Bartsch and Rehder (*2*) (described from a specimen which had also bored into a lead-sheathed power cable). Turner (*3*) has synonymized *M. funisicola* with *M. striata* and has stated (*4*) that she considers the specimens from the lead sheaths to be abnormal; probably because they had been boring in such hard material.

The greatest diameter of any of the holes in the section of attacked lead sheathing available to us (125 mm long; 180 mm circumference) is about 6 mm, at a depth of about 2 mm in the lead. These findings are comparable to those of Snoke and Richards (*5*) for an attack they reported. Five of the ten borings in this section had completely penetrated the sheathing. All these attacks were concentrated in a 215° arc of the sheath, but supposedly there were other sections which were completely attacked.

The present report constitutes the second verified record of *Martesia striata* boring into lead. A third record (*5*) is probably attributable to this species. All three attacks have occurred in Florida waters: Boca Ciega Bay, St. Petersburg; Ortega River, Jacksonville (*5*); and Lake Worth, West Palm Beach (*2*). All three attacks were upon the lead sheathing of power cables, and, at least in the Boca Ciega Bay and Ortega River attacks, occurred in the same type of cable (after the outer steel wrapping had rusted through). The type of cable sheathing attacked is presumably in service in *Martesia* inhabited waters in other areas, and it is surprising that there are no other records of attacks on these cables. The conditions in Boca Ciega Bay are turbid ones, and there is considerable silting in the causeway area (Florida Power Corporation diver's report). The salinity is usually moderately high (estimated at 25 to 32 per mil, based on determinations made at various points in the bay) although during rainy seasons it may be much lower (1959 had the highest recorded rainfall on record for St. Petersburg, although the rainfall for August of that year, 9.54 inches, was close to the 45 year average, 9.16 inches). Lake Worth conditions probably approximate those in Boca Ciega Bay. The Ortega River attack probably occurred under turbid conditions and at a low degree of salinity (*7*).

In the event of future attacks, certain data should be recorded in order to facilitate the determination of the specific conditions under which attacks occur. The following information would be most useful: locality, general locality description, date, depth, salinity, turbidity, temperature, weather conditions, nature of bottom, position on bottom of attacked section, surface nature of lead (oxidized, clean, and so on), thickness of lead, position of individual penetrations, density of attack, and whether other substances were penetrated before the lead was reached.

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References and Notes

1. The identification was kindly verified by Dr. Ruth D. Turner.
2. P. Bartsch and H. A. Rehder, *Smithsonian Inst. Publs. Misc. Collections No. 104* (1945).
3. R. D. Turner, *Johnsonia* 3, No. 34, 65 (1955).
4. R. D. Turner, personal communication.
5. L. R. Snoke and A. P. Richards, *Science* **124**, 443 (1956).
6. This report is contribution No. 37 from the Marine Laboratory, Florida State Board of Conservation.
7. Personal observations (V.G.S.).

23 December 1959

Physiological Measurements on a Live Whale

Abstract. Temperature, respiration, and electrocardiographic measurements were made on a stranded 45-ft. finback whale. This proved to be a practical means of getting physiological information on the large cetaceans.

The stranding of a 45 foot finback whale (*Balaenopterus physalus*) on a Provincetown, Mass., beach provided a fortuitous opportunity to make multiple temperature, respiration, and electrocardiographic measurements. We know of no other such measurements on large, live cetaceans. The whale died after 36 hours.

The whale was entirely exposed at low tide. It breathed at a steady rate of about once every 20 seconds. The animal lay mostly on its left side and appeared to breathe primarily with its right lung: only the right blow hole opened. It was clear that only a partial inspiration was possible due to the overlying bulk of the animal. At high tide the whale was almost completely afloat. It was then pressed against the beach by a slow swimming motion of its tail. Its breathing while partially submerged was slower and more regular, with intervals ranging from 90 to 120 seconds. When we have observed the same species at sea, it generally breathed six to eight times at intervals of half a minute and then sounded for 5 to 8 minutes.

Samples of the expired air were taken with a syringe held 6 to 8 inches inside the blow hole during expiration. Gas analysis of three samples showed that they contained 1.42, 1.50, and 1.66 percent CO_2 and 19.22, 19.30, and 19.22 percent O_2, respectively (RQ of 0.8 to 0.9). The gas samples were taken near the end of expiration (which lasted about 2 seconds), so they would more nearly represent alveolar air. The apparently poor utilization of the inspired air coupled with the rapid, shallow breathing indicate pulmonary insufficiency and suggest that anoxia contributed to the whale's death.

Temperature distribution in the blubber was determined with a thermistor-tipped hypodermic needle. The skin temperature during low tide was uniformly cold (10° to 14°C), over all of the animal except the dorsal fin. This was warm to the touch and had a temperature of 23°C on the thin trailing edge. The sea temperature was 6°C, and the air varied between 6° and 10°C. The temperature in the blubber increased linearly with distance from the surface, reaching values of 27° to 31°C at the point of maximum penetration of the needle (15 cm).

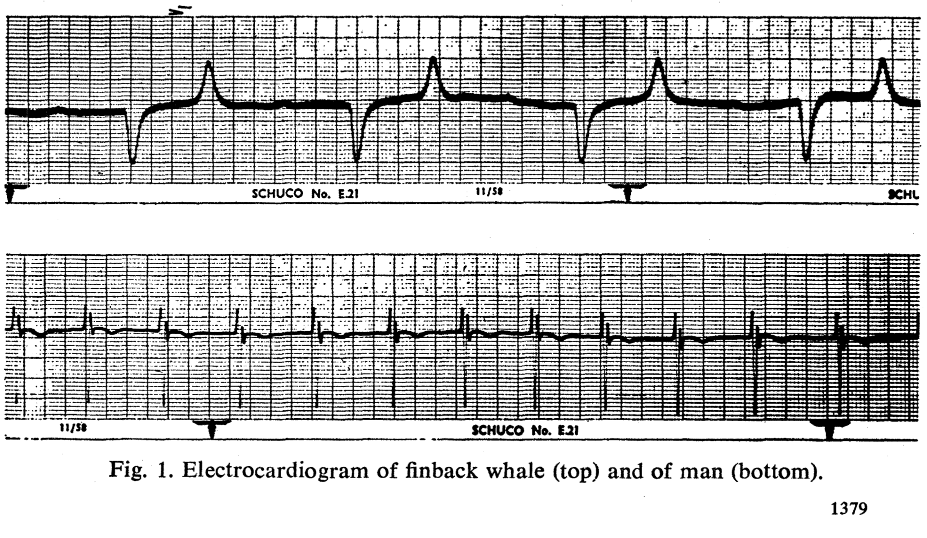

Fig. 1. Electrocardiogram of finback whale (top) and of man (bottom).

These slightly different gradients are probably due to variations in blubber thickness. We could not be certain that the thermistor probe penetrated completely through the blubber layer; therefore we do not know whether our maximum readings represent a true deep body temperature. A rectal temperature of 33°C was obtained with a flexible tube-mounted thermistor inserted 18 inches. Readings of 30° to 33°C were obtained under the tongue with the same instrument. From previous experience (*1*), we feel that the whale was 1° or 2° cooler than would be expected. The general mechanism of thermal regulation appears to be the same as that found in live porpoises which are approximately 1/100 as large as this whale (*2*). The blubber is an insulating layer, and the preferred pathway of heat disposal is through the uninsulated extremities.

Electrocardiograms were taken on an instrument which one of us (A.S.) uses in his private medical practice. Because the whale was completely out of the water we were able to place leads in most of the positions analogous to those used on humans. Figure 1 shows a precordial lead record from the whale heart as contrasted to a normal human record. The pulse rate was about 27 beats per minute, compared with 70 in man and more than 600 in the smallest mammals. However, it was still nearly twice that recorded on a much smaller white whale (*3*). Since the beats closely follow one another we presume that this represents tachycardia and that a more normal rate would be 8 to 10. The high rate probably reflected the deteriorating condition of the animal. The time scale for the events in a single beat are much longer than any known previously. The PR interval, for instance, was about 0.68 second as compared to half this in the white whale and a maximum of 0.20 second in humans.

This cursory report is given to show that it is possible to obtain physiological information from a beached cetacean if one has the instruments ready to take advantage of the situation. A live whale in the water is an imposing experimental subject, but grounded animals are more manageable and fairly frequent. Unfortunately most people consider the great mass of potentially putrifying meat more of a potential health hazard than a scientific opportunity. We would appreciate immediate notification of similar live strandings anywhere in New England.

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22 January 1960

Inhibition of δ-Aminolevulinic Acid Dehydrase by δ-Oximinolevulinic Acid

Abstract. δ-Oximinolevulinic acid competitively inhibits δ-aminolevulinic acid dehydrase at low concentrations.

The enzyme δ-aminolevulinic acid (ALA) dehydrase involved in the conversion of ALA to porphobilinogen has been studied by Gibson, Neuberger, and Scott (*1*) and others (*2*). In studying analogues of ALA it was found that δ-oximinolevulinic acid acted as a competitive inhibitor of ALA dehydrase, producing significant inhibition at low concentrations.

δ-Aminolevulinic acid and β-ketoadipic acid were purchased from the Nutritional Biochemicals Corporation. δ-Chlorolevulinic acid was synthesized by the method of Neuberger and Scott (*3*). δ-Oximinolevulinic acid was synthesized by the method of Neuberger, Scott, and Shuster (*4*). δ-Acetamidolevulinic acid was provided by Anthony Schrecker of the National Cancer Institute. 2-Amino-4-thiazolepropionic acid was prepared as described by Neuberger and Scott (*3*).

The homogenates were prepared from CAF$_1$ mouse livers as described by Gibson, Neuberger, and Scott (*1*). Each tube contained 1 ml of liver

Table 1. Inhibition of δ-aminolevulinic acid dehydrase by various compounds.

Concentration (*M*) Compound added	Concentration (*M*) ALA added	Volume of compound and ALA added (ml)	Ratio of compound to ALA	Inhibition (%)
		δ-Chlorolevulinic acid		
7.5×10^{-2}	7.5×10^{-3}	1.32	10:1	100
7.5×10^{-3}	7.5×10^{-3}	1.32	1:1	96
7.5×10^{-4}	7.5×10^{-3}	1.32	0.1:1	0
7.5×10^{-5}	7.5×10^{-3}	1.32	0.01:1	0
		δ-Acetamidolevulinic acid		
7.5×10^{-2}	7.5×10^{-3}	1.32	10:1	37
7.5×10^{-3}	7.5×10^{-3}	1.32	1:1	0
7.5×10^{-4}	7.5×10^{-3}	1.32	0.1:1	0
7.5×10^{-5}	7.5×10^{-3}	1.32	0.01:1	0
		β-Ketoadipic acid		
7.5×10^{-2}	7.5×10^{-3}	1.32	10:1	100
7.5×10^{-3}	7.5×10^{-3}	1.32	1:1	79
7.5×10^{-4}	7.5×10^{-3}	1.32	0.1:1	11
7.5×10^{-5}	7.5×10^{-3}	1.32	0.01:1	0
		2-Amino-4-thiazolepropionic acid		
7.5×10^{-2}	7.5×10^{-3}	1.32	10:1	92
7.5×10^{-3}	7.5×10^{-3}	1.32	1:1	8
7.5×10^{-4}	7.5×10^{-3}	1.32	0.1:1	0
7.5×10^{-5}	7.5×10^{-3}	1.32	0.01:1	0
		δ-Oximinolevulinic acid		
7.5×10^{-2}	7.5×10^{-3}	1.32	10:1	100
7.5×10^{-3}	7.5×10^{-3}	1.32	1:1	96
7.5×10^{-4}	7.5×10^{-3}	1.32	0.1:1	75
7.5×10^{-5}	7.5×10^{-3}	1.32	0.01:1	27
7.5×10^{-1}	5×10^{-2}	.33	10:1	95
7.5×10^{-2}	5×10^{-2}	.33	1:1	92
7.5×10^{-3}	5×10^{-2}	.33	0.1:1	72
7.5×10^{-4}	5×10^{-2}	.33	0.01:1	26

Table 2. Inhibition of δ-aminolevulinic acid dehydrase by δ-oximinolevulinic acid.

Tube	Concentration (*M*) δ-Oximinolevulinic acid added	Concentration (*M*) ALA added	Volume of compound and ALA added (ml)	Ratio of compound to ALA	Inhibition (%)
1	7.5×10^{-2}	7.5×10^{-3}	1.32	10:1	94
2	7.5×10^{-3}	7.5×10^{-3}	1.32	1:1	90
3	7.5×10^{-4}	7.5×10^{-3}	1.32	0.1:1	66
4	7.5×10^{-5}	7.5×10^{-3}	1.32	0.01:1	29
5	7.5×10^{-4}	9×10^{-2}	1.32	1:120	27
6	7.5×10^{-4}	6×10^{-2}	1.32	1:80	38
7	7.5×10^{-4}	3×10^{-2}	1.32	1:40	47
8	7.5×10^{-4}	7.5×10^{-3}	1.32	1:10	58