while wading; the injury is to the leg or foot is under water and the attacking creature is not seen. Occasionally an identification can be made if the patient has subsequently speared the animal (e.g. a stone-fish) or if some part of the sting or proboscis is left in the wound.

R. Likeman,
Kavieng Hospital, KAVIENG.
The Diseases & Health Service of Papua New Guinea Dept. of Public Health.

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A NOTE ON THE DISTRIBUTION AND PREVALENCE OF ANOPHELES SUBPICTUS GRASSI

Sir—

Anopheles subpictus has been recorded from the following localities - (1) the Port

Moresby area (Hill, 1925, and current surveys by Dept. of Health); (2) Motu Motu, 150 miles west of Port Moresby (Bang et al., 1947); (3) Mirivasi in the Papuan Gulf (Roberts and O'Sullivan, 1948); (4) D'Entrecasteaux Islands (Spencer, 1965); (5) Wewak (Peters & Christian, 1963); (6) Milne Bay (Lee & Woodhill, 1944); (7) New Britain (Lee & Woodhill, 1944; Spencer, Spencer, & Venters, 1974); (8) Madang (Dept. Health report, 1974).

With such a wide known distribution, this species will undoubtedly be found in many other localities along the Papua New Guinea coastline.

Bang et al. (1947) recorded that this species was "extremely prevalent, came frequently to feed on humans, was present in houses in the daytime, and was found infected" (with malaria).

A. subpictus was found at numerous points around the entire perimeter of the three large islands of the D'Entrecasteaux Group, sometimes in large numbers. Specimens occurred in all types of catch - day and night indoor-resting, day outdoor-resting, leg-biting, exit-traps - and occurred commonly. Larvae were readily found. In 1957 this species constituted 1% of 2228 anophelines taken in leg-biting catches on Goodenough Island in this Group; in 1958 it represented 14% of a total of 1572 anophelines. Breeding was in pools and on the edges of brackish tidal creeks.

As *A. subpictus* occurs in the Port Moresby area it is possible that specimens will be found in aircraft originating from there and landing at Australian airports.

M. Spencer
Formerly Entomologist, Dept. of Public Health, Papua New Guinea

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SEA TURTLES

Colin Limpus
Colin James Limpus, M.Sc., Ph.D., Dip.Ed., is a Senior Zoologist with the Queensland National Parks and Wildlife Service at Townsville. He has been studying and writing about marine turtles for about 20 years, virtually all his professional life. (National Parks and Wildlife Service, Paiarenda, Townsville, Qld. 4810).

◄ Sea turtles, which occur in all tropical seas, are a regular food of coastal peoples. Some turtles are toxic.

Sea turtles are hunted widely for food throughout tropical areas. They form a significant part of the diet of many coastal peoples and hundreds of thousands of sea turtles are eaten annually. Occasionally some turtles are toxic and when such a turtle is shared within a village community, it can be devastating causing the deaths and/or debilitation of entire families or even a major portion of a village. Fortunately such events do not occur often.

Turtle poisoning is now a rare event in northern Australia. It has not been reported from here during the last 40 years. While locals in Torres Strait talk of deaths from eating Hawksbill Turtles at 'about the Second World War' there appear to be no remaining medical records from the area from that time. The popular account by Hudson (1935) of the death of five breast-fed infants following the eating of a Hawksbill Turtle by their mothers appears to be the only first hand account of death due to turtle poisoning in Australia.

The Hawksbill Turtle is regarded as poisonous by most indigenous coastal-dwelling people and turtle poisoning has occurred often enough in the past for it to be evident in their traditions. It is rarely eaten but, if a Hawksbill is to be eaten, strict rules govern its preparation. Only selected persons in a community (usually elders) are permitted to prepare Hawksbill Turtle for eating. Care must be taken in the butchering of the turtle to remove poisonous parts prior to cooking. In different districts, different organs must not be cut during preparation: gall bladder, urinary bladder or a 'poison gland' in the ventral thoracic region, neck or shoulder. MacGillivray (1852), naturalist on HMS *Fly* in 1849, recorded that for the natives of Prince of Wales Island, Torres Strait, 'hawksbill turtle and its eggs are forbidden to women suckling'.

The Australian endemic Flatback Turtle has been identified as poisonous to eat only from Bamaga and then only in an unsubstantiated report (Limpus *et al.* 1983). Elsewhere in north Queensland, Northern Territory and Western Australia the Flatback has been eaten often, with no reported poisoning.

The Green Turtle has not been implicated in any poisoning case in Australia despite the fact that it is eaten frequently.

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TOXIC PLANTS & ANIMALS
A GUIDE FOR AUSTRALIA

1987

Editors

JEANETTE COVACEVICH

PETER DAVIE

JOHN PEARN

urtle poisoning the problem is here considered rom a world wide perspective.

LIST OF SEA TURTLES REPORTED AS POISONOUS

The following species have been implicated in oisoning cases (see Silas and Fernando 1984; lalstead 1970 for reviews).

FAMILY CHELONIIDAE: *Caretta caretta* Linnaeus) (Loggerhead Turtle*); *Chelonia epressa* (Garman) (Flatback Turtle*); *Chelonia nydas* (Linnaeus) (Green Turtle); *Eretmochelys mbricata* (Linnaeus) (Hawksbill Turtle).

FAMILY DERMOCHELIDAE: *Dermochelys coriacea* (Linnaeus) (Leatherback Turtle*).

The asterisk denotes doubt about the validity of the report or the validity of the species identification in reports available. Some Hawksbill Turtles and, less frequently, some Green Turtles are also poisonous to eat.

GEOGRAPHIC DISTRIBUTION OF REPORTED CASES OF TURTLE POISONING

The main problem areas in which fatalities from turtle poisoning occur appear to be within

able 1: Incidence of fatalities among cases of sea turtle oisoning. ? denotes uncertainty in the data recorded.

Locality	Date	Species of turtle eaten	No. of cases	No. of deaths: Adults	Children (breastfed)	Total	Author
NDIA							
uticorin, Tamil Nadu	02 Jan 1961	*Eretmochelys imbricata*	9	1	2	3	S&F
akkiwadangarai, Quilon	27 May 1961	*Eretmochelys imbricata*	130	14	4	18	PHRBP, S&F
unnadagal, near Tuticorin	19 Apr 1977	*Chelonia mydas*	250	0	5 (2)	5	S&F
lanapad, near Tuticorin	03 Aug 1977	*Eretmochelys imbricata*	300	2	8 (1)	10	S&F
olachchi, Kanyakumari Dist.	1979	unknown	many	0	4	4	S&F
uticorin	Jun 1980	*Eretmochelys imbricata*	—	1	9 (1)	10	S&F
uticorin	22 May 1983	*Chelonia mydas*	6	0	4 (1)	4	S&F
RI LANKA							
anatura, S. of Columbo	Oct 1840	*Chelonia mydas*	28	—	—	18	Te
aruppankulyirupu	Jun 1888	*Eretmochelys imbricata*	—	—	—	12	D
ondativu, Jaffna	Jun 1921	*Eretmochelys imbricata*	24	—	—	7	D, Ha
addukoddai, Northern Provence	Apr 1923	*Eretmochelys imbricata*	—	—	—	4	D
AIWAN							
aryu	26 Apr 1939	unknown	57	—	—	7	Ha
HILIPPINES							
ntayan Is. near Cebu	Nov 1917	"large turtle" ? *Chelonia mydas*	33	—	—	14	Ta
nao, Cotabato, Mindnao	05 Feb 1954	*Eretmochelys imbricata*	—	5	6	11	R&C
mini, Cotabato	1954	*Eretmochelys imbricata*	—	—	—	—	R&C
DONESIA							
etherland Indies"	1933	unknown	2	—	—	1	Ha
tavia	1935	"large turtle" ? *Caretta caretta* or *Eretmochelys imbricata*	4	—	—	1	Ha
oi, Japan Is., West Irian	3 May 1935	*Eretmochelys imbricata*	52	—	—	9	Ha
ipun, West Irian	24 Aug 1954	unknown	6	—	—	2	Ha
PUA NEW GUINEA							
asea Peninsula	before 1979	*Eretmochelys imbricata*	35	0	2	2	S
STRALIA							
Queensand	before 1908	*Eretmochelys imbricata*	1	0	0	0	B
rres Strait	before 1935	*Eretmochelys imbricata*	5	0	5 (5)	5	Hu
NTRAL PACIFIC							
ribai, Gilbert Islands	about 1949	*Eretmochelys imbricata* group	—	—	—	5	Ha
RIBBEAN							
ndward Islands	1697	unknown	2	0	0	0	Ha

hor code: B = Banfield (1913); D = Deraniyagala (1939); Ha rom a series of papers reviewed by Halstead (1970); Hu = son (1935); PHRBP = Pillai et al. (1962); R&C = Ronquillo Cacas-Borja (1963); S = Soning (1982); S&F = Silas and ando (1984); Ta = Taylor (1921); Te = Tennent (1861).

Loggerhead Turtle, *Caretta caretta*.

the Indo-Pacific region (Table 1). All recorded fatalities have been confined to the region bounded by Torres Strait in northern Australia, Gilbert Islands in the central Pacific, Taiwan and India. In the western Indian Ocean, Hughes (1973, 1976) reported that it had been illegal to sell Hawksbill meat in Mauritius since 1884 as a result of numerous poisonings on the island. However, he could find no documented cases of Hawksbill Turtle poisoning in Madagascar where the species is eaten by some Malgache. Turtle poisoning is extremely rare in the Atlantic, Caribbean and east Pacific regions (Carr 1952). McKeown (1977) did not refer to the Hawksbill Turtle as poisonous when discussing the importance of the species as a food resource in the Solomon Islands. Bustard (1972) reported 'the hawksbill is eaten ... in Fiji without, to my knowledge, any ill effects being reported'.

TOXIN

The toxin responsible has been named chelonitoxin but it has not been isolated or studied. Good clinical studies of the effect of the toxin also are lacking. It is assumed to be a neurotoxin. While the apparent central neurological effects in the life threatening stages of severe turtle poisoning are reminiscent of ciguatera, the pronounced interaction with the upper gastro-intestinal tract during earlier stages is not. It can not be assumed that the same toxin is involved in turtle and ciguatera poisoning.

ORIGIN OF THE TOXIN

In some countries, e.g. western Philippines, Hawksbill Turtles are regularly eaten with no known incidence of poisoning. In other places, e.g. south eastern India, and central Philippines, Hawksbill poisoning has been well documented. In areas where toxic Hawksbill Turtles occur, toxicity is sporadic and may occur at any time of the year. The origin of the toxin is unknown but the majority of researchers are of the opinion that the toxin is derived from poisonous marine algae or invertebrate animals eaten by the turtles. While there is no direct evidence to support this, it offers an explanation about why not all Hawksbill Turtles are toxic. The concept of the toxin passing along a food chain is further supported by the observation that the toxin has been passed to the breastfed babies from nursing mothers who have eaten part of a poisonous turtle. In extreme cases the breastfed babies have died without their mothers suffering any recognised symptoms (Silas and Fernando 1984). If these deaths are really the direct result of the toxin, then this is a unique toxin that can kill the breastfed babe without harming the mother.

In areas of the Philippines where Hawksbill Turtles are eaten regularly without ill-effects, it appears that the viscera are usually discarded and only the skeletal muscle is eaten. Elsewhere with reported poisoning cases, when the parts of the turtles eaten are identified, parts of the viscera have been eaten as well. While empirical evidence is lacking, this suggests that not all parts of a poisonous Hawksbill Turtle are equally toxic.

The eggs of sea turtles have not been found to be poisonous.

TOXICITY

Ingestion of poisonous turtle flesh can be lethal to humans and animals such as dogs and goats. Silas and Fernando (1984) reported six separate incidents of groups of people eating turtles — 723 individuals were poisoned with a fatality rate of 8%. Children appear to be more at risk than adults. Children younger than 12 years accounted for 49 (68%) of 72 fatalities from 12 turtle poisoning incidents for which the age sturcture of the fatal cases has been reported (Table 1). Breastfed babies who died following their mother's ingestion of poisonous turtle flesh accounted for 11 of these fatalities.

The freshness of the turtle meat appears to have no bearing on the toxicity. The toxic components are neither removed through

washing the meat during preparation nor destroyed during normal cooking procedures.

MEDICAL ASPECTS

The clinical aspects of sea turtle poisoning were reviewed by Silas and Fernando (1984). The symptoms which develop from within a few hours to even a week after the ingestion of poisoned turtle flesh are reported to consist usually of nausea, vomiting, diarrhoea, tachycardia, pallor, severe epigastric pain, sweating, coldness of the extremities and vertigo. Acute stomatitus consisting of a dry burning sensation of the lips, lining of the mouth and throat is sometimes reported; so also in some cases, a sensation of tightness of the chest. Though there is pronounced hypersalivation, swallowing becomes difficult and the patient may be lethargic and unresponsive. The oral symptoms are said to develop gradually and become increasingly severe after a few days resulting in the tongue developing a white coating and eventually becoming covered with multiple pinhead size reddened pustular papules. The pustules may break down into ulcers or persist for several months. Deep reflexes may be diminished. In severe cases somnolence is pronounced. It may be difficult to awaken the patient who gradually lapses into coma which is rapidly followed by death. Children may develop convulsions shortly before death. Death has often been attributed to respiratory failure.

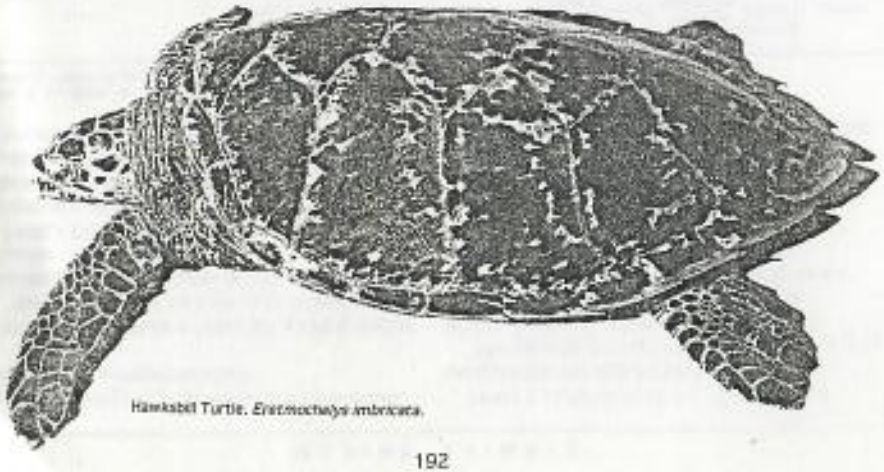
The toxins/toxin appear/appears to have no direct action on the heart and has elicited no allergic responses to date.

TREATMENT

No specific treatment has been recommended for turtle poisoning. For emergency medical advice telephone the Marine Envenomation Hotline in the Townsville General Hospital Casualty Section: — telephone no. (008) 015160 (local call fee only, from anywhere in Australia).

Because so little is known of the problem the following steps are recommended in the event of a suspected turtle poisoning case:

1. Seek immediate medical assistance. While waiting for medical help, the patient should be kept calm, encouraged to drink (not alcohol) to offset dehydration, and observed for respiratory problems. Airways should be kept clear should vomiting or convulsions occur, and ventilatory support administered where necessary.
   Where appropriate medical care is available the following generalised treatment should be considered:
   - intravenous rehydration
   - prevention of hypoxia
   - monitoring of vital functions
   - in severe cases, suppression of convulsions with appropriate drugs.
2. Freeze any remaining turtle flesh (cooked or uncooked) as soon as possible for analysis.
3. Recover the remaining parts of the turtle, especially the head or carapace, for positive identification.
4. Contact the author, who will liase with the medical personnel in further investigation of the problems of turtle poisoning.



Hawksbill Turtle. *Eretmochelys imbricata*.

PREVENTION

There are no distinctive features which allow the individual toxic turtle to be recognised. Within Australia, do not eat Hawksbill Turtles. In other countries, as a general rule eat only Green Turtle. If one is considering eating turtle in an area where the species has been reported to be toxic, then a portion of the meat should be tested. It is usually recommended that some of the turtle (I would suggest liver and muscle) be fed to a dog, cat or bird and that 24 hours elapse before the turtle is eaten. Nursing mothers and children should be discouraged from eating turtle flesh.

Green Turtle. *Chelonia mydas*.

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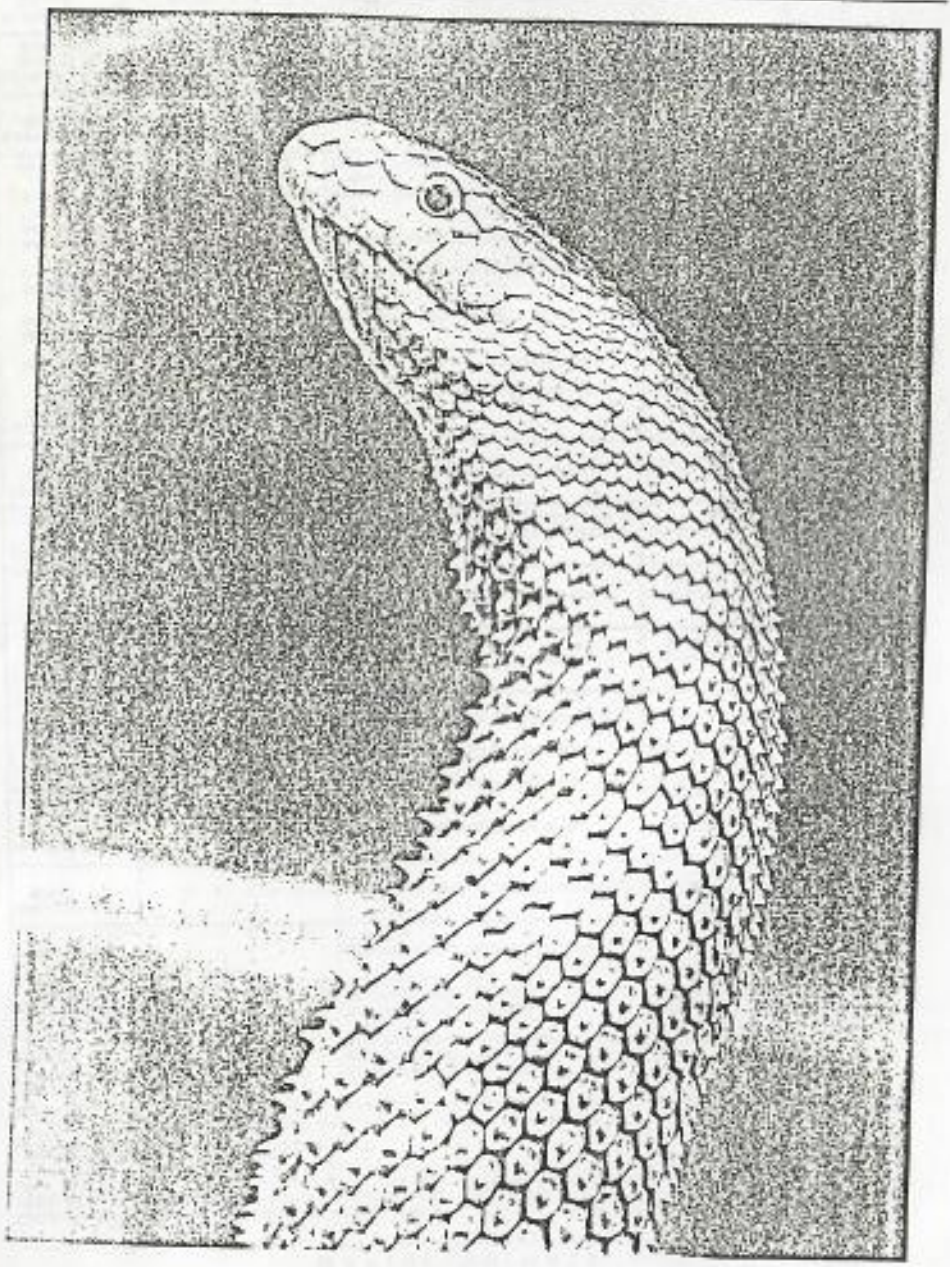


SEA SNAKES

Colin Limpus

Colin James Limpus, M.Sc., Ph.D., Dip Ed., Senior Zoologist, Queensland National Parks and Wildlife Service has been interested in reptiles (especially snakes) virtually all his life. This led him to studies in sea snake toxicology. Current research interests centre on the population ecology of sea turtles and crocodiles. (National Parks and Wildlife Service, Paluarenda, Townsville, Qld. 4810)

◄ *Laticauda hydrophis*, a very toxic species, is common in turbid inshore waters of northern Australia

Sea snakes (family Hydrophiidae) are specialised aquatic snakes that inhabit the tropical and warm temperate waters of the Indo-Pacific Oceans. They are closely related to the terrestrial snakes of the family Elapidae which includes most Australian venomous land snakes as well as cobras and kraits. Both families are characterised by relatively short hollow fangs at the anterior of each maxillary bone (i.e. fangs towards the front of the upper jawline). Their fangs can not be rotated to lie against the roof of the mouth as is the case with vipers and rattlesnakes. The most obvious external characteristics of the sea snakes which distinguish them from other snakes families are the upright paddle-shaped tail and the valvular nostrils. Sea snakes which have scales, nostrils and no gill slits should not be confused with eels which are scaleless fish with gill slits but no nostrils.

Currently approximately 52 species of sea snakes are recognised. About 32 of these have been recorded from the waters of the Australian continental shelf (Table 1). There is still much confusion in their taxonomy. Identification of many species requires dissection to determine such features as the type of muscle attachment to the venom gland or the number of tooth sockets in the maxillary bone. Keys for the identification of the Australian sea snakes can be found in Cogger (1983) and Dunson (1975).

All the species occurring in Australian waters are essentially marine, although at least three species (*Hydrophis elegans*, *Aipysurus eydouxii* and *A. laevis*) can also be found well into the upper tidal reaches of our rivers. There are no fresh water 'sea snakes' in Australia, as is the case in the Philippines and Solomon Islands. The general feeding habitats of the Australian species are summarised in Table 1. All except those of the genus *Laticauda* are totally aquatic, spending their entire life in water, even to the extent of bearing live young at sea. They must however come to the surface to breathe. The *Laticauda* species are semi-aquatic, spending most of their time ashore where they also lay parchment-shelled eggs. They feed at sea. There have been no *Laticauda* colonies documented from Australia and the isolated specimens recorded have probably been waifs. Of the fully aquatic species, all except *Pelamis platurus* feed

Table 1: Sea snakes of Australia and the surrounding continental shelf waters with a summary of their distribution, habitat, diet and mode of reproduction. Distribution code: I/M = Indonesia-Malaysia, TS = Timor Sea, NWS = Northwest Shelf, AS = Arafura Sea, GC = Gulf of Carpentaria, GP = Gulf of Papua, CS = Coral Sea including the Great Barrier Reef.

Sea snake species	Distribution							Life style and principal habitat	Diet	Reproduction	
Genus species	I/M	TS	NWS	AS	GC	GP	CS			Eggs	Live young
Aipysurus											
apraefrontalis		+	+	+				benthic feeding, coral reefs	fish		
duboisi		+	+	+	+	+	+	benthic feeding, coral reefs	fish		+
eydouxii	+	?	?	+	+	+	+	benthic feeding, muddy bottom estuaries and bays	fish eggs (blennid)		+
fenestrulatus		+	+	+				benthic feeding, coral reefs	fish		
fuscus		+	?					benthic feeding, coral reefs	fish, fish eggs		+
laevis		+	+	+	+	+	+	benthic feeding, reefs	fish, prawn, crab, worm, fish eggs		+
tenuis		?	+	+				benthic feeding, ? soft bottom inshore waters			+
Acalyptophis peronii		+	+	+	+	+	+	feeds down burrows, soft bottom inshore waters	fish (goby)		+
Astrotia stokesii		+	+	+	+	+	+	benthic feeding, inter-reefal areas	fish (frogfish, stonefish, grunters)		+
Disteira kingi		?	?	+	+	+	+	benthic feeding, soft bottom inshore waters	fish (elongate)		+
major		?	+	+	+	+	+	benthic feeding, soft bottom inshore waters	fish (elongate)		+
Emydocephalus annulatus		+	+	+	+	+	+	feeds down burrows, coral reefs	fish eggs (blennid, goby)		+
Enhydrina schistosa	+	?	?	+	+	+	+	benthic feeding, shallow soft bottom waters	catfish (tachysurid, plotosid)		+
Ephalophis greyi			+		+			? intertidal and shallow inshore waters	fish (goby)		
mentoni				+	+			? intertidal and shallow inshore waters			
Hydrelaps darwiniensis			+	+	+	+		muddy intertidal and shallow inshore waters	? crabs		+
Hydrophis											
belcheri	?	?	?	?	?	?	?				
caerulescens	+	?	?	?	+	?		benthic feeding, soft bottom inshore waters	fish (elongate)		+
elegans			+	+	+	+	+	benthic feeding, soft bottom inshore waters	fish (very elongate)		+
fasciatus	+	+		+					fish (small eels)		+
gracilis	+	?	?	?	?	+	+	feeds down burrows, soft bottom inshore waters	fish (small eels)		+
inornatus	+	?	?	?	+	?	?				
melanosoma			+	+	+	+	+	benthic feeding, soft bottom inshore waters			+
melanocephalus					?	+	+	benthic feeding, coral reefs	fish (small eels)		+
melanosoma			+			+					+
Pelamis platurus						+	?				+
ornatus	+	+	+	+	+	+	+	benthic feeding, soft bottom inshore waters	fish		+
perlicus				+	+	+		benthic feeding			+
Laticauda colubrina						+	+	benthic feeding, muddy bottom inshore waters	fish (non specific)		+
Laticauda											
crockeri						+	+	semi aquatic, benthic feeding at sea	fish (small eels)	+	
laticaudata	+					+	+	semi aquatic, benthic feeding at sea	fish (elongate)	+	
Pelamis platurus	+	+	+	+	+	+	+	surface feeding, oceanic planktonic	fish (non specific)		+

at the bottom. Some even feed exclusively in burrows made by fish and shrimps. The need for these bottom feeding snakes to come to the surface to breathe limits their occurrence to the relatively shallow waters of the continental shelf. Only *Pelamis* feeds at the surface, floating motionless waiting to strike at small fish seeking refuge beneath it. *Pelamis* is the only sea snake able to colonise oceanic waters, being dispersed along drift lines. As a result it is the most widely distributed sea snake, being found from the African coast in the west to the American coast in the east (Dunson and Ehlert 1971).

The bottom-feeding sea snake species tend to feed on fish that are relatively sedentary and dwell along the bottom, in burrows in the bottom or in reef crevices. The fish eaten by sea snakes are usually elongate and tend to be scaleless or to have fine scales. The most commonly eaten species are from the families which include the eels and gobies. Voris and Voris (1983) discuss their feeding strategies. The various species display an interesting array of specialisation (Table 1). The very small headed *Hydrophis gracilis* feeds on small eels that live in burrows. The large headed *Astrotia stokesi* feeds on bulky fish such as banded frogfish and stonefish which sit on the bottom while *Enhydrina schistosa*, another large headed species, feeds almost totally on the more active fork-tailed catfish. The more extreme feeding specialisation occurs with *Emydocephalus annulatus* and *Aipysurus eydouxii* which feed exclusively on clumps of fish eggs laid in burrows or depressions respectively. The least specialised feeder of the sea snakes would be *Aipysurus laevis* which feed opportunistically on a range of fish species, crabs, prawns, clumps of fish eggs and polychaete worms. Sea snakes, like their terrestrial relatives can distend their jaws to swallow prey items larger than the diameter of their head and neck. *E. schistosa* can swallow catfish up to twice the diameter of the snake's neck. The poisonous spines of such prey present no major problems during swallowing. Once bitten a fish is rapidly paralysed in a fully relaxed state and any spines fold back as the fish is swallowed head first.

Sea snakes are themselves food for a limited range of predators. Sharks, especially Tiger Sharks *Galeocerda cuvieri*, consume large numbers of sea snakes. While some teleost fish have been reported to prey on sea snakes, most tested have refused to eat them (Heatwole 1975). The marine feeding raptors, Osprey (*Pandion haliaetus*), White-bellied Sea Eagle (*Haliaetus leucogaster*) and Brahminy Kite (*Haliastur indus*) regularly feed on sea snakes in some localities.

More detailed accounts of their biology by species can be found in Cogger (1983), Dunson (1975) and Smith (1926).

TOXICOLOGY

Sea snakes display a great similarity with respect to their venom apparatus, the properties of the venom and the effects of envenomation. Therefore for the purposes of this account the family will be treated as a whole. Those wishing to pursue a more indepth treatment of the toxicology of the Australia sea snakes should refer to Baxter and Gallichio (1974), Limpus (1978 a,b), Minton (1983), Sutherland (1983), and Zimmerman and Heatwole (this volume).

Venom Apparatus

The venom apparatus of a sea snake is similar to a terrestrial elapid. It consists of paired venom glands, one on each side of the head lying posterior to the eye, and each linked to the hollow fang by a venom duct. The fangs may be partly obscured by folds of mucous membrane and appear as lumps on either side of the roof of the mouth.

The fangs are specialised tubular teeth. At the anterior end of each maxillary bone are two sockets into which fangs can be cemented (see figure). There is a diastema between the fangs and the other backwards recurved teeth (maxillary teeth) attached to the posterior of the maxillary bone. There are other teeth attached to the palatine and pterygoid bones in the roof of the mouth and to the dentary bone of the lower jaw but they are not part of the venom apparatus. Fangs are constantly being replaced throughout the life of the snake. Generally there is only one fang attached to a maxillary bone. The functional fang is equivalent to a tooth that has been rolled to form a hollow tube and operates like a hypodermic needle (see figures herein). Reclining behind the functional fang in folds of the gum is a series of developing replacement fangs. When a fang becomes chipped and cracked it can not be repaired.

Table 2: A selection of sea snake venom yields and venom toxicities relative to white mice with particular emphasis on those species occurring in Australian waters.

Species	Locality	Venom yield (dry weight) mg	Toxicity LD_{50} mg/kg	Injection route	Reference
Acalyptophis peronii	Coral Sea	0.33	0.079	s.c.	4
Aipysurus duboisii	Coral Sea	0.43	0.044	s.c.	4
Aipysurus eydouxii	Queensland Thailand	0.48 0.60	>11.7 > 4.0	i.v. i.v.	1 5
Aipysurus laevis	Queensland Coral Sea Northwest Shelf	10.45 5.26 6.60	0.25 0.084 0.13	i.v. s.c. i.m.	1 4 6
Astrotia stokesii	Queensland	31.30	0.32	i.v.	1
Disteira major	Queensland	22.80	0.21	i.v.	1
Emydocephalus annulatus	Coral Sea	0.15	>25.0	s.c.	4
Enhydrina schistosa	Malaysia Thailand Vietnam	– – –	0.09 0.14 0.35	i.v. i.v. i.v.	2 2 3
Hydrophis cyanocinctus	Malaysia Hong Kong Vietnam	– – –	0.35 0.70 0.07	i.v. i.v. i.v.	2 2 3
Hydrophis elegans	Queensland Northwest Shelf	8.91 – – 5.50	0.12 0.20 0.28 0.12	i.v. i.v. i.v. i.m.	1 1 1 6
Hydrophis melanocephalus	Coral Sea	0.13	0.111	s.c.	4
Hydrophis ornatus	Northwest Shelf	8.50	0.30	i.m.	6
Laticauda hardwickii	Thailand Vietnam	– –	0.71 0.44	i.v. i.v.	2 3
Laticauda semifasciata	Taiwan Philippines	– –	0.21 0.30	i.v. i.v.	2 2
Pelamis platurus	Taiwan Coral Sea	– 0.25	0.18 0.067	i.v. s.c.	2 4

References: 1 Limpus 1978 a, b, 2 Tu and Tu, 1970, 3 Cheymol et al. 1967, 4 Minton, 1983, 5 Tu, 1974, 6 Tamiya and Pyffer, 1974

use their venom to immobilize prey are extremely toxic to natural prey species and to mammals. However, the fish-egg eating species have been found to be effectively non-toxic to mice (Table 2). Excluding these latter species, most sea snakes have a toxicity that is similar to that of most of the dangerous Australian elapids when their activity is compared using mice (Sutherland 1983).

Studies of the toxic components of sea snake venom usually identify 1–3 neurotoxic fractions which account for most of the protein content and most of the toxicity of the whole venom. These neurotoxins contain 60–72 amino acid residues and have a molecular weight of about 6500. Their principal activity is to block nerve conduction by competing with acetylcholine at the postsynaptic membrane receptor sites. A myotoxic protein with phospholipase A activity which causes myoglobinuria in mice has been isolated from *Enhydrina schistosa* (Fohlman and Eaker 1977). A similar myotoxin is probably present in most sea snake venoms. For further information on the individual toxins contained in sea snake venoms, the reader is directed to the following: Fohlman and Eaker (1977), Fox *et al.* (1977), Frylund *et al.* (1972), Maeda and Tamiya (1976, 1978), Tu and Toom (1971), Zimmerman and Heatwole (this volume).

Venom Action

In pharmacological preparations sea snake whole venom has little or no effect on the functioning of heart muscle or smooth muscle. Nerve conduction along axons is not effected by even high concentrations of the venom. However, in isolated nerve-muscle preparations, the venom blocks the transmission across the neuromuscular junctions without affecting the contractile capacity of the skeletal muscle.

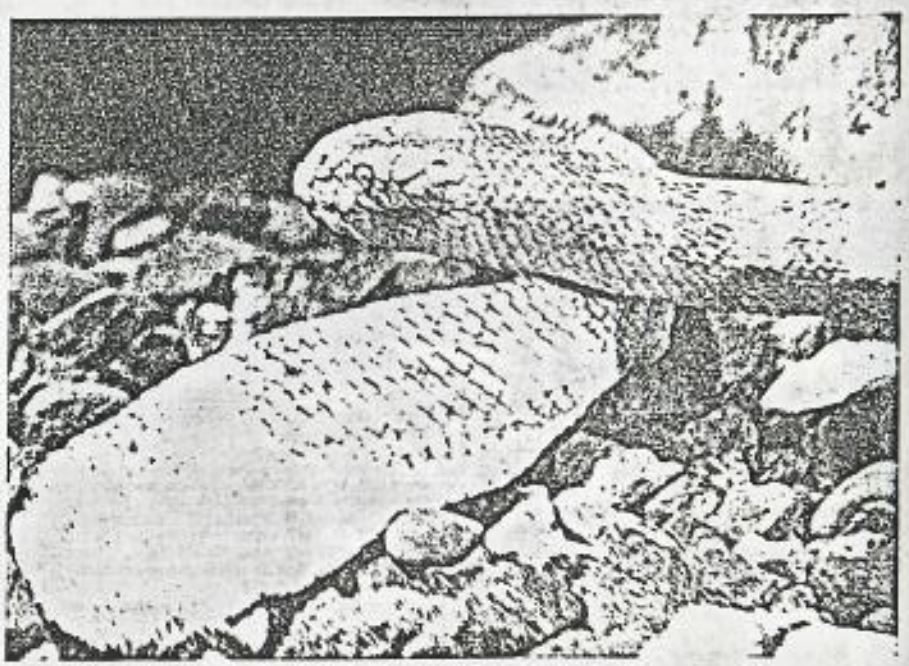
In prey species such as fish, envenomation causes rapid onset of flaccid paralysis of skeletal musculature (including the respiratory musculature) and death follows soon after.

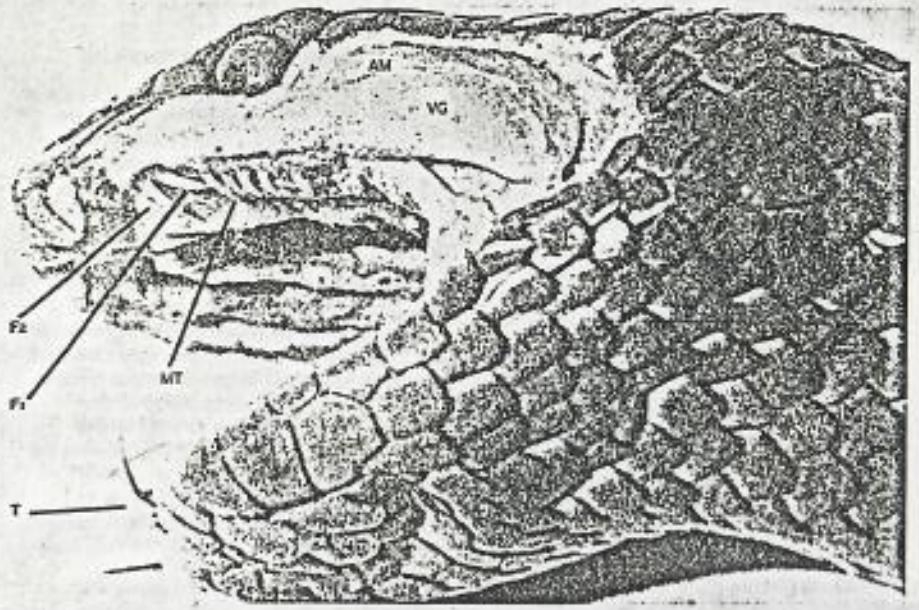
In contrast with the large number of bites and fatalities reported from southeast Asia, sea snake bites occur infrequently in Australia, and no fatalities have been confirmed. Nevertheless, several cases have been reported recently in which the patient probably would have died had not antivenom been administered (Mercer *et al.* 1981, *Astrotia stokesii*; Fulde and Smith 1981, probably *Pelamis platurus*; Audley 1985, *Astrotia stokesii*; Dobb 1986, species unidentified).

SIGNS AND SYMPTOMS

Apart from an initial mild sting at the time of the bite, sea snake bite causes no pain at the envenomation site. This contrasts with the immediate intense local pain associated with most other dangerous marine envenomation. Like terrestrial elapids, sea snakes can deliver single or multiple bites. In some instances the puncture marks may be difficult to discern. There is usually no tissue necrosis at the bite site. It should be noted that a bite does not necessarily mean an envenomation has occurred. The release of the venom is under the control of the snake and it may choose to release some or none of its venom in a particular bite. This probably accounts for most of the extreme variability in degree of debilitation following sea snake bite. Most bites result in no signs of envenomation. In contrast, in the event of a severe envenomation all or some of the following signs may be in evident (after Sutherland 1983):

Disteira major.





▲ The anatomy of the sea snake venom apparatus is illustrated in this dissected head of *Aipysurus laevis*. AM = *adductor externus superficialus* muscle. F_1 = fang. F_2 = reserve fang. MT = maxilary teeth. VG = Venom gland. The blind tongue (T) protrudes from the lower jaw but is not part of the venom apparatus.

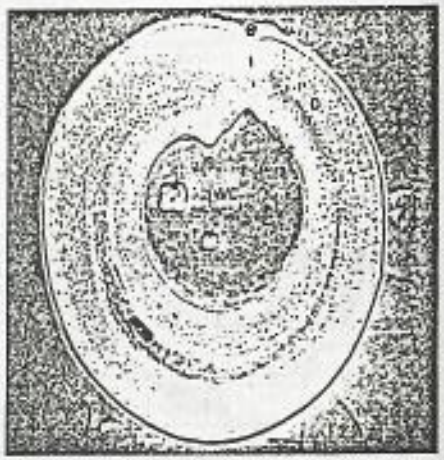
▼ Scanning electron micrograph (10 4X) of the maxillary bone and attached teeth of the sea snake *Disteira major*. M = maxillary bone. F_1 = functional fang. F_2 = reserve fang. MT, ... MT_5 = series of grooved maxillary teeth.

Instead it is replaced by the largest reserve fang which rotates up into the adjacent vacant fang socket and is cemented into position. Only then does resorption of the bone of attachment of the fang undergoing replacement occur. The worn out fang eventually falls out, leaving an empty socket next to a fully-funtional new fang. It is these fangs undergoing the final stages of replacement which are on occasions left behind following a bite. Just prior to this stage of replacement there will be for a brief time two fangs attached to the one maxillary bone.

The fangs of mature specimens of most sea snakes are in the range 2.5 - 4.5 mm (Limpus 1978a). Such fang lengths are more than adequate for effectively delivering venom into dermal tissue of prey species and humans. Generally the larger the head the longer the fangs. *Astrotia stokesii*, which has the largest head of all sea snakes can have fangs up to 7mm in length. The two fish-egg eating species, *Aipysurus eydouxii* and *Emydocephalus annularus*, have the shortest fangs, usually less than 1.5 mm. The fang lengths of sea snakes are comparable to those of the Australian elapid snakes.

The venom gland is a compound tubular gland that secretes a mixture of products. Proteins are synthesised mostly in the posterior bulbous portion of the gland while mucopolysaccharides are synthesised in the anterior portion. The many small tubules within the gland coalesce into a central primary duct that runs the length of gland. The secretary products of the gland are stored in the tubules until used. The bulbous region of the gland is surrounded by a supporting capsule of fibrous connective tissue and smooth muscle. The more anterior portions of the gland have only the fibrous connective tissue sheath. The bulbous region of the venom gland is free along its lower margin but attached to the *adductor externus superficialus* muscle along its upper and posterior margins. The venom gland opens at the base of the anterior face of the fang. Contraction of the venom gland musculature squeezes the venom from the gland to the fang. Pressure from the mucous membranes surrounding the fang directs the venom flow through the tubular fang. The snake is able to expel venom from each gland independently.

Some dangerous misunderstandings of the biting capability of sea snakes exist. Many people believe that sea snakes have small fangs and are therefore not very dangerous. Their fangs are certainly shorter than those of some species of land snake, especially vipers and rattlesnakes. Such an argument is however irrelevant to the question of envenomation as almost all sea snakes have fangs long enough to penetrate to at least the capillary rich dermal layers of the human skin. Another commonly hold belief is that the smallness of the sea snakes mouth would prevent it from delivering an effective bite to most parts of the human body. This is a falacy as even small-headed species such as *Hydophis elegans* have been able to bite a man's thigh.



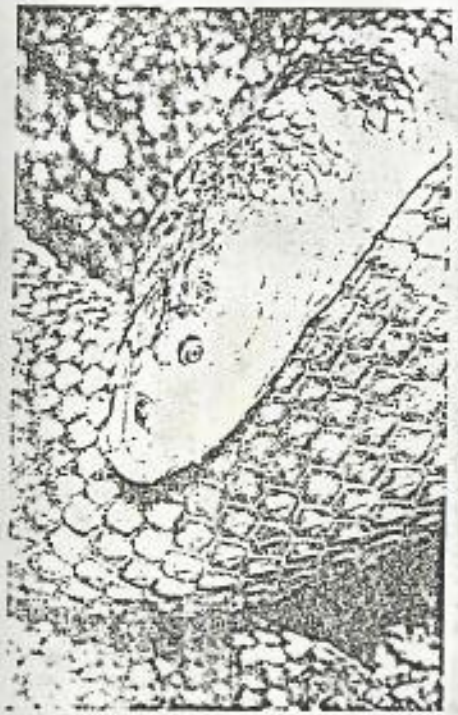
Scanning electron micrograph (120X) of a cross section of the reserve fang see previous figure. The venom canal (VC) is enclosed by folds of dentine (D). The outer enamel (E) layer of the tooth is continuous across the front face of this tubular fang.

Venom

Fresh sea snake venom is usually a clear (or slightly cloudy) colourless liquid which dries to a white solid. The venom yield from sea snakes (Table 2) tends to be smaller than that of comparable size terrestrial elapids. The larger headed species can be expected to give venom yields comparable to that of the many of the more dangerous elapids. In the extreme, large *Astrotia stokesii* may yield up to 150 mg of dry venom from a single milking. Sea snakes that

1. Generalised muscle aches pains and stiffness of movement developing within one-half to one hour of the bite. Spasms to the jaw muscles or even general tonic spasms may develop.
2. Moderate or severe pain on passive movement of arm, thigh, neck, or trunk muscles developing one to two hours after the bite.
3. Myoglobinuria (darkening) usually becomes evident on inspection of the urine three to six hours after the bite.
4. The above may also be accompanied by such signs as blurred vision, apparent drowsiness or retching.
5. Rapid collapse and shock can occur in the more severe cases.

With severe envenomation, death can be expected to occur from respiratory paralysis unless antivenom is administered.



FIRST AID

Bites from sea snakes almost always occur on the limbs. Movement of the venom from the bite site can be delayed by use of the standard pressure/immobilisation first aid method (White; Williamson *et al.* this volume). Seek medical assistance as soon as possible. Be prepared to provide artifical ventilation and extracardiac massage in the event of a severe bite.

▲ *Enhydrina schistosa*

◄ *Hydrophis elegans.*

ANTIVENOM

An effective sea snake antivenom suitable for treating sea snake bite cases has been developed by Commonwealth Serum Laboratories. If the specific sea snake antivenom is unavailable then Australian Tiger Snake antivenom or even polyvalent snake antivenom (Australia – Papua New Guinea) can be used. Refer to CSL antivenom literature or Sutherland (1983) for specific detailed clinical management of sea snake victims.

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