SEASONAL TEMPERATURE INFLUENCE ON VAGAL CONTROL OF DIVING BRADYCARDIA IN THE FROG (RANA PIPIENS)

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INTRODUCTION

Basically similar physiological adjustments occur in diving amniote vertebrates and in fish removed from water (Andersen, 1966; and additional reviews by Irving, 1939, 1964; Scholander, 1940, 1961-62, 1963, 1964; and Andersen, 1964). In these vertebrates there is a pronounced, vagally induced bradycardia (slow heart rate) which generally is achieved in a few seconds or a few minutes. Jones & Shelton (1964) have reported, however, that diving bradycardia in the frog (Rana temporaria) required 15-30 min. to become fully established and was to a great extent independent of the vagus nerve. Other amphibians (R. pipiens, R. esculenta, Xenopus muelleri, X. laevis, Bufo bufo) display a similar slowly developed, non-reflex, diving bradycardia (Shelton & Jones, 1965; Jones, 1966, 1967).

This difference between amphibians and other vertebrates is puzzling. There is also additional conflicting evidence. Leivestad (1960) reported that the pattern of diving bradycardia in the toad (Bufo bufo) suggested reflex mechanisms. For the first 20 min. of a dive the heart rate alternated in quick succession between bradycardia and tachycardia, although averaging over time revealed a gradual slowing; metabolism also gradually decreased.

It is our intent, therefore, to analyse diving bradycardia in the frog by taking into account three factors in an attempt to clarify the nature of the controlling mechanisms. First, amphibians do not necessarily enter an asphyxiant environment when diving; cutaneous respiration may eliminate the need for drastic and rapid physiological adjustments. This, however, depends on the second factor, temperature, which has a twofold effect; (1) metabolism increases with increasing temperature ($Q_{10}$) so that cutaneous respiration ($Q_{10}$) may be unable to meet oxygen demands, and (2) temperature directly influences vagal reflexes. Young (1959), using vagus-heart preparations of R. pipiens, reported that with decreasing temperature cardiac inhibition by the vagus was partially blocked at 14° C.; complete block occurred at 10° C. When temperature was again increased, a partial response did not return until a temperature of 14-16° C. had been reached, while complete vagal inhibition of the heart was not re-established below about 18° C. Jones and Shelton (1964) conducted their experiments between 12 and 18° C., and their records are primarily at the lower temperatures; in light of

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Young's results, at least a partial vagal block is thus suspected. Leivestad's (1960) experiments were conducted at 19-21°C.

The third factor considered is the influence of restraint. This point needs to be emphasized. It is becoming more apparent that variability of response during dives, even in the same animal, may be influenced by experimental procedure, restraint, age, training, temperament, fright, previous diving, and anticipation of diving. In frogs, resistance to the restraining apparatus or discomfort during experiments could reflect on both metabolism and heart rate and thus increase the need for physiological adjustments when diving. Since previous experiments (Leivestad, 1960; Jones & Shelton, 1964) involved restrained animals, this factor, along with temperature and asphyxia, is considered in the following analysis of the control of diving bradycardia in amphibia.

METHODS AND MATERIALS

Female frogs (Rana pipsiens) obtained from the Lemberger Company, Oshkosh, Wisconsin, and weighing between 30 and 100 g. were used in these experiments. Prior to use the animals were maintained for at least 2 weeks at either 4-6°C or 18-20°C in frequently changed or continuously running water. They were not fed, and experiments were not conducted on animals kept longer than 6 or 7 weeks.

For diving, frogs were fastened to a frog board with clips securing the appendages and with rubber straps over the back. Body and leg movements were thus restricted, but circulation, breathing, and head movements were not greatly hindered. The frogs were left in position on the water surface for an hour before initial records were taken. The experimental diving aquarium, filled with well-aerated water, was placed in a controlled-temperature water bath.

Electrocardiograms (e.c.g.) were obtained by placing a stainless steel or silver electrode on the ventral surface of the body or by threading the electrode through the mid-ventral line of the skin so that the tip rested on the sternum. A second, reference, electrode was placed in the surrounding water. During vagal stimulation two recording electrodes were placed close together near the sternum, and the electrocardiogram (e.c.g.) was recorded differentially. The e.c.g. signal was relayed via a pre-amplifier to an oscilloscope and a chart recorder. Average heart rate was determined for each minute by counting over 30 sec. intervals.

For vagotomy experiments and for experiments on the effect of temperature on vagal inhibition of the heart, the nerve was exposed surgically dorsal to the angle of the jaw. The methods have been described in detail elsewhere (Lund, 1967). Although healing was usually complete in 1 week following vagotomy, 2 weeks or more were allowed before diving, and post-mortem dissections to verify vagotomy (successful in all cases) were conducted. Squibb D-turbocurarine chloride (6-0-7.5 mg./kg. body weight) was used during surgery and when stimulating the vagus nerve. Injections were given through the dorsal lymph sac.

Stimulation was applied to the vagus nerve with a calibrated battery inductorium via silver stimulating electrodes. Before stimulation the nerve was lifted out of the animal and afterwards returned to the moist environment of the surrounding tissues. Addition of Ringer's solution was not necessary, and the nerve remained functional for several hours. Stimulation of nerves other than the vagal branch containing the
cardiac fibres, or of surrounding tissues, had no noticeable inhibitory influence on the heart. The stimulus voltage, usually 4-6 V, was adjusted to insure maximal response at room temperature. The vagus was usually stimulated only once at each temperature except at the lower and higher temperatures when additional stimulation was applied to determine response variability. Some frogs were allowed to recover, were kept for several weeks, and appeared normal.

In the vagal stimulation experiments the difference between internal body temperature and surface temperature was maintained within ± 0.5°C. The frog and apparatus were placed in an insulated aquarium, and body temperature was raised or lowered, usually twice for each animal, by approximately 1°C every 5-10 min.

Fig. 1. Heart rates of frogs diving in September at various temperatures. (A, C, D) acclimated 18-20°C, (B) acclimated 4-6°C. Diving temperatures and body weights (A) 10°C, 56 g., (B) 10°C, 54 g., (C) 15°C, 42 g., (D) 20°C, 46 g. Note in this and other figures points represent heart rates determined (when possible) for each minute by averaging over a 30 sec. interval (× ± 15 sec.). Downward arrows indicate immersion; upward arrows, emersion. Points not connected by lines designate occurrence of at least one period of struggling. Check marks indicate first occurrence of sinus arrhythmia.
Fig. 2. Heart rates of frogs diving in July at low temperatures. Acclimated 4–6°C. Diving temperatures and body weights (A) 10°C, 35 g., (B) 10°C, 39 g., (C) 15°C, 38 g., (D) 15°C, 32 g.

Fig. 3. Heart rate during consecutive diving in July showing influence of diving duration on recovery. Acclimated 4–6°C; diving temperature 20°C; body weight 35 g.
Vagal control of diving bradycardia in the frog

RESULTS

(a) Diving bradycardia in restrained frogs

Bradycardia in restrained, diving frogs was achieved in two forms. Upon immersion the heart rate decreased either (1) rapidly (hereafter rapid bradycardia), indicating a reflex mechanism similar to that of other vertebrates, or (2) slowly (hereafter slow bradycardia), indicating a non-reflex mechanism. Frequently, both mechanisms appeared to operate in the attainment of the lower levels of heart rate, and thus the classification as to slow or rapid bradycardia is somewhat arbitrary. The mechanism which predominated was dependent on temperature and season. No influence of previous temperature acclimation was apparent. These observations are illustrated in the diving records of September and July (Figs. 1-4). A minimum of four experiments was conducted for each set of conditions.

In September rapid bradycardia was always observed during dives at water temperatures of 10°C and above (Figs. 1 and 4). In contrast, diving bradycardia in July experiments was attained primarily slowly at 10°C, either rapidly or slowly or in combination at 15°C, and primarily rapidly at higher temperatures (Figs. 2 and 3).

A gradual slowing of the heart rate usually began with immersion; the onset of rapid bradycardia was not immediate, but in September usually occurred within the first minute. In July, however, this onset was frequently further delayed. After their onset, low heart rates were attained frequently in less than 1 min. although 2 or 3 min. were sometimes required, particularly if struggling occurred. Slowly
attained bradycardia required 10–20 min. to achieve the final rate. The degree of heart-rate reduction was greatest when rapid bradycardia occurred (Figs. 2C and D). The approximate heart-rate ranges observed during various periods in these experiments are presented in Tables 1 and 2.

The onset of rapid bradycardia was always accompanied by sinus arrhythmia with beats frequently occurring in doublets. An increased amplitude of the T-wave frequently occurred as well. (The first occurrence of sinus arrhythmia in the diving records presented has been illustrated by a check mark and is thus also an index of the onset of rapid bradycardia.) Sinus arrhythmia was not observed during slow bradycardia but frequently did occur late in a dive when lower heart rates were already approached. (Note that in Fig. 2A as compared to Fig. 2B an accelerated heart-rate decline was associated with the late occurrence of sinus arrhythmia during slow bradycardia.) These e.c.g. irregularities usually continued throughout a dive, but frequently a tendency to resume regularity occurred once bradycardia was firmly established, and in such cases heart rates tended to increase slightly. These changes in the e.c.g. are considered indicative of vagal activity.

Table 1. Approximate heart-rate ranges of frogs observed during diving in September

<table>
<thead>
<tr>
<th>Water temp. (°C)</th>
<th>Pre-dive</th>
<th>Full bradycardia</th>
<th>1st min. recovery</th>
<th>15 min. recovery</th>
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| 20               | 55–70    | 10–20            | 40–45            | 50–60

Bradycardia occasionally tended to be unstable with heart rates somewhat higher than otherwise observed.

Table 2. Approximate heart-rate ranges of frogs observed during diving in July

<table>
<thead>
<tr>
<th>Water temp. (°C)</th>
<th>Pre-dive</th>
<th>Full bradycardia</th>
<th>1st min recovery</th>
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<td>15</td>
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</table>
| 20               | 55–65    | 10–20            | 40–45            | 50–60

Rapid bradycardia resulted in lower heart rates than those attained with slow bradycardia.

Upon emersion, regularity of the heart beat was immediately restored. The increase in heart rate, after long dives, occurred in two phases: (1) a rapid increase took place during the first minute followed by (2) a more gradual increase over the next several minutes. Heart-rate recovery to pre-dive levels was complete within 15 min. at 20° C. but was usually incomplete at lower temperatures; over-shoots were frequent at higher temperatures. This apparent variability presumably resulted from an initial difference between body temperature and water temperature. Pre-dive cloacal temperature was similar to water temperature at 20° C., was higher by three or four degrees at water temperatures of 10 and 15° C., and was lower at water temperatures above
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20°C. Upon immersion, cloacal temperature soon approximated to water temperature and differed only slightly for some time after emersion. Another factor found to influence heart-rate recovery was the duration of the dive (Fig. 3). Generally, the shorter the dive the more complete and rapid was the recovery; the order of repeated dives is unimportant. Complete recovery and occasionally temporary tachycardia occurred during the first minute after short dives at 20°C. Leivestad (1960) showed that metabolism in the toad is slowly reduced during a dive to 20% of the pre-dive levels. Thus, the gradual increase in heart-rate after the first minute of recovery in these experiments is perhaps correlated with an increase in metabolism to pre-dive levels. Lastly, at all temperatures frogs were lethargic after long dives, and lower heart rates could be a reflection of their condition.

Diving experiments at temperatures above 20°C. (Fig. 4) indicated that an oxygen debt was incurred and definite signs of stress were evident. Upon emersion, if frogs were still alert, tachycardia with increased heart-beat amplitude was frequently observed, and pulmonary ventilation was more rapid than before diving. During dives, bradycardia was often unstable and tended to break down, at which time the heart rate would increase, and the heart beat would become regular. These observations were more pronounced in cold-acclimated frogs than with frogs acclimated at 18–20°C. Below 20°C. frogs can probably sustain long dives without incurring an oxygen debt, but at 20°C. signs of stress were sometimes evident. In one case, however, bradycardia was successfully maintained during a 30 min. dive at 30°C., and such records were not uncommon at 25°C. At high temperatures, and after a more or less complete breakdown of diving bradycardia, a complete loss of control of the heart rate was frequently observed; that is, the heart rate would alternate between short periods of tachycardia and bradycardia. In such cases, a rapid and immediate increase in heart rate was not observed upon emersion, the buccal cavity contained water, and in two observations the lungs were deflated and also filled with water. These frogs were extremely lethargic but recovered when returned to a lower temperature.

In all experiments struggling by the frogs usually accelerated heart rates and influenced bradycardia. When struggling occurred before and after dives, several minutes were required before heart rates were reduced to previous levels. During a dive accelerated heart rates due to struggling were more temporary as bradycardia was being established and were brief or negligible once lower heart rates were reached. The effects were of longer duration when bradycardia was being established slowly rather than rapidly. The onset of rapid bradycardia was always preceded by struggling. If struggling occurred following onset, however, rapid bradycardia was usually interrupted, and the heart beats temporarily became regular; the time required to achieve minimum rates was thus prolonged.

Expulsion of air following immersion was not as highly correlated with the onset of rapid bradycardia as was struggling. This indicates that rapid bradycardia is not induced by asphyxia or by lung deflation but rather by a stress resulting from restraint.

(b) Vagal influence on diving bradycardia

The reflex nature and e.c.g. irregularities of diving bradycardia in frogs indicate vagal influences similar to those in other vertebrates. The breakdown of bradycardia
at high temperatures indicates partial or complete elimination of governing neural mechanisms. Therefore, in order to verify vagal control, diving experiments using atropinized or vagotomized frogs were conducted. The frogs were acclimated at

Fig. 5. Record of diving in December before and after atropine injection (2.5 mg./kg.). Acclimated 18-20° C.; diving temperature 20° C.; body weight 41 g.

Fig. 6. Heart rate of diving vagotomized frogs in October. (A), left vagus severed; (B), right vagus severed; (C), both vagi severed. Acclimated 18-20° C.; diving temperature 20° C.; body weights (A) 74 g., (B) 72 g., (C) 76 g.

18-20° C., and the experiments were conducted at 20° C., since rapid bradycardia normally occurs at this temperature without a severe stress being imposed. This is so at least in all months from late June to early in March.
Seven experiments on the influence of atropine methyl bromide (0.2–3.0 mg./kg. body wt., intraperitoneally) on bradycardia were conducted during December. Figure 5 illustrates diving bradycardia before and after injection. After injection and upon immersion a gradual and steady decline in heart rate occurred during the first 10–15 min.; the delay before decline as illustrated in Fig. 5 may or may not be present. Irregularities of the electrocardiogram never occurred. The lower bradycardia levels approximated to 40 beats/minute; one animal, however, achieved 30 beats/minute. Upon emersion, recovery of heart rate was retarded, and the rate increased only gradually to pre-dive levels.

Figure 6 illustrates the influence of vagotomy on diving bradycardia. During October three experiments were conducted for each of three conditions (see Fig. 6). If either vagus was left intact, rapid diving bradycardia and associated sinus arrhythmia were always observed; differences between the two vagi were not apparent. With bilateral vagotomy only slow bradycardia occurred, and the heart beat always remained regular. The lower heart rates of about 30 beats/minute were not reached for 15–20 min., and recovery after emersion was slow. In contrast to all previous experiments, struggling during diving in bilaterally vagotomized frogs resulted in an immediate but brief decrease in heart rate followed by an increase; the normal response in intact frogs was an increase in heart rate immediately after struggling.

Contrary to expectation, pre-dive heart rates of vagotomized frogs were frequently lower than pre-dive rates of intact frogs at the same temperature. Pre-dive rates of vagotomized frogs were found, however, to vary widely from about 40 beats/minute to about 60–65 beats/minute. Vagotomy resulted in a substantial decrease in muscular tone, particularly evident in the abdominal area, and in oedema. This loss of tone may in part account for lower pre-dive heart rates in vagotomized frogs; it presumably reduces the influence of restraint.

**Fig. 7.** Electrocardiogram of frog in September illustrating influence of decreasing temperature on left vagal inhibition of the heart. Arrows designate 20 sec. duration of 4.5 V stimulus; records read left to right. Acclimated 18–20°C; body weight 79 g.

(c) Vagal stimulation and temperature

Young's (1959) experiments indicate that vagal inhibition might not be expected to occur at the low diving temperatures. Young did not indicate the time of year of his experiments, but in the September experiments reported here reflex bradycardia was always observed at 10°C. The influence of low temperature on vagal inhibition of the heart was therefore determined in September on frogs acclimated at 18–20°C. Ten experiments were conducted on either the right or left vagus.
Electrical stimulation of the left vagus was found to be more effective than that of the right vagus in inhibiting the heart. At room temperature a 4.5–5.0 V. stimulus of 20 sec. duration delivered to the left vagus was sufficient to insure a maximal, usually complete, inhibitory response. The right vagus under similar conditions required approximately 6 V. to insure maximal, though usually incomplete, inhibition.

With decreasing temperature cardiac inhibition by the left vagus (Fig. 7) was partially blocked at approximately 11° C.; that is, an occasional heart beat would occur during stimulation. Complete block occurred at 7–8° C., and at 6° C. an increase of stimulus duration to 2 min. failed to slow the heart. The effects of decreasing temperature on inhibition by the right vagus were similar. With increasing temperature cardiac inhibition by the left vagus was again substantial by 12° C., and complete inhibition returned at about 14° C. Cardiac inhibition by the right vagus was substantial at 14° C., and at 17–18° C. had returned to normal. It is apparent that in the September diving experiments, at water temperatures of 10° C. and above, substantial cardiac inhibition by the vagus can occur and is no doubt responsible for the reflex bradycardia observed.

**DISCUSSION**

These results demonstrate that a rapidly achieved diving bradycardia does occur in restrained amphibians; furthermore, as in other vertebrates, this rapid bradycardia is vagally induced, for it is eliminated by atropine or bilateral vagotomy. In agreement with Jones & Shelton (1964), a slowly attained diving bradycardia, independent of the vagus, may also occur. However, bradycardia without vagal influence is not as marked as that attained with vagal influence. Therefore, in amphibians two forms of diving bradycardia can be distinguished by rate of attainment, control, and degree of heart-rate reduction.

The question now arises as to why Jones & Shelton did not, with the possible exception of a single animal, obtain similar results. As suggested in the introduction, three factors—asphyxia, temperature, and restraint—must be considered when dealing with diving bradycardia in amphibians. The influence of temperature perhaps best explains their results. This is based on two lines of evidence. First, with our range of 10–30° C., bradycardia was slowly achieved only in July and only at the lower temperatures. Jones and Shelton conducted their experiments at 12–18° C. during late spring and summer. Whereas we frequently observed rapid bradycardia at 15° C. in July, they apparently did not observe it even at 18° C.; however their experiments were conducted on *Rana temporaria*. Secondly, there is a substantial difference between the temperatures at which Young’s (1959) results were obtained and those at which vagal inhibition was partially or completely blocked in the experiments here reported. If this is because of a seasonal difference, and if the circumstances of Young’s results approximate to conditions for July, the following hypothesis seems valid. A seasonal variability exists regarding the temperature at which vagal inhibition of the heart can occur; vagal inhibition is reduced or eliminated at higher temperatures in July than in September. Experiments on this point have been initiated, and positive results have been obtained. This hypothesis may explain why Jones and Shelton did not observe reflex bradycardia. A seasonal variability in tonic vagal discharge does exist in the toad (Iriuchijima, 1959).
Vagal control of diving bradycardia in the frog

A second question concerns the control of slowly attained, nonvagally induced, diving bradycardia. This type of bradycardia has been shown to occur when the animals are restrained, but attempts to demonstrate it in unrestrained frogs at 20°C were unsuccessful since the animals would not voluntarily remain submerged more than a few minutes. A pronounced bradycardia was never observed unless the frogs were prevented from surfacing. An important point, however, was that pre-dive heart rates were considerably lower in unrestrained than in restrained frogs. At 20°C, pre-dive rates of unrestrained frogs were approximately 35 beats/minute; no obvious differences in rate could be detected between frogs fully out of water resting on a platform, or merely floating at the water surface. At 20°C, pre-dive heart rates of restrained frogs were generally about 60 beats/minute. This point is significant in that the lower heart rates attained during diving by atropinized or bilaterally vagotomized frogs correspond to the pre-dive heart rates of unrestrained frogs.

![Heart rate of curarized frog in June illustrating consecutive diving begun 20 min. after injection. Acclimated 18–20°C.; diving temperature 20°C.; body weight 46 g. Compare with Fig. 1C and D.](image)

This observation led to the study of diving at 20°C in restrained but curarized frogs (Fig. 8). Such animals, if undisturbed, had pre-dive heart-rate levels similar to normal, unrestrained frogs. When restrained curarized frogs were first submerged, a rapid and pronounced bradycardia occurred; this bradycardia, however, was temporary, and after several minutes the heart rates increased to pre-dive levels before emersion. Upon emersion, little change in heart rate was evident. After three or four consecutive dives the heart rate remained essentially stable whether the frog was again submerged or allowed to remain at the surface. These observations indicate that bradycardia is not inevitable but show that reflex bradycardia may occur upon forced immersion. Perhaps reflex bradycardia would be less likely to occur if frogs were trained to dive under conditions of restraint.

Jones & Shelton (1964) also studied diving in unrestrained frogs but at temperatures lower than 20°C. Examination of the heart rates and temperatures of their records shows that the lowest bradycardia levels of some restrained frogs correspond to pre-dive rates of unrestrained frogs. Their records of restrained frogs showing a pronounced bradycardia may well be due to some vagal influence, as they suggest, since...
irregularities in heart beat were observed occasionally when bradycardia was fully developed; we also noted irregularities in our July records at 10° C.

Upon surfacing a pronounced tachycardia may indeed occur in unrestrained frogs at 20° C., similar to that shown in the records of Jones and Shelton. This tachycardia is temporary, however, and the heart rates decrease regardless of whether the frogs again dive or remain at the surface, although diving does facilitate this decrease.

The influence of restraint is also evident in the diving bradycardia of toads described by Leivestad (1960). The reflex bradycardia observed was undoubtedly vagally induced; the diving temperature was 19–21° C. Leivestad indicated that the instability of the bradycardia resulted from struggling which we also observed to interrupt bradycardia temporarily. The gradual overall reduction in heart rate requiring 20 min. to attain, as observed by Leivestad, may perhaps be due to his method of restraint. In attempts to study diving in unrestrained frogs we tried placing the animals in small cages, leaving them otherwise unrestricted. During submersion the frogs tried to surface almost continually, but in the brief periods when heart rates could be recorded temporary reflex bradycardia was occasionally observed. Leivestad employed somewhat similar methods to submerge his animals.

In the absence of vagal inhibition, slowly achieved diving bradycardia, as observed in restrained frogs, presumably results from asphyxia (hypoxia, hypercapnia, or both) rather than from immersion. Immersion alone, in unrestrained frogs or in curarized frogs, might not induce bradycardia. Four possibilities might explain the action of asphyxia. Asphyxia may act directly on the heart; it may act to decrease sympathetic activity as has been suggested by Jones & Shelton (1964); it may act on the central nervous system as a whole, thereby decreasing muscle tone and general metabolism; or it may act at the tissue level by increasing lactates and other metabolic products. Perhaps all these effects are additive. Jones (1967) reported that a relationship existed between oxygen consumption and heart rate of diving amphibians, but that this was not one-to-one. He found heart rate did not decrease in 100% oxygenated water and that bradycardia was less pronounced if the water around the frog was agitated during diving. It should be noted, however, that in order to establish a relationship between heart rate and oxygen or carbon dioxide, an analysis of blood-gas concentrations is required.

The above discussion suggests that diving bradycardia in amphibians, either slowly or rapidly attained, may not normally occur under natural environmental conditions. Cutaneous respiration is probably sufficient to alleviate asphyxia, at least below 20° C. in fully aerated water. Amphibians do exhibit control mechanisms similar to those of other vertebrates, but because of cutaneous respiration defense against asphyxia is seldom required. Restraint, however, stresses the animal; heart rate and presumably metabolism are greatly increased. Thus with forced immersion, reflex bradycardia follows either out of 'fear' or out of necessity, since immersion imposes demands which cutaneous respiration cannot adequately meet. Atropine or bilateral vagotomy blocks the response. Low temperatures can also block the response with threshold apparently varying with season. Slow bradycardia presumably represents a more or less passive response to partial asphyxia.
SUMMARY

1. The influences of asphyxia, temperature, and restraint on diving bradycardia in amphibians have been considered.

2. In restrained diving frogs two forms of bradycardia were distinguished on the bases of rate of attainment, control, and degree of heart-rate reduction. A rapidly attained immersion bradycardia could be eliminated by atropine or bilateral vagotomy and was thus similar to that of other vertebrates. A slowly attained asphyxiant bradycardia, independent of vagal influences, also occurred but was less pronounced.

3. Electrical stimulation experiments were conducted in September to determine the influence of temperature on vagal inhibition of the heart. With decreasing temperature partial block of vagal inhibition occurred at about 11° C., and complete block occurred at 7°-8° C.; on rewarming, partial block returned around 11°-12° C., and complete vagal inhibition of the heart was re-established at about 14° C.

4. Though both forms of bradycardia may occur in any one dive, the form predominating was dependent on temperature and season. A seasonal variability regarding the temperature at which vagal inhibition of the heart can occur has been suggested.

5. A pronounced diving bradycardia, either slowly or rapidly attained, probably does not occur in unrestrained frogs under normal environmental conditions.

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REFERENCES


