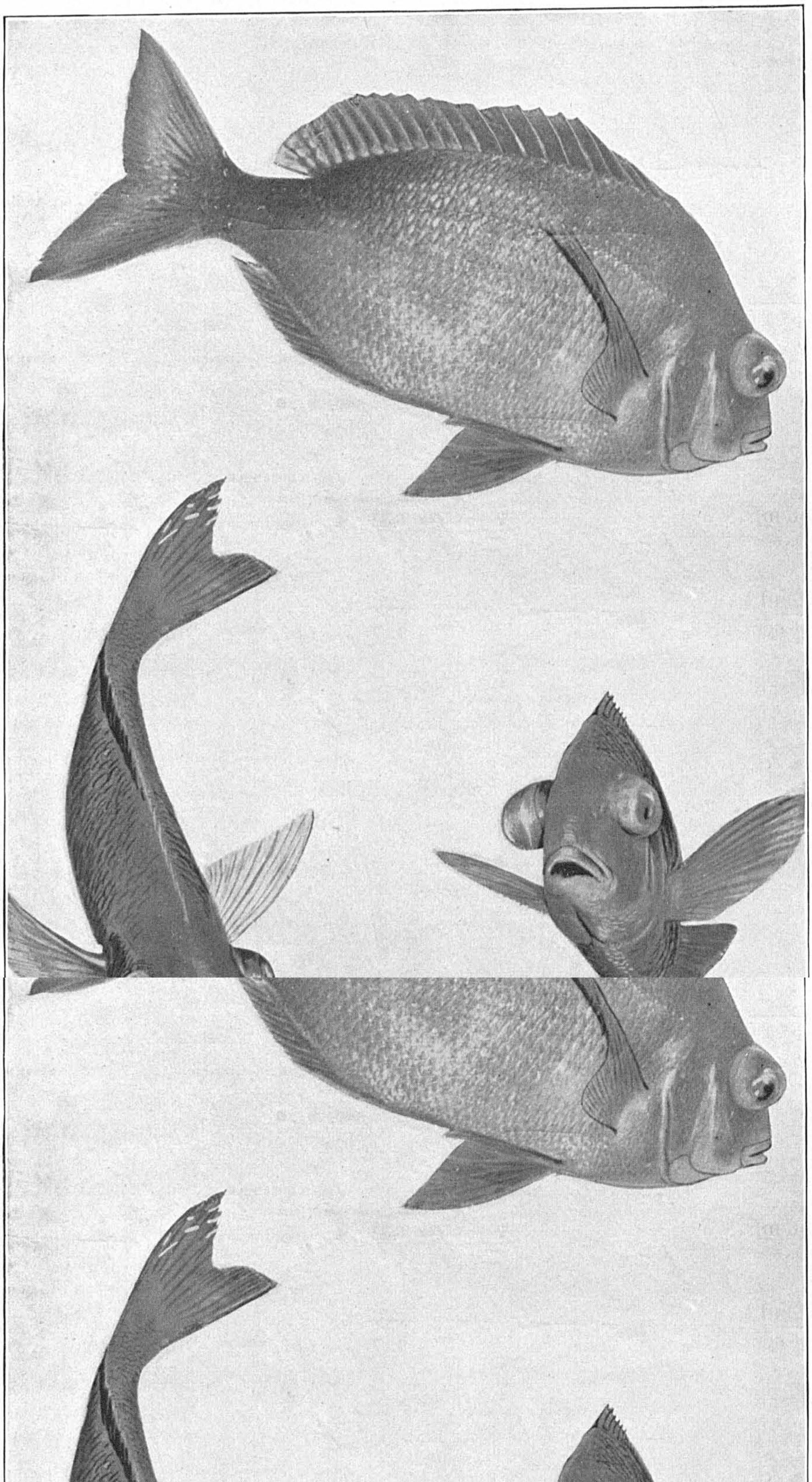

Contributions from the Biological Laboratory of the U. S. Fish Commission, Woods Hole, Massachusetts.

THE GAS-BUBBLE DISEASE OF FISH AND ITS CAUSE.

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For several years it has been noticed that many salt-water fish when kept in aquaria develop, after a longer or shorter period, a disease which is first manifested in the formation of vesicles of gas on the fins or other parts of the body. These vesicles gradually increase in size and number, and finally invade all superficial parts of the animal. The fins, one after another, become affected, and the vesicles frequently form in the eyeball, beneath the cornea, or in the loose connective tissue of the orbit, so that the eyes are forced from their sockets; less frequently the bubbles gather beneath the mucous membrane lining the mouth and gill-arches, or beneath the integument, particularly along the lateral line, so that the scales are raised from the surface. The presence of these vesicles often disturbs the equilibrium of the fish so that it swims about with its head elevated, or, more frequently, directed downward or tilted to one side.

The disease has been noticed only in fish kept in the aquaria. It has not been found in specimens taken from the sea, nor has it been observed in fish kept in "cars" in deep water, nor in those retained in "pounds," the water of which is from 6 to 12 feet in depth. The affected fish live a shorter or longer time after the vesicles begin to appear, some succumbing in a few hours, others resisting the disease for several weeks. Young "puffers" usually die in less than 24 hours after being placed in the aquaria, but several scup lived for weeks after their eyes had actually dropped from their sockets.

The following fish were affected by the disease during the spring and summer of 1898, and the list includes all the fish, with two exceptions, which were kept in the aquaria of the Fish Commission at Woods Hole, Mass., from March to September:

Common name.	Scientific name.	Bathymetric range.
		Fathoms.
Winter flounder	Pseudopleuronectes americanus	1 to 30
Summer flounder	Paralichthys dentatus	1 30
Hog-choker	Achirus fasciatus	1 30
Tautog	Tautoga onitis	1 30
Tomcod	Microgadus tomcod	1 15
Squirrel hake	Urophycis tenuis	1 300
Hake	Urophycis chuss	1 150
Sea-robin	Prionotus carolinus	5 80
Sea-robin	Prionotus strigatus	5 80
Puffer	Spheroides maculatus	2 10
Toad-fish	Opsanus tau	1 8
Stickleback	Gasterosteus bispinosus	1 5
Cunner	Tautogolabrus adspersus	1 35
Pipe-fish	Siphostoma fuscum	1 100
Sea bass	Centropristes striatus	1 40
Butter-fish	Rhombus triacanthus	3 10
Rudder-fish	Palinurichthys perciformis	1
Scup	Stenotomus chrysops	2 20

The following invertebrates showed signs of the disease: Squid, naked mollusks, scallops (*Pecten tenuicostata*), hydroids (*Parypha crocea*). Squid egg-sacs and some of the green algæ also developed internal bubbles apparently of the same nature.

Minnows (*Fundulus heteroclitus* and *Cyprinodon variegatus*) and skates (*Raja erinacea*) in no case were affected.

In most fish the disease manifests itself first on the fins, but in the scup it attacks the socket of the eye and gradually forces the eyeball from the orbit (plate 12). In the adult puffer the dorsal fin is first attacked; in the young puffer the base of the tail. In the pipe-fish the bubbles appear about the snout and later spread to other parts of the body. In young winter flounders (about 1 centimeter long) the body cavity frequently contains a large bubble.

The disease is not confined to the aquaria at Woods Hole. Mr. L. B. Spencer, who is in charge of the Battery Park Aquarium in New York City, writes me that he has noticed it there for several years.

From the appearance of the fish, from the rapid development of the disease in fish introduced into the aquaria, and from the nonappearance of the disease in fish outside of the aquaria, it at first seemed to me that the aquaria had become infected with some disease-producing organism, that the organism was a "gas producer," and that it spread rapidly through the tissues of the fish and produced the gas which collected as bubbles in various parts of the body. With this idea in mind the task of finding, isolating, and studying the organism was begun.

All of the tissues of the fish were searched thoroughly for foreign organisms; hundreds of cultures were made on the ordinary and also on special media, such as fish-bouillon, fish-gelatin, and fish-agar. The cultures were kept warm, cold, and at the temperature of the water in the aquaria; cover-glass "smears" were repeatedly examined; but all to no purpose. Not a sign of any pathogenic organism was found. The only pathological change noticed in the tissues was a remarkably emphysematous condition of epidermis, muscles, connective tissue, fat, etc., in the neighborhood of the vesicles. I became convinced that the disease could not be attributed to the invasion of micro-organisms.

Two phenomena suggested a new line of inquiry: (1) None of the small shallow-water fish developed bubbles; (2) the deep-sea fish when brought to the surface by line or dredge often show a protrusion of the eyes and an expansion of the tissues, comparable to the conditions under consideration. Could not the disease be the direct result of the reduction of pressure upon the tissues of the fish, a reduction which must occur when animals habituated to a life in deeper water are compelled to live in the shallow water of the aquaria?

To answer this question the following experiment was made: Several young scup were placed in water in a flask; the air was then exhausted from above the water, thus reducing the pressure. As soon as the pressure was at all diminished, the fish immediately sought the bottom of the flask, heads down, and made every effort to seek deeper water and thus regain their normal pressure. By their continued struggles they soon became exhausted and came to the surface. The atmospheric pressure was then restored and they became quiet and swam about naturally. The reduction of pressure was repeated three or four times at intervals of 30 minutes. After the experiment the eyes of the fish showed well-developed bubbles. The experiment was repeated on the following day, with the same fish, with the result that the bubbles enlarged until the eyes began to protrude.

Other experiments, indicated in the following table, were made:

Fish.	Total length of time subjected to reduction of atmospheric pressure.		Results.
	Hrs.	*Mins.*	
Scup (5 cm. long)	1	0	Bubbles in eyes.
Same fish	1	0	Bubbles enlarged; eyes protrude.
Scup (5 cm. long)	1	12	One eye shows bubbles and hemorrhage.
Sea-robin (6 cm. long)	3	49	Bubbles on pectoral fin and on "feelers."
Sea-robin (6 cm. long)	0	14	Bubbles on lower jaw.
Puffer	4	4	Bubbles on tail.
Minnow (*Cyprinodon variegatus*)	6	0	No change.

Normal fish were kept as controls in all cases and showed no change. Whenever scup were subjected to this reduction their eyes were affected first; bubbles formed and the eyes protruded in much less time, but in exactly the same way as in scup placed in the aquaria. In the young sea-robins bubbles appeared on the pectoral fins and on the finger-like appendages, while in the young puffers they appeared at the base of the tail. In all these cases the parts first affected were the same as those subject to the disease in the aquaria. The minnows unaffected in the aquaria were unaffected by the removal of the atmospheric pressure.

Now, the question naturally arises, will the increase in pressure improve the condition of the fish already suffering from the disease? Small scup taken from the aquarium, already showing protrusion of the eyes and bubbles on the head and fins, were subjected to a pressure equal to 16 feet of water. In 24 hours many of the bubbles had disappeared, and the eyes had returned to their normal condition. Under this treatment puffers recovered from a decidedly diseased condition in 24 hours. The data of these experiments may be tabulated as follows:

Fish.	Length of time subjected to pressure of 16 feet of water.	Results.
	Hours.	
Scup with bubbles on head	24	Bubbles disappeared.
Scup with protruded eye	24	Eye nearly normal.
Scup with bubbles on head and protruded eye	24	Entirely normal.
Puffers (3.5 cm. long). Bubbles at base of tail	24	Do.
Puffer (3.5 cm. long). Bubbles on dorsal fin	24	Normal.
Puffer (3.5 cm. long). Bubbles on eye and on fin	24	Eye nearly normal. Bubbles on fin disappeared.
Puffer (3.5 cm. long). Large bubble under right fin.	24	Normal.

Control animals checked the results of these experiments.

In both the reduced and the increased pressure experiments above tabulated young fish were used, but whenever adult animals were employed similar results were obtained.

The change in pressure which fish must undergo when transferred from the ocean to aquaria is not small in amount. We have only to consider that at the surface the pressure is about 14.7 pounds per square inch, and that for every foot in depth it increases at the rate of 0.445 pound per square inch. At a depth of 5½ fathoms the pressure is just double that at the surface, and at a depth of 300 fathoms it would amount to over 800 pounds.

Now, as may be seen by referring to the table of the depths from which the fish under consideration were taken, a reduction of pressure must take place when fish are confined in shallow aquaria. Their tissues relax under the reduction, and any gas present must expand. Since, according to the law of the expansion of gases, a volume of gas occupies just twice the space at the surface that it does at 5½ fathoms, a fish drawn from 300 fathoms to the surface must suffer an expansion of its contained gases to over 54 times their original volume.

Gas is present in all fish, in the alimentary tract, in the air bladder, and in the blood and other fluids of the body. As this gas expands and seeks an outlet the tissues are loosened and torn apart; the intestine and air bladder are greatly distended and perhaps ruptured, the circulation of the blood is impeded or stopped, and vesicles of gas form in various parts of the body.

These phenomena have been noticed for a long time in fish taken from great depths. Such fish are usually dead when they reach the surface, their eyes are protruded, their air bladder is ruptured, their intestines are everted from mouth or anus, and their scales are often lifted from the skin. The greater the depth from which they come the more pronounced are the changes. So great is the pressure to which some of the deep-sea fish are subjected (*Melamphaes beanii* from 2,949 fathoms sustains a pressure of 3.9 tons per square inch), that the structure of the bones, connective tissues, and muscles is peculiarly modified. Even in fish taken from 15 or 20 fathoms one may note the protrusion of the eyes, the eversion of the intestine, and the noise of the expanding gas working through the tissues.

We are thus led to the conclusion that the "gas-bubble disease" is caused by a reduction of pressure. Naturally, the disease has never been noticed except in the aquaria; normally shallow-water fish are never affected, and the greater the normal depth of a fish the more severely is it affected. Occasionally certain deep-water fish voluntarily seek the surface, but it is only when they can not return to deeper water that the changes brought about by the expansion of gases become evident.

It is interesting to compare these changes in fish with those observed in higher animals when subjected to alterations of pressure. Animals at the surface of the earth are subjected to an atmospheric pressure of about 14.7 pounds per square inch. When this pressure is diminished or increased, physiological changes result. Mountain climbers, at an elevation of 2 miles, suffer from increased rapidity of respiration, quickened pulse, painful, throbbing headache, flow of blood from the nose, eyes, and mouth, nausea, and vomiting. On the other hand, when the pressure is increased to from 50 to 60 pounds per square inch, as in caissons, workmen experience an annoying sensation about the tympanum, an irritation of the skin called the "puces," an alteration of the voice, deep, slow, and easy respiration, active digestion, an absence of thirst, and an increased secretion of saliva and urine. Return to normal pressure has the same effect as going into rarefied air. The "puces" is replaced by a chill, respiration becomes difficult, the pulse is rapid and hard, there are pains in the joints and muscles, and persistent cramps, paralysis, and coma are frequent; death may result in a few minutes. In such cases autopsies have shown congestion of the viscera, emphysematous spots on the lungs, and other indications that the blood contained bubbles of gas.*

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*I am indebted to Mr. Frederick T. Lewis for calling my attention to these facts. See "The physiological effects of compressed air," in the Boston Medical and Surgical Journal, October 6, 1898.

Experiments have shown that under compressed air the amount of oxygen absorbed by the blood increases with the pressure, according to the law of the absorption of gases. It may be the liberation of this oxygen and other gases under reduced pressure that causes death in the above cases.

In recording experiments upon the influence of high pressures upon various animals, Monsieur P. Regnard says: *

A fish without an air bladder, or one in which the air bladder has been emptied of gas, can be submitted to a pressure of 100 atmospheres, 1,470 pounds per square inch, without injury. When the air bladder is not emptied a very curious phenomenon is observed. Under pressure of several atmospheres the gas of the air bladder dissolves in the blood, and at the moment of decompression it suddenly becomes disengaged in the blood vessels, forming a foam which stops all circulation, and the animal dies; for the same reason fish drawn from the depths of the sea are dead when they reach the surface.

The bearing of this matter of pressure on geographical distribution should be considered. It might seem that there are no barriers to universal distribution in the sea save those indicated by the isotherms, but the isobars must indicate the location of effectual barriers for certain species.

Since even slight changes of pressure are detrimental, and each fish swimming in the sea tends to remain at about one level, it is reasonable to suppose that when a fish leaves this level the difference of pressure becomes uncomfortable and there is a tendency for it to return to its normal habitat. The air bladder is at present considered not an organ under the influence of the muscles of the fish, by means of which it alters its specific gravity, but rather a delicate gauge, which notifies the animal of changes of pressure above or below the normal.

Were a fish to pass too far beyond its normal depth, either up or down, the expansion or contraction of the gas of the air bladder would so change the specific gravity of the animal that its muscular activity would no longer be able to cope with the upward or downward tendency, and the fish would be carried to the surface or the bottom. Thus the very presence of an air bladder works the destruction of the fish. Cases are reported where deep-sea fish have come to the surface in this way, evidently having been carried too far from their normal level in struggles with their prey.

It is true that slight changes of level may be brought about by the secretion or absorption of gas by the walls of the air bladder. That the walls of the air bladder secrete and absorb gas has been shown by experiment. I have repeatedly emptied the air bladders of fish and found a small amount of gas secreted in 24 hours. But this process of secretion and absorption is relatively slow, and permanent changes of habitat could be brought about only very gradually; moreover, only very slight changes are possible for each species.

Thus we see that fish are surrounded by barriers of pressure, and that each species and probably each individual is constrained to remain at a certain level, and the restricted distribution of certain fish is thus explained.

* Recherches expérimentales sur l'influence des très hautes pressions sur les organismes vivants. Compt. Ren., XCVIII, March 21, 1884, p. 745.