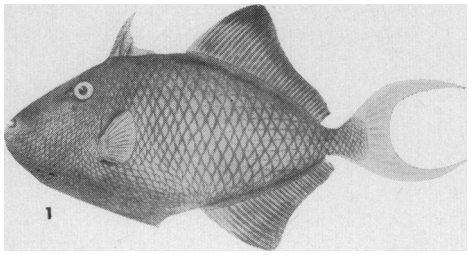

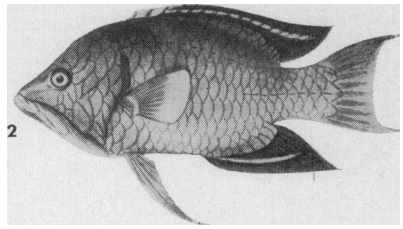

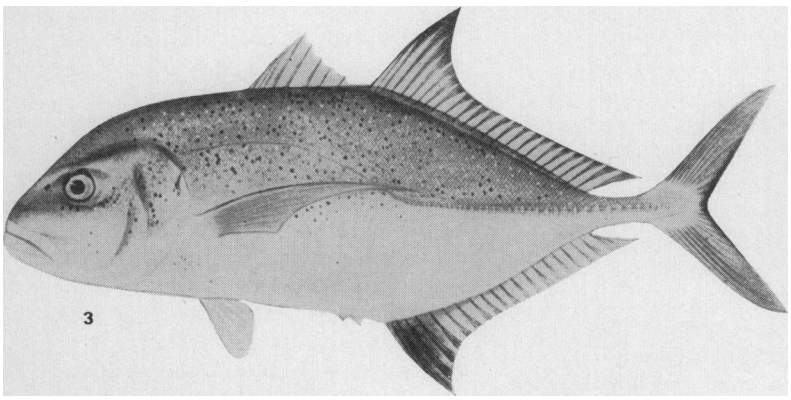

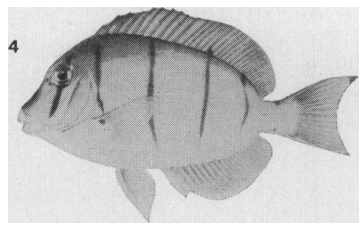

Poisonous Fishes

BRUCE W. HALSTEAD, M.D.

MAN'S KNOWLEDGE of poisonous fishes dates back to ancient times. At least one of the Mosaic laws is believed to have been aimed directly at eliminating poisonous fishes from the Israelite diet, "These ye shall eat of all that are in the waters: all that have fins and scales shall ye eat: And whatever hath not fins and scales ye may not eat: it is unclean unto you" (Deuteronomy 14: 9–10). The French archeologist Claude Gaillard reported that hieroglyphics and figures of the deadly *Tetraodon lineatus* appear frequently on ancient Egyptian tombs (*1*), and according to the Egyptologist Keimer, this scaleless species was recognized as poisonous during the time of the Pharaohs. Galen is said to have reported in his De Alimentis that the flesh of the moray is dangerous to eat (*2*). Alexander the Great forbade his soldiers to eat fish during conquests because he believed that some species produced skin disorders.

Peter Martyr, the first historian of the West Indies, made the earliest reference to ciguatera in 1555 (*3*). In de Landa's famous Relacion de Las Cosas de Yucatan, written in 1566, the lethal qualities of puffers are mentioned (*4*).

Beginning with the 19th century, hundreds of publications appeared on the subject of fish

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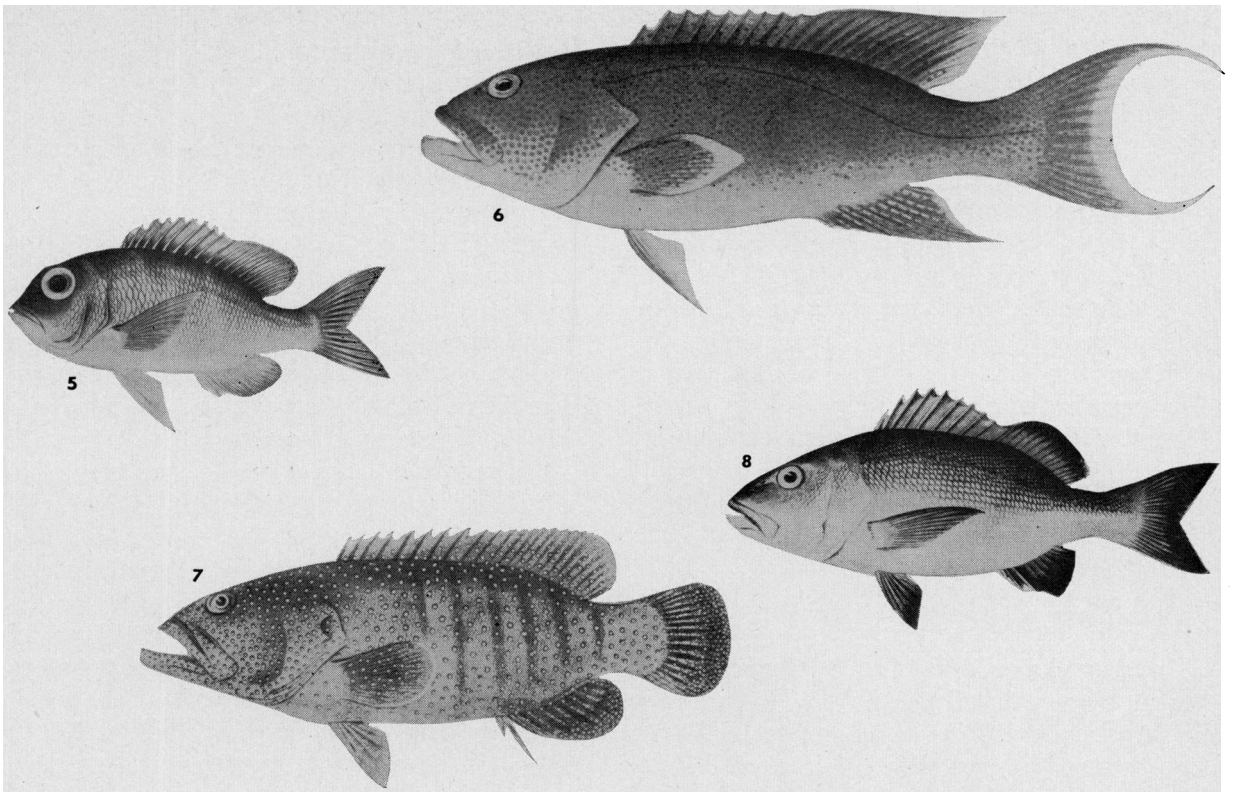


poisoning, including some comprehensive reviews and bibliographies (*3*, *5–11*).

Because of their somewhat sporadic and unpredictable appearance, generally affecting only small numbers of persons at any one time, poisonous fishes have not attracted a great deal of attention in medical circles. However, the public health significance of poisonous fishes was pointed up in the series of outbreaks which occurred in Midway, Johnston, and the Line Islands, beginning about 1943 and reaching a peak about 1946 (*12*, *13*).

Recent mass intoxications in the western Pacific have once again directed attention to the public health importance of poisonous marine organisms. These outbreaks began about 1952, became increasingly severe during 1955, and have continued until the present time, intoxicating a number estimated at more than 40,000 persons. The bulk of the outbreaks have taken place in Japan, the Philippine Islands, and more recently in Viet Nam. The causative agents were octopus, *Octopus vulgaris*, *O. döfleini*, squid, *Omnastrephes sloani pacificus*, Japanese horse mackerel, *Trachurus japonicus*, common Japanese mackerel, *Scomber japonicus*, flying fish, *Prognichthys agoo*, and oceanic bonito, *Katsuwonus pelamis*. All of the outbreaks have been seasonal, taking place during June to the middle of September. Bacteriological tests have been negative, and the degree of freshness of the organisms seems to have no bearing on the matter. The symptoms are similar to those produced by bacterial food poisoning, but no human pathogens have been isolated by Japanese epidemiologists. The mortality rate in the largest series of Japanese fish outbreaks in 1955 was 0.77 percent (*14*).

According to a report received from the Bureau of Fisheries of the Philippine Government, another series of outbreaks of fish poison-

Some fishes associated with ciguatera are illustrated across the top of these pages. They are: **1.** Triggerfish, *Odonus niger*. **2.** Wrasse, *Epibulus insidiator*. **3.** Jack, *Caranx melampygus*. **4.** Surgeonfish, *Acanthurus triostegus*. **5.** Snapper, *Monotaxis grandoculis*. **6.** Seabass, *Variola louti*. **7.** Grouper, *Cephalopholis argus*. **8.** Red Snapper, *Lutjanus vaigiensis*.

ing took place in April 1957. The complete number of persons involved has not been determined as yet, but several deaths have been reported. The causative agent in most of the Philippine poisonings has been the oceanic bonito, *K. pelamis*. Philippine public health authorities are establishing regulations prohibiting the sale of the oceanic bonito in some localities. Some intoxications from fishes have been reported in Viet Nam, but as yet no details are available as to the nature of the outbreaks.

Since intoxications resulting from poisonous fishes are frequently confused with a variety of other ailments, a review of the epidemiological and clinical aspects of the problem of ichthyosarcotoxism is timely. The chemistry of these poisons has been reviewed elsewhere (*31*).

Epidemiology

Judging from the public health and toxicological reports that have appeared to date, any marine fish can under certain circumstances become poisonous as a result of its food habits. Toxicity is not species specific, except possibly in puffers or tetraodontoid fishes. More than 300 species of marine fishes have been incriminated as causative agents in producing human intoxications of the ciguatera type alone. Most poisonous fishes are shore forms rather than oceanic inhabitants. Fishes captured in deep waters far from shore are generally safe to eat. Some of the more common families of marine fishes which have caused human intoxications are listed under the various clinical types of fish poisoning.

Poisonous fishes are largely circumtropical in distribution, but on occasion occur in temperate waters. With the exception of 2 or 3 boreal species they are currently believed to have a maximum distributional range of 45° north and 45° south of the Equator. Particularly large populations of poisonous fishes are known to occur in the central Indo-Pacific area and in the West Indies. They are more common near islands than along continental shores. Within these general areas, poisonous fishes seem to be quite circumscribed in distribution. They may vary considerably in their distribution about a single island. A species may be toxic in one part of the island, but edible in another. Lagoon fishes are more likely to be poisonous than those living on seaward reefs.

The toxicity of puffers or tetraodontoid fishes generally follows closely their reproductive cycle. The toxin content of the fish is greatest just prior to spawning. For most puffers, spawning takes place during late spring or early summer. Toxin content is lowest during fall and winter. There is some evidence that the barracuda (*Sphyraena* spp.) may follow a similar, but less pronounced, seasonal pattern. However, in most other species there is no evidence of a seasonal fluctuation.

The poison in tetraodontoid fishes is usually more concentrated in the liver, gonads, and skin, but other parts of the viscera may also be toxic. Puffer musculature is generally nonpoisonous. The distribution of poison is exceedingly variable in other kinds of fishes. If a fish is toxic, the musculature will be found to be poisonous in about 50 percent of the specimens, and the viscera, in about 90 percent or more. However, the distribution of the poison in the body of the fish varies from one individual to the next.

No consistent pattern has been observed as to the relationship between the size of the fish and its toxicity. It has been observed that unusually large specimens of jacks (*Caranx* spp.), grouper (*Epinephelus* spp.), and barracuda (*Sphyraena* spp.) are more likely to be toxic than smaller specimens. This has not been found to be necessarily true in other species.

Aboriginal peoples have numerous methods by which they attempt to distinguish a poisonous fish from an edible one. The significance of color change in silver coins, color of the fish, condition of the gills, position of the scales, ad infinitum, is generally without scientific foundation. To the best of our knowledge, one cannot detect a poisonous fish by its appearance. The most reliable field method is to feed a small sample of the fish to a kitten or a dog. If the animal remains asymptomatic for 12 hours, the fish is generally safe to eat. Investigations are currently in progress at the School of Tropical and Preventive Medicine, Loma Linda, Calif., with the hope of developing a reliable rapid chemical field test.

Origin of the Poison

The origin of ichthyosarcotoxins in nature has been the subject of much speculation and folklore. Numerous theories have been propounded, but few have any scientific basis (*15*, *16*). It is certain, with the single exception of scombroid poison, the poisons are not bacterial in origin. Putrefactive processes do not play a part in the production of fish poisons. There is no evidence of any relationship between the presence of ichthyosarcotoxins and radioactive substances in the water. It is believed that most of these poisons are derived from the food which the fish eats. Food probably plays an important role in the direct production of even puffer poison.

The most widely accepted theory propounds the idea that the toxic substances, or their precursors, are obtained directly from marine littoral algae. It is doubtful that plankton play any role in the mechanism. Herbivores feed on the plants. Carnivores feed on the herbivores and man may become poisoned from eating either of them. Stomach content analyses have revealed that algae are more frequently present in poisonous fishes than any other types of food (*17*), and toxicological tests of these algae have shown some of them to contain toxic substances (*18*). Moreover, controlled laboratory feeding tests demonstrate that it is possible to induce toxicity in carnivorous species without evidence of detrimental effects on the fish, by feeding them toxic fish flesh. Further chemical and stomach analyses will be required before the toxin-food chain theory can be definitely established. Experimental work in attempting to determine the origin of fish poisons in nature is currently in progress by the author.

The incidence of fish poisoning cannot be accurately determined since the disease is frequently misdiagnosed and generally unreported. In the past, several hundred cases of puffer poisoning were reported each year in Japan, during some years accounting for as much as 44 percent of all fatal cases of food poisoning in that country. Japanese public health authorities state that puffers continue to be their greatest single cause of food fatalities (*19*, *20*). Probably the second most common type of fish poisoning is ciguatera. Third on the list would be scombroid poisoning, with the other forms of fish poisoning being relatively uncommon. It is unfortunate that there are so few reliable statistical data available. Local public health authorities should encourage the reporting of outbreaks, since poisonous fish may constitute a serious public health and economic threat in some regions.

Recent biotoxicological surveys that have been conducted in the tropical Pacific give some idea as to the incidence of poisonous fishes in specific localities.

Locality	*Number species tested*	*Percent of species found toxic*
Phoenix Islands (*16*)	93	29
Johnston Island (*16*)	60	75
Galapagos Islands (*29*)	57	67
Cocos Island (*30*)	33	67

Among important field studies (*7*, *8*, *21–23*), Hiyama's work (*7*) is particularly useful, magnificently illustrated, and contains a valuable review of the poisonous fish problem in Saipan and the Marshall Islands. Only meager statistical data are contained in any of these reports, but they do aid in developing an appreciation of the scope of the problem in the regions discussed.

Clinical Characteristics

Since the chemical characteristics of fish poisons have not been defined, poisonous fishes may be tentatively classified on the basis of their phylogenetic relationships and symptomatology of the intoxications which they produce (*10*, *24*). Certain fish groups are classified under more than one category. Although the weaknesses of this somewhat artificial system are recognized, it appears to be the most practical classification at present. Forms of ichthyosarcotoxism caused by fishes may be subdivided into the following clinical categories (see table).

Lamprey and Hagfish Poisoning

The slime and flesh of certain lampreys and hagfishes are reported to produce a gastrointestinal upset (*32*). The clinical characteris-

Diagnostic characteristics of the various types of ichthyosarcotoxism

Type of poisoning	Causative fish	Symptomatology
Lamprey or hagfish	Lampreys, hagfish (slime, flesh).	Gastrointestinal upset(?). Precise symptomatology unknown.
Elasmobranch	Sharks, rays (liver, flesh)	Gastrointestinal symptoms predominate in mild cases; neurological symptoms in severe cases. Deaths reported, but case fatality rate unknown.
Chimaeroid	Chimaeroid or ratfish (flesh, viscera).	Neurological symptoms(?). Precise symptomatology unknown.
Gymnothorax or moray eel.	Moray eels (flesh, viscera)	Violent neurotoxic symptoms with convulsions and paralysis predominating. Case fatality rate about 10 percent or more.
Scombroid	Scombroid fishes—tuna, bonito, skipjack, etc. (inadequately preserved flesh).	Histamine-like reaction with nausea, vomiting, flushing of the face, swelling of the lips, urticaria, and pruritus. Symptoms subside usually within 12 hours. Antihistaminic drugs effective.
Puffer	Tetraodontoid or puffer-like fishes (viscera).	Rapid onset and extremely violent neurotoxic symptoms: paresthesias, motor paralysis, convulsions, death by respiratory paralysis. Case fatality rate about 61 percent.
Ciguatera	More than 300 species of tropical reef fish, including snapper, seabass, grouper, barracuda, wrasse, parrotfish, and surgeonfish.	Onset may be gradual or sudden. Symptoms mild to severe. Gastrointestinal and neurotoxic. Paresthesias, extreme weakness, joint aches, myalgia and paradoxical sensory disturbance predominate. Case fatality rate about 7 percent.
Gempylid diarrhea	Gempylids, escolars (flesh)	Diarrhea develops rapidly but is painless. No other untoward effects.

tics of the intoxication, termed lamprey and hagfish poisoning, have not been completely defined.

Elasmobranch Poisoning

The most severe cases of elasmobranch poisoning usually result from the ingestion of the liver of sharks and rays. The musculature of some elasmobranchs is also reported as mildly toxic with the symptoms seldom more than that of a mild gastroenteritis with a predominating diarrhea. Ingestion of toxic liver results in the onset, usually within a period of 30 minutes, of nausea, vomiting, diarrhea, abdominal pain, headache, a rapid, weak pulse, malaise, cold sweats, oral paresthesia, a burning sensation of the tongue, throat, and esophagus. As time progresses, the neurological and other symptoms become more pronounced, resulting in extreme weakness, trismus, muscular cramps, sensation of heaviness of the limbs, blepharospasm, dilatation of pupils, hiccups, visual disturbances, joint aches, delirium, ataxia, incontinence, dysuria, respiratory distress, coma, and death. The recovery period, if the victims recover, varies from a day or two to several weeks. The mortality rate is not known.

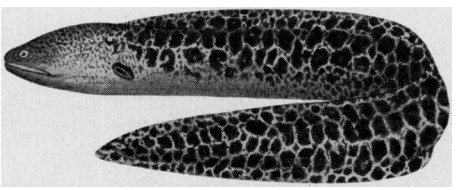
Chimaeroid Poisoning

The musculature and viscera of certain chimaeroids or ratfish have been incriminated as containing a neurotoxin. This form of ichthyosarcotoxism, if it occurs, would be termed chimaeroid poisoning. Only vague references have appeared in the literature regarding the toxicity of this group of fish, by Pellegrin in 1899, and by Phisalix in 1922. Halstead and Bunker reported in 1952 that the reproductive organs were toxic. Nothing is known about the nature of the toxin, or its relationship to other forms of ichthyosarcotoxism.

Moray Eel Poisoning

The musculature and viscera of some of the tropical marine muraenid or moray eels con-

tain a violent poison. The disease is termed gymnothorax or moray eel poisoning (*25*, *26*). Symptoms of tingling and numbness about the lips, tongue, hands, and feet, with a feeling of heaviness in the legs, usually develop within several minutes to 8 hours after ingestion of the toxin. These symptoms may be followed by nausea, vomiting, diarrhea, abdominal pain, malaise, metallic taste, sore throat, laryngeal paralysis, meningismus, laryngeal spasm, aphonia, excessive mucus production, foaming at the mouth, intense perspiration, increased body temperature, crying out as if in pain, conjunctivitis, paralysis of the respiratory muscles, ataxia, general motor incoordination, trismus, violent clonic and toxic convulsions, abnormal deep and superficial reflexes, coma, and death. Sensory reactions to deep and superficial pain are usually normal. Characteristic signs of this form of intoxication appear to be the absence of thoracic respiration with pronounced abdominal breathing, profuse perspiration, violent convulsions, purposeless movements, and the extended period of time

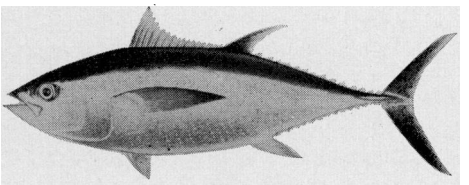


Moray eel, *Gymnothorax javanicus*

in which areflexia is present. In some cases the reflexes may be absent for a period of 2 months or more. The case fatality rate is estimated to be about 10 percent, and death is believed to be the result of respiratory paralysis.

Scombroid Poisoning

Some of the scombroids (tuna, skipjack, and bonito) may on rare occasions cause ciguatera, but usually they produce an entirely different form of intoxication, termed scombroid poisoning. This is the only type of fish poisoning in which bacteria appear to play an etiological role in the formation of the toxin. If scombroids are inadequately preserved, a toxic "histamine-like" substance is formed, possibly from the decarboxylation of histidine, a normal constituent of fish flesh. Kawabata and associates have recently termed this substance "saurine" (*33*). Victims complain of fish having a "sharp or peppery" taste. The symptoms most often present are nausea, vomiting, flushing of the face, intense headache, epigastric pain, burning of the throat, difficulty in swallowing, thirst, pruritus, swelling of the lips, and urticaria, which are typical of a histamine reaction. Symptoms generally subside within 12 hours.



Tuna, *Neothunnus macropterus*

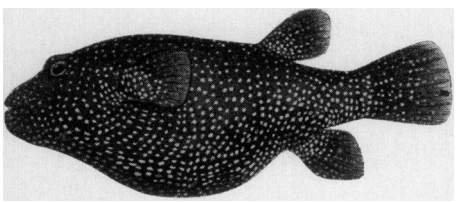
For some unknown reason scombroid fish appear to be more prone to producing intoxications of this type than other fish. The mortality rate is unknown.

Puffer Poisoning

The most violent form of fish poisoning is produced by tetraodontoid or puffer-like fishes. The disease is characterized by rapidly developing, violent symptoms. The onset and symptoms in puffer poisoning vary according to the person and the amount of poison ingested. However, malaise, pallor, dizziness, paresthesias of the lips and tongue, and ataxia most frequently develop within 10 to 45 minutes after ingestion of the fish, but cases have been reported in which the symptoms did not develop for 3 hours or more. The paresthesias which the victim usually describes as a "tingling or prickling sensation" may subsequently involve the fingers and toes, then spread to other portions of the extremities, and gradually develop into severe numbness. In some cases the numbness may involve the entire body, in which instance the patients have stated that it felt as though their bodies were "floating." Hypersalivation, profuse sweating, extreme weakness, precordial pain, headache, subnormal temperatures, decreased blood pressure, and a rapid, weak pulse usually appear early in the succession of symptoms.

Gastrointestinal symptoms of nausea, vomiting, diarrhea, and epigastric pain are sometimes present early in the disease, whereas in other cases they are totally lacking. Contradictory statements appear in the literature relative to pupillary changes, but these differences can probably be resolved on the basis of the time at which the examination is made. Apparently the pupils are constricted during the initial stage and later become dilated. As the disease progresses the eyes become fixed and the pupillary and corneal reflexes are lost.

Shortly after the development of the paresthesias, respiratory symptoms become a prominent part of the clinical picture. Respiratory distress, increased rate of respiration, movements of the nostrils, and diminution in depth of respiration are generally observed. Respiratory distress later becomes very pronounced, and the lips, extremities, and body become intensely cyanotic. Petechial hemorrhages involving extensive areas of the body, blistering, and subsequent desquamation have been reported. Severe hematemesis has also been known to occur. Muscular twitching, tremor,



Puffer, *Arothron meleagris*

and incoordination become progressively worse and finally terminate in an extensive muscular paralysis. The first areas to become paralyzed are usually the throat and larynx, resulting in aphonia, dysphagia, and later complete aphagia. The muscles of the extremities become paralyzed and the patient is unable to move.

As the end approaches, the eyes of the patient become fixed and glassy, and convulsions may occur. The victims may become comatose, but in most instances they retain consciousness and their mental faculties remain acute until shortly before death. Death results from a progressive ascending paralysis involving the respiratory muscles. On the basis of Japanese statistics, the case fatality rate is 61.5 percent. If death occurs, it generally takes place within the first 6 hours, or within 24 hours at the latest. The prognosis is said to be good if the patient survives for 24 hours.

Ciguatera

More than 300 species of marine fish have been incriminated as causative agents of this form of ichthyosarcotoxism. Fish most commonly involved are seabass or grouper, barracuda, snapper, parrotfish, wrasse, surgeonfish, and various other types of reef fishes.

The time of onset and symptomatology of ciguatera varies greatly according to the person, species of fish, toxicity, and amount and portion of fish ingested. Tingling about the lips, tongue, and throat followed by numbness may develop immediately or at any time within a period of 30 hours after the ingestion of the toxin. The usual time interval for the development of symptoms is from 1 to 6 hours. The initial symptoms in some instances consist of nausea, vomiting, metallic taste, dryness of the mouth, abdominal cramps, tenesmus, and diarrhea, followed by perioral tingling and numbness. The muscles of the mouth, cheeks, and jaws may become drawn and spastic with a feeling of numbness. Generalized symptoms of headache, anxiety, malaise, prostration, dizziness, pallor, cyanosis, insomnia, chilly sensations, fever, profuse sweating, rapid weak pulse, weight loss, myalgia, and joint aches are frequently present. Victims usually complain of a feeling of profound exhaustion and weakness. The feeling of weakness may become progressively worse until the patient is unable to walk. Muscle pains are generally described as a dull, heavy ache, or cramping sensation, but on occasion may be sharp, shooting, and affect particularly the arms and legs. Victims complain of their teeth feeling loose and painful in their sockets.

Visual disturbances, consisting of blurring, temporary blindness, photophobia, and scotoma, are common. Pupils are usually dilated and the reflexes diminished. Frequently reported are skin disorders, which are generally initiated by an intense generalized pruritus, followed by erythema, maculopapular eruptions, blisters, extensive areas of desquama-

tion—particularly of the hands and feet—and occasionally ulceration. There may also be a loss of hair and nails.

In severe intoxications, the neurotoxic components are especially pronounced. Paresthesias involve the extremities, and paradoxical sensory disturbances may be present in which the victim interprets cold as a "tingling, burning, dry-ice or electric-shock sensation," or hot objects may give a feeling of cold. In regard to the paradoxical sensory disturbance, the classical case usually cited is that in which an American naval officer who was poisoned by eating an amberjack was later observed subconsciously blowing on his ice cream, which was "burning" his tongue, in order to cool it. Ataxia and generalized motor incoordination become progressively worse. The reflexes are diminished and muscular paralyses develop. There may be clonic and tonic convulsions, muscular twitchings, tremors, dysphonia, dysphagia, coma, and death by respiratory paralysis. The limited morbidity statistics show a case fatality rate of about 7 percent. Death may occur within 10 minutes, but generally requires several days.

In those instances in which the victim survives, recovery is slow and convalescence may be very prolonged, with extreme weakness, sensory disturbances, and excessive weight loss being the last symptoms to disappear. When patients have survived severe intoxication, complete recovery has required a period of several years. Several workers have reported cases in which the symptoms persisted for as long as 25 years. Individuals who have been severely intoxicated have stated that during periods of stress, fatigue, exposure, or poor nutrition, there is a recurrence of the myalgia and joint aches similar to those suffered during the original acute period of the disease (*10*).

Gempylid Diarrhea

Some of the gempylids or escolars contain an oil within their flesh and bone marrow which has a pronounced purgative effect when ingested. Gempylid diarrhea (*27*) develops rapidly, and is said to be without cramping or pain. Since there are no other untoward effects, ingestion of the oil can hardly be considered as an intoxication in the usual sense of the word.

Treatment of Fish Poisoning

The treatment of fish poisoning is largely symptomatic. There are no specific antidotes, and an attack does not impart immunity. Gastric lavage and catharsis should be instituted at the earliest possible time. In many instances, 10 percent calcium gluconate given intravenously has given prompt relief, whereas in others it has been ineffective. Paraldehyde and ether inhalations have been reported to be useful in controlling the convulsions. Nikethamide or one of the other respiratory stimulants is advisable in cases of respiratory depression. In cases where excessive mucus production is a factor, aspiration and constant turning are essential. Atropine has been found to make the mucus more viscid and difficult to aspirate, and is not recommended. Oxygen by inhalation and intravenous administration of fluids supplemented with vitamins given parenterally are usually beneficial. If laryngeal spasm is present, intubation and tracheotomy may be necessary. In case of severe pain, opiates such as morphine, given in small divided doses, will probably be required. Cool showers have been found to be effective in relieving severe itching. Patients suffering from the paradoxical sensory disturbance should be given fluids slightly warm or at room temperature, as well as vitamin B complex supplements. Antihistaminic drugs will be found to be useful in the treatment of scombroid poisoning.

Prevention of Fish Poisoning

Scombroid fishes appear to be particularly susceptible to putrefaction and the development of toxic substances. Geiger has shown that the "histamine" content of the flesh of the Pacific mackerel, *Pneumatophorus diego*, increases from 0.9 mg. per 100 gm. of tissue to about 95 mg. per 100 gm. when kept at room temperature (20° to 25° C.) for about 10 hours (*28*, *33*). In warmer climates scombroids should be promptly refrigerated or eaten soon after capture. Toxic scombroid flesh cannot always be detected by appearance. The toxin

content may be very high with little or no evidence of putrefaction. Scombroid meat having a sharp or peppery taste should be discarded. If the whole fish is available the gills should be examined. Gills should be bright red and without evidence of off-odor. If there is the slightest evidence of putrefaction discard the fish.

If one eliminates scaleless fish from the diet there is no opportunity to come in contact with either tetraodontoids or moray eels, since both are without scales. In Japan, puffer fishes should be purchased from a first-class authorized restaurant having a licensed puffer cook. It is important that the individual preparing the food has a thorough knowledge regarding the fish and its toxicity. One can generally have this assurance by dealing only with the higher class restaurants. It is advisable to eliminate all of the visceral organs and skin from the diet regardless of the species. Although the testes are usually nontoxic, it must be kept in mind that this organ is frequently confused with the violently toxic ovaries, particularly during the season of the year in which the reproductive organs are in the dormant state.

Ordinary cooking does not inactivate the toxin. It may still be present in lethal quantities even when passed through a steam retort in the commercial canning process. The poison can be inactivated chemically by cooking the meat in a strong solution of sodium bicarbonate, ordinary baking soda, for a prolonged period of time. This latter technique also destroys the flavor of the fish and renders it useless for consumption.

Our knowledge regarding the toxicity of most species of tetraodontoid fishes, exclusive of Japanese forms, is exceedingly meager. We do know, however, that many of the tropical tetraodontoid species have produced violent death at one time or another. Unless a species is definitely known to be nontoxic, which is questionable even under the best of circumstances, all tropical forms should be eliminated from the diet.

If it is a question of survival—either eat the puffer or die—and the edibility of the fish is unknown, it is recommended that the fish be eviscerated promptly, and only the musculature be used. Moreover, the meat should be cut or torn into small bits, and soaked in water for a minimum of 3 or 4 hours. During this period the flesh should be kneaded while in the water, and the water changed at frequent intervals. The toxin is water-soluble and leaching will effectively remove it.

In dealing with potential ciguatera type of fishes it should be kept in mind that the most reliable methods of determining edibility involve the preparation of tissue extracts which are injected intraperitoneally into mice, or feeding samples of the viscera and flesh to cats, or dogs, and observing the animals for the development of toxic symptoms. Viscera, that is, liver and intestines of tropical marine fish should never be eaten. Also, the roe of most marine fish is potentially dangerous, and in some cases may produce rapid death. Fishes which are unusually large for their species should be discarded, particularly barracuda (*Sphyraena*), jacks (*Cranax*), and grouper (*Epinephelus*), during their reproductive season. If one is living under survival conditions and questionable fish must be eaten, it is advisable to cut the fish into thin fillets and soak them in several changes of water for at least 30 minutes. This will serve to leach out the poison which is readily water-soluble. Do not use the rinse water for cooking purposes. If a questionable species is cooked by boiling, the water should always be discarded. Again it should be pointed out that ordinary cooking procedures do not attenuate ichthyosarcotoxins.

Summary

Fish poisoning is a disease of antiquity. Fishes are believed to become poisonous as a result of their food habits—feeding on marine algae. There is no evidence that plankton or radioactive substances are a factor in the production of the poisons. Poisonous fishes are largely circumtropical in their distribution. Toxin content is greatest in puffers during their reproductive season of the year, but this is probably not true of most other fishes. The distribution of the toxin within the body of the fish is subject to considerable fluctuation, but if the fish is poisonous, some of the poison will be present in the viscera in about 90 percent of the cases. Poisonous fishes cannot be detected by their appearance.

Fish poisoning should not be confused with bacterial food poisoning, with which it has no etiological connections. The overall incidence of fish poisoning is not known. There are eight clinical types of ichthyosarcotoxism recognized at present. With the exceptions of gempylid diarrhea and scombroid poisoning, all other types of ichthyosarcotoxism are characterized by neurotoxic symptoms. The treatment of fish poisoning is symptomatic. Little is known about the chemical and pharmacological properties of fish poisons.

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"Model" Law to Cut Poison Deaths

The American Medical Association's Committee on Toxicology has formulated a "model" law for the precautionary labeling of hazardous substances in commercial, household, and industrial chemical products.

The labeling would apply to hazardous substances defined as toxic, irritating, sensitizing, corrosive, flammable, explosive, or radioactive under customary or reasonably anticipated conditions of handling or use.

"Model" text would require declaration of hazardous ingredients and warning statements on the label and in the literature accompanying chemical products.

Requirements of the model law are:

- Labeling of all chemical products containing hazardous substances that are not now regulated.
- Applications of the same labeling standards to chemicals for domestic use and for export. (This will refute the common complaint that inferior standards apply to foreign customers.)
- Prohibition of re-use of food and drug containers bearing their original labels.
- Identification and warnings for strongly sensitizing chemicals which cause allergic or inflammatory reactions in living tissue on contact.

A significant departure in the new law is the deletion of the word "poison." Reference standards, based on animal tests, provide a more consistent and reliable index of poisonous properties than did the former label, according to the committee, not only because there are variations in existing legal limits for poison but also because there is a lack of agreement among scientists on a definition of the term.

Inadequate labeling of potentially harmful chemicals, the committee reports, has been a major handicap to a successful attack on the problem of accidental poisoning. Moreover, lack of information about hazardous ingredients may complicate or delay treatment in emergency situations.

The committee's work in formulating a model law was endorsed by various organizations such as the National Drug Trade Conference, the American Public Health Association, the Cincinnati Academy of Medicine, and the Pennsylvania Pharmaceutical Association.