Electrocardiogram of the Diving Seal

By Herschel V. Murdaugh, Jr., M.D., James C. Seabury, B.S., M.D., and William L. Mitchell, B.S.

The diving mechanism in the seal is believed to be comprised of two major changes in the circulation which afford an efficient means of maintaining an adequate oxygen supply to the brain for a prolonged period of inability to breathe.

One circulatory response to diving is the arterial constrictor response which prevents the flow of blood through muscle beds during the dive. Therefore, the muscle mass does not remove oxygen from the circulating blood, and a high blood oxygen saturation is maintained for a longer period of time.

Accompanying this decrease in perfused vascular bed is the other circulatory alteration, bradycardia with decrease in cardiac output. This bradycardia appears to be reflex in origin, inasmuch as it may begin within one heart beat of submersion, and it is so consistent that an electrocardiogram may be used to clock the periods of diving.

This type of reflex bradycardia with diving was first reported by Bert in 1870, when he found that the duck developed bradycardia when its head was submerged in water. Since that time, the diving-reflex bradycardia has been reported in the penguin, beaver, muskrat, porpoise, seal, rabbit, Florida manatee, and water snakes. In 1899, Richet demonstrated that the administration of atropine to the duck would prevent the duck from being able to dive. Irving et al. reported that atropine prevented the reflex bradycardia in the porpoise. Forster and Nyboer found that the atropinized rabbit did not have reflex brady-
Subcutaneous needle electrodes were inserted through procainized, shaved areas of skin dorsal to each foreflipper. The subcutaneous needle electrodes were attached to 50-foot Teflon insulated leads. When inserted subcutaneously, they were pushed in until approximately 1 cm. of insulation was under the skin. After insertion of the electrodes the incision was allowed to close by clotting, and a water-repellent spray was applied to the site of each electrode insertion.

The seal was then placed in a small, nonrestrictive, canvas harness to which was attached a 45-foot rope, and the copper tip of the ground lead for the electrocardiogram was either placed in the bay or pushed into the ground.

The seals were studied in an aquarium and in the bay. An aquarium with plastic sides was constructed in which the seal seemed content to swim and dive freely, and electrocardiograms were performed in this aquarium during normal voluntary submersion by the seal, during feeding by the seal, and during struggle induced by pushing the seal's head under water. Additional electrocardiograms were taken at dockside as the seal swam in the bay, with escape being prevented by the rope leash on the harness. All electrocardiograms were taken with a direct-writer electrocardiogram machine.

Atropine was given by intramuscular route.

**Results**

During repeated voluntary diving, there occurred a reflex bradycardia with the heart rate decreasing to as slow as seven per minute. During voluntary diving, whether in the bay or in the live car, the bradycardia was accompanied by some increase in the amplitude of the T wave, but inversion of P or T waves did not occur. On occasions there was noted a block of conduction with the appearance of a P wave which was not followed by a QRS complex, but the QRS complex remained unchanged during the bradycardia. The bradycardia persisted when the seal ate while submerged.

The reflex bradycardia did not appear "in anticipation" of diving, but the heart rate often returned to normal when the seal was approaching the surface of the bay and before it had its nostrils above water. If the seal was approaching the surface and decided to dive again before surfacing, the bradycardia returned.

When the seal was forcibly submerged by pushing its head under water, it struggled violently and a less pronounced bradycardia occurred which was accompanied by ventricular extrasystoles, runs of ventricular tachycardia, and periods of atrial fibrillation. If the seal, while swimming in the bay, happened to entangle its rope leash on one of...
the mooring chains of the dock, it tried to loosen itself from the harness and thus struggled. This struggling was not associated with abnormalities of P wave, T wave, nor the development of arrhythmias.

Atropine, administered intramuscularly in a dose of 0.04 mg. per pound body weight, completely prevented the development of reflex bradycardia to diving within 20 to 30 minutes after administration (fig. 1). One atropinized seal drowned after submersion without bradycardia for less than three minutes.

In figure 2, the P-R interval is plotted against heart rate, and it is noted that the P-R interval accompanying bradycardia during struggle is less prolonged than the P-R interval accompanying a normal dive.

**Discussion**

There appears a distinct difference in electrocardiograms obtained in the seal which dives voluntarily or without struggle and the seal which struggles against forceful submersion under water or forceful prevention of its ability to breathe. This difference indicates that the electrocardiograms obtained during struggling are not normal physiological electrocardiograms. Teleologically, it does not seem practical that the seal should develop arrhythmias during the course of diving in normal life.

The development of heart block during normal diving with an occasional P wave not followed by a QRS complex could be explained by a pronounced vagal effect. The prevention of reflex bradycardia by atropine supports the thesis that the normal reflex bradycardia is mediated via vagal stimulus. The fact that one atropinized seal drowned after only three minutes of submersion without bradycardia suggests that the atropine also blocked the arterial constrictor reflex of the seal, since a normal seal can remain under water 15 to 20 minutes.

The inversion of T waves and the shorter P-R interval during bradycardia with struggling would imply an added sympathetic-like effect in addition to the vagal effect. The finding that struggle occurring upon encounter of a barrier during a normal dive in the bay was not accompanied by inversion of P
waves or T waves would imply that the abnormal electrocardiogram during forced struggle may be more closely related to a fright phenomenon than to physical exertion.

It is of interest that the seal will have bradycardia during struggle, and that such bradycardia serves to make the interpretation of the electrocardiograms of the seal difficult. King et al. recorded the electrocardiogram of a whale with a harpoon electrode. If the whale, as an aquatic mammal, also has bradycardia in response to a stressful situation, the cardiac output of the whale under normal circumstances may be even more striking than can be estimated from the reported rate and size of the whale heart.

This diving-reflex bradycardia assumes greater significance when consideration is given to the broad phylogenetic range it includes. The fact that bradycardia occurs in reptiles, birds, aquatic mammals, and a terrestrial mammal demonstrates that it is a commonly evolved reflex in the animal kingdom. Also, because atropine can block this reflex in these species, bradycardia appears to have a similar mechanism in these varieties of animals.

Summary

During the normal dive, the seal has a reflex bradycardia that is not accompanied by inversion of P waves or T waves or by atrial or ventricular arrhythmias, although the seal does have an occasional block of conduction across the A-V node during normal diving. The reflex bradycardia during the dive appears to be mediated through vagal stimulus.

The electrocardiograms of the seal during voluntary diving and during forced submersion with struggle are different.

References


Electrocardiogram of the Diving Seal
HERSCHEL V. MURDAUGH, JR., JAMES C. SEABURY and WILLIAM L. MITCHELL

doi: 10.1161/01.RES.9.2.358

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1961 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/9/2/358

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation Research can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation Research is online at:
http://circres.ahajournals.org/subscriptions/