FACTORS INFLUENCING SUBMERGENCE AND THE HEART RATE IN THE FROG

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INTRODUCTION

In view of the intermediate position held by the amphibians between air-breathing and water-breathing vertebrates, surprisingly little is known of the external or internal factors regulating respiratory exchange and related circulatory phenomena in these animals. Spurway & Haldane (1953) have suggested that the breathing behaviour in the Amphibia represents the complete expression of a pattern which becomes truncated in the air-breathing descendants and regained in diving birds, reptiles and mammals. Whether this controversial view is accepted or not, there can be no doubt that a study of respiratory exchange in the Amphibia must give some insight into the development of an air-breathing from the earlier water-breathing system. Before examining the detailed neural pathways involved in respiratory regulation, however, it is important to know what environmental factors govern gas exchange in air and water and so make it possible for the animal to submerge or to breathe in air.

It has been known for some time that submersion produces respiratory and circulatory changes of approximately the same type in those reptiles, birds and mammals which are habitual and well-adapted divers (Andersen, 1961, 1963; Irving, Scholander & Grinnell, 1941; Johansen, 1959; Scholander, 1940; Scholander, Irving & Grinnell, 1942) and to some extent in pearl divers who are not so well adapted (Scholander, Hammel, LeMessurier, Hemingsen & Garey, 1962). These changes consist of apnoea and a slowing of the heart (bradycardia) together with a curtailment of blood flow in which the muscles, periphery, and most of the visceral organs are depleted of blood. As far as has been established the circulatory changes are entirely reflex, whereas the apnoea may be reflex or may contain 'voluntary' components. Certainly many factors are known to affect the inhibition of the rhythmic respiratory centre and in man at least, as Spurway & Haldane (1953) point out, excitation always overcomes the inhibitory processes for the lungs of drowned men always contain water. Whether excitation tends to become dominant and so perhaps be the significant factor in causing the other diving vertebrates to break surface is not clear.

Although a certain amount is known about the mechanics of lung ventilation in Amphibia (Krogh, 1941; de Marneffe-Foulon, 1962) and about the relative importance of skin and lungs as the exchanging surface (Krogh, 1904; Dolk & Postma, 1927), there is very little information for these semi-aquatic animals which can be compared with that available for the less well-adapted diving vertebrates. During submersion the requirements of an amphibious vertebrate would be different in some respects from those of the diving but purely air-breathing types. Rhythmic activity in the respiratory...
centre must be inhibited in both cases but the demands placed on the circulatory system and on the general body metabolism ought to be dissimilar. Poczopko (1960) has shown that in the frog there is an increase during submersion in cutaneous circulation as measured by the number of open skin capillaries. He also suggests on the basis of a decrease in heart rate, as determined on the exposed heart, that there is a reduction in the flow of blood to other organs. The diving bradycardia has been confirmed by Leivestad (1960) working on the toad, and by using a calorimetric method this author has found that at 20°C the tissue metabolism is reduced to 20% of the resting level in air as also is the oxygen consumption. The animal does not appear to build up an oxygen debt, however. The situation in the amphibians so far examined seems therefore to be complicated in that it falls between that seen in diving birds and mammals where an oxygen debt must be incurred during submersion, and that in a truly amphibious animal where the same respiratory and metabolic equilibria could be maintained in both aquatic and terrestrial environments. Though the submerged amphibian increases the exchange capabilities of the skin it appears likely that the body surface is inadequate to meet the demands placed on it at temperatures approaching 20°C, so that some radical reorganization of the metabolism is necessary if oxygen debt is to be avoided.

Under these circumstances the aquatic environment must place a fairly severe restriction on the amphibian and it is significant to have some measure of the capacity of the animal for submerging and remaining submerged. In the present paper an attempt is made to determine the influence of environmental gas concentrations on the balance between submergence and air breathing in the frog and the effect the change from one type of respiratory exchange to the other has on breathing and heart rate.

METHODS

The experiments described have been carried out on some 150 frogs (*Rana temporaria* L.), varying in weight from 18–40 g. The work was done in the winter, spring and summer of one year and, since seasonal variations are known to exist in amphibian metabolism (Krogh, 1904), further details of temperature and season are given in the appropriate sections. The frogs were held in tanks within the department and no seasonal variation could be seen in their activity. So far as methods are concerned the experiments fall into three main categories: those in which the animal was fastened to a Perspex board, those in which the animal was not restricted in its movement about the experimental tank, and those in which the animal had been vagotomized and was under anaesthesia. Care was taken in the experiments in which the animal was restrained that the attachment of the limbs to the board did not occlude the limb veins and to this end the clamps were protected with sponge rubber and only lightly fastened. The first two types of experiment were carried out in a Perspex tank of 12 l. capacity through which water flow at a rate of 1–2 l./min. could be maintained. This kept the temperature constant within 1°C for the duration of an experiment. The air temperature in the tank was also kept constant and as near as possible to the water temperature since temperature is known to have a considerable effect on heart rate. Changes due to such things as evaporative cooling could not be avoided, however, and where possible the time that the animal spent completely out of water was kept to a minimum.
Oxygen and carbon dioxide content of the incoming water was varied by saturation with gas mixtures in which the constituent gases were accurately monitored by means of flow-meters. As a further safeguard samples of the water were taken regularly from the tank and the concentrations of the two respiratory gases were determined. Oxygen was measured by Winkler's method, the thiosulphate titration being usually replaced by measuring the optical density of the liberated iodine in an E.E.L. absorptiometer. Free carbon dioxide was determined from the pH and carbonate equilibrium, the former being measured on a direct reading meter and the latter by sulphuric acid titration to pH 4.

The electrocardiogram (e.c.g.) was obtained by means of steel or silver wire electrodes which were insulated with varnish except at the tip. When the animal was attached to the Perspex board the electrode could be fixed to the board and located under the thorax without the necessity of piercing the skin. At other times the electrode was pushed through the skin into the region of the pectoral girdle. When the animal was free to move about in the tank the electrodes had to be sewn into position with a loop of cotton and connected to the amplifier with very fine, varnished, copper wire. The indifferent electrode was located either in the water of the tank or in the limbs of the frog. The e.c.g. signal was amplified in a Tektronix 122 preamplifier and displayed on an A.E.I. pen recorder or on a Tektronix 502 oscilloscope.

The experiments in the 12 l. tank were carried out on unanaesthetized animals and in all of these at least 1 hr. was allowed to elapse after establishing the animal in the apparatus before any recordings were taken. For the experiments in which vagotomized frogs were used, anaesthetized animals were fixed to a wax block in a much smaller tank (1 l.). The anaesthetic was Sandoz MS 222 in solution in the water bathing the frog. For the vagotomy itself the animal was deeply anaesthetized in 300 mg. MS 222 per litre. The whole of the visceral branch of the vagus was sectioned bilaterally in the angle of the jaw just central to the point of origin of the cardiac branch. The approach to the nerve in this region involved a 1 cm. aperture in the skin and the minimum of damage to the animal. Post-mortem examinations were made to determine that the operations had been successful. The frog was allowed to recover from the anaesthetic after vagotomy and then re-anaesthetized at a light level (50–100 mg. MS 222 per litre) for the experiments on heart rate.

RESULTS

(a) The effect of gas tension changes on submergence

The frogs were watched as they moved about the large tank where they could, without effort, raise their nostrils above the water surface. The experiments lasted from 2 to 4 hr. They were carried out in winter and early spring in water temperatures from 7 to 10° C. Under condition of air saturation (and, in a few experiments, of oxygen enrichment) the animal would spend very little time at the surface breathing air and would commonly remain under water for the full duration of a 3 or 4 hr. experiment (Fig. 1). During submergence the nostrils were closed and the rhythmic breathing movements usually stopped though occasionally oscillations of the buccal floor were seen. Since the nostrils were closed this probably represented movement of air from lungs to the buccal cavity and back again. Sometimes air was lost from the mouth
during this manoeuvre. As the oxygen content of the water was reduced below air saturation the animal spent more time at the surface breathing and, below 5 mg. oxygen per litre, air breathing was always seen in the experiments, even those of only 2 hr. duration.

When similar experiments were carried out in water containing higher concentrations of carbon dioxide than the air-saturation value, it was found that the animals were not on the whole very sensitive to this gas. Though the behaviour of the frogs was rather variable the presence of increasing concentrations of carbon dioxide had little effect until levels around 100 mg./l. were reached (Fig. 2). Concentrations higher than this led to the animals spending a consistently longer time at the surface than they did at lower concentrations.

(b) The heart rate during submergence

The experiments described in this and the succeeding section were carried out in the late spring and summer at water temperatures between 12 and 18°C. Complete submergence of the frog when fastened to the Perspex board always resulted in the appearance of a pronounced bradycardia (Fig. 3), though the periods of submergence in fully aerated water did not exceed those observed in the untethered animal. During
the submergence transient increases in heart rate were observed, usually related to short bursts of activity, but complete release from the bradycardia did not occur until the animal was brought up into air. In general it was found that the heavier frogs showed a greater decline in rate than the lighter ones (Fig. 3). When the diving bradycardia was fully developed the heart occasionally became arrhythmic; atrio-ventricular blockage was observed on only a few occasions. The PR interval increased as the heart rate decreased whereas the RT interval often shortened, though, not consistently so. These changes were reversed when the animal surfaced. The full extent of the decline in heart rate usually took some 15–30 min. to become apparent and in this respect the amphibian differs from the diving reptiles, birds and mammals in which a much more rapid response is seen. The recovery to the pre-submergence rate was, on the other hand, a very rapid process and was usually complete in a minute or less.

\[\text{Fig. 2. The relationship between carbon dioxide content of the water in which frogs are submerged and the time which they spend visiting the surface to breathe air. Temperatures 8–10° C.}\]

In order to confirm that the slowing of the heart was not produced by the somewhat artificial circumstances of the dive in the experiments described above, a few determinations were done on frogs which were allowed to roam freely in the tank and were attached to the recording apparatus by fine wires only. The water contained gases at air-saturation levels and so the frogs remained submerged for long periods. The heart
rate tended to be quite low and the occasional visit to the surface was invariably followed by an increase in rate (Fig. 4).

Several factors may be involved, together or separately, in the appearance of the bradycardia. Submergence may give rise to changes in the general metabolism due to shortage of oxygen and perhaps excess carbon dioxide, as well as to the more direct stimuli produced by the water surrounding the body or the cessation of rhythmic breathing. Any of these things could be the signal which results directly in slowing of the heart. Immersion of the body of the frog, excluding the head, had no noticeable effect on heart rate. However, immersion of the head alone, to the level of the tympanic membranes, caused a depression in heart rate but to a lesser extent than did total submergence. For example, in two frogs immersion of the head caused the heart rate to fall to 75 and 56% of the values recorded in air, whereas total immersion of these two animals resulted in decreases to 62 and 38% respectively. The anoxic conditions

Fig. 3. The effect of submersion on the heart rate of five frogs. The downward-pointing arrow indicates submersion, the upward ones emersion.
Factors influencing submergence and heart rate in the frog

Fig. 4. The heart rate of three frogs moving freely in the experimental tank. Selected sections from recordings taken over longer periods of time. Arrows indicate submersion and emersion as in Fig. 3.

Table 1. The reduction of heart rate produced by total immersion, prevention of buccal movements in air, and prevention of buccal movements with the trunk in water

Temperature 13–15° C. The heart rate at the beginning of each experiment and the lowest rate achieved after treatment are given. The lowest rate is also expressed as a percentage of the initial rate in air.

<table>
<thead>
<tr>
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<th>Total immersion</th>
<th>Floor of buccal cavity clamped</th>
<th>Buccal cavity clamped and body immersed</th>
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<tbody>
<tr>
<td>Frog 1</td>
<td>42–25 (60 %)</td>
<td>41–35 (85 %)</td>
<td>44–30 (68 %)</td>
</tr>
<tr>
<td>Frog 2</td>
<td>40–25 (63 %)</td>
<td>42–36 (86 %)</td>
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<tr>
<td>Frog 3</td>
<td>45–24 (55 %)</td>
<td>43–35 (81 %)</td>
<td>45–26 (58 %)</td>
</tr>
<tr>
<td>Frog 4</td>
<td>39–25 (64 %)</td>
<td>43–35 (81 %)</td>
<td>45–27 (60 %)</td>
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produced by immersion of the head of the frog ought to be reproduced if the buccal floor is prevented from making ventilation movements. This was done by lightly clamping the floor to the roof of the buccal cavity by means of a small, U-shaped, screw clip. This treatment was hardly as effective as immersion of the head but in nine out of eleven experiments a bradycardia was produced which became more pronounced if the animal's body was then submerged to the level of the tympanum, the head being in air. The effect of this on the heart rate in four frogs is summarized in Table 1. The heart rate went up on emersion but complete recovery was not seen
until the clamp was removed. Submersion is thus not an indispensable factor, though it may be a contributory one, in the production of bradycardia.

When the animal was submerged in water whose oxygen content had been lowered below the air-saturation level it was found that the bradycardia became more pronounced. The critical level for producing an effect greater than that seen in air-saturated water was about 5 mg. oxygen per litre and the greatest reduction in rate occurred when the water was devoid of oxygen (Fig. 5). A curious feature of these experiments was that after submergence in water containing oxygen below this critical amount there appeared to be some acclimation on the part of the frog. Subsequent immersions in water containing oxygen at air saturation did not cause the same reductions in heart rate as were produced before the treatment in water depleted of oxygen (Fig. 5). Repeated immersion in water saturated with air never produced any evidence of this type of acclimation.

The effect of low concentrations of carbon dioxide on the heart rate was negligible, and not until gas mixtures containing more than 2-3% carbon dioxide were used, and the concentration in water reached approximately 50 mg./l., was there any noticeable enhancement of the normal bradycardia (Fig. 6). Extremely high concentrations around 200 mg. carbon dioxide per litre had a very marked depressant effect on heart rate, and recovery from this treatment was slow because breathing movements were not immediately resumed when the animal was brought to the surface. When regular breathing eventually developed there was a pronounced increase in heart rate.

Finally, in an attempt to decide between the level of anoxia and the breathing movements as agents important in the production of, and recovery from, bradycardia, some experiments were carried out in which the animal was brought up into an atmosphere of nitrogen. The nitrogen was contained in an open-bottomed Perspex box on the surface of the water. A constant stream of the gas was passed through the box but contamination from the water which was oxygenated at air-saturation level, and probably oxygen from the animal itself, resulted in oxygen levels which were as high
Factors influencing submergence and heart rate in the frog

as 0.3%. The oxygen content of the gas was continuously monitored by means of a polarographic oxygen electrode and was never found to exceed this level, which was assumed to be insignificant in this type of experiment.

Both the e.c.g. and the breathing movements were recorded in these experiments, the latter by means of a photocell on to which a shadow of the buccal cavity floor was cast. Two types of excursion were shown by the buccal floor (Fig. 7), the smaller ones corresponding to buccal ventilation alone and the larger to change of gas in the lungs. The extra downward excursion which occurs on the records at the end of shallower buccal movement represents a greater expansion of the buccal cavity as it is filled from the lungs. After submergence, or after periods of anoxia otherwise induced, the breathing pattern in air was different from that seen in the animal before diving (Fig. 7, cf. records c and f with a). The frequency of the movements increased and there was always a preponderance of the pulmonary type of excursion. After an initial period, consisting almost entirely of lung-ventilating movements, there was a progressive return to a balance between buccal and pulmonary ventilation. The animals also appeared to be breathing more deeply after a period of submergence than before. This was difficult to confirm with the recording system used because slight movements on the part of the animal invariably changed the light path sufficiently to make long-term comparisons unreliable. It would appear from this increased breathing that the animals were working off an oxygen debt, unlike the toads used by Leivestad (1960) in his experiments. No determinations of oxygen consumption or tissue metabolic rates have yet been made on the frog, however, and so direct confirmation of oxygen debt is lacking. When the frog was brought up into an atmosphere devoid of oxygen the pattern of breathing movements was quite characteristic and very different from that already described. A large number of pulmonary ventilations was seen but the
usual alternation of pulmonary and buccal ventilation did not develop. Instead the movements were grouped into periods, 10–15 sec. in length, when the lungs were ventilated, sometimes alternating with periods of entirely buccal ventilation. Lung-filling movements continued throughout the period at the surface but the buccal respirations, when present, decreased in amplitude and stopped completely so that long respiratory pauses (up to 60 sec.) separated the groups of pulmonary movements (Fig. 7c).

Bringing the frog up into an atmosphere of nitrogen caused an immediate cessation of the bradycardia (Figs. 7d, 8) the heart rate often increasing to the level recorded before the dive. When the animal was left to breathe the nitrogen, the rest of the body being immersed in well oxygenated water, the heart rate quite quickly decreased again (Fig. 7e). The bradycardia thus developed was never as complete as that achieved by submergence. On re-submerging the animal after a period of nitrogen breathing the heart rate decreased even further and frequently reached a new, low level (Fig. 8). This type of treatment did not appear to be at all harmful to the animals and they survived long periods of breathing nitrogen when the body was immersed in well-aerated water. Recovery was complete (Fig. 7f).
Factors influencing submergence and heart rate in the frog

Fig. 8. The effect of emersion into air and nitrogen on the heart rate of a frog (25 g.). 13°C. Arrows indicate submersion and emersion as in Fig. 3.

Fig. 9. Bradycardia produced by submersion of a frog before (a) and after (b) bilateral vagotomy. 17°C. Arrows indicate submersion and emersion as in Fig. 3.

c) The effect of vagotomy on diving bradycardia

The lightly anaesthetized frog, as used in these experiments, was not different from the unanaesthetized animal as far as the reaction of the heart rate to submergence was concerned (Fig. 9a). After vagotomy the heart rate was not immediately determined and the frog was allowed to recover; in total a period of 2–3 hr. elapsed before the next records were taken. After this time the heart rate, again determined on the lightly
anaesthetized frog, was often found to be at the same level or even lower than before vagotomy. The significance of this observation may be questionable since the anaesthetic used is known to have an accelerating effect on fish hearts (Shelton & Randall, 1962) and will probably have some effect here. When the animal was submerged the decline in heart rate was seen as before (Fig. 9b). A remarkable degree of agreement was obtained in four out of ten experiments between the final rate achieved after 30 min. submergence in both the intact and vagotomized animals. The speed at which the bradycardia was developed varied slightly and in most cases no consistent changes were seen which could be attributed with certainty to the nerve section. The recovery processes tended to occur more slowly than in the intact animal as Fig. 9 shows, though not always so clearly as seen in this figure. In one experiment evidence was obtained that vagal inhibition was playing a part in the development of diving bradycardia. The heart rate fell rapidly on submergence, the frequency fluctuated considerably, and the beat became arrhythmic after 5 min. When the vagi were sectioned in this animal bradycardia was produced on submersion but over a much longer time course and the beat remained rhythmic for the full 30 min. period. Clearly some individual variation exists in the way in which the heart rate is decreased when the frog submerges. These results suggest that a considerable part of the decrease is independent of vagal inhibition but that this nerve may sometimes play a part in both the development and cessation of bradycardia.

DISCUSSION

In spite of some superficial similarities the metabolic adjustments shown during a dive by birds and mammals on one hand and Amphibia on the other differ in many important respects. In conditions of good aeration and at temperatures up to approximately 12° C. it appears that frogs can maintain an effective relationship with the environment which will enable them to remain submerged for protracted periods without incurring an oxygen debt. Above this temperature no determinations have been made of the ability of the frog to remain submerged but it might be expected that this decreases progressively as the temperature increases. At temperatures approaching 18° C. the measurements of breathing rate after a dive indicate that a steady state is no longer maintained and that the frog begins to incur an oxygen debt whilst under water. This cannot be a substantial one, however, and was not detected by Leivestad (1960) in the toad. Although we found some increase in breathing after submergence there was no compensating increase in heart rate above the normal level. To maintain this complete or nearly complete steady state when submerged some redistribution of blood is necessary, and in the amphibia a decrease in the resistance of the skin capillaries occurs, favouring cutaneous gas exchange. In comparison the skin is depleted of blood during the diving of higher vertebrates. The other very important factor is the ability of the amphibian to lower its basal metabolic level as gas exchange becomes difficult and oxygen less readily available to the body (Leivestad, 1960). The bradycardia which develops on diving is probably more an expression of this latter factor than of the redistribution of blood within the body. The rather long period of time taken for the bradycardia to develop suggests that this is the case. By contrast, in the diving birds and mammals there can be no possibility of maintaining a steady state;
oxygen must be conserved from the outset and sent to organs such as the brain where oxygen shortage would be most damaging. In these animals a rapid curtailment of blood flow through the majority of organs is required and, in fact, the heart rate falls quickly as the transfer from one type of circulation to the other is reflexly produced.

Oxygen concentration has been demonstrated to be a very important factor in determining both the ability of the animal to remain submerged and the extent to which the heart rate is reduced when submerged. At concentrations below 5 mg. of oxygen per litre the effect on both of these is very noticeable. Carbon dioxide has also been shown to have its effect both on bradycardia and on the ability to remain submerged, but at concentrations which are so high as to be unlikely in nature. Krogh (1904), and many other authors, have suggested that this gas is eliminated very rapidly through the skin in Amphibia and so accumulation in the body is unlikely unless the animal finds itself in environments containing very high levels.

Oxygen concentration is not the only important factor having an effect on heart rate, however. The experiments in which frogs were brought up into nitrogen show quite clearly that the breathing movements themselves are in some way able to influence heart rate. The release of rhythmic breathing can, without changing the animal’s oxygen supply, briefly terminate a bradycardia produced by submersion. The converse appears to be true only to a slight extent. The sudden stoppage of rhythmic breathing has an immediate effect on heart rate but this is usually small compared with the slow, steady decline which follows.

An explanation of the results described in this paper can be given in terms of the controlling influence of oxygen concentration and the connexion between breathing movements and heart rate. Submersion slows the heart because the breathing movements cease and the oxygen exchange is curtailed. Total submersion is more effective in the production of bradycardia than is stopping the breathing movements with a clamp or by immersion of the head alone because it gives a more complete anoxia, diffusion of oxygen through water being slower than in air. Emersion of the frog releases the bradycardia immediately because the onset of breathing has a stimulating effect on the heart and there is also, under normal circumstances, a recovery from anoxia. Other factors concerned in control have clearly not been eliminated completely but for the present there is no need to postulate more than the two above.

A complete analysis of the receptor systems and pathways of co-ordination cannot be given from the results at present available. Some evidence that the carotid gland is an important oxygen receptor has been provided by Smyth (1939), and this may play a part both in the response of the whole animal and in the production of bradycardia. A surprising feature of the bradycardia is that it is still found in vagotomized animals. The normal inhibitory pathway is thus eliminated as an essential participant in the response to oxygen lack. If the heart is slowed via a nervous connexion this can only exist in the sympathetic nerve supply to the heart. Since activity in this system accelerates the heart it must therefore be supposed that the cardiac slowing is produced by a decrease in sympathetic tone. Such a hypothesis is not well supported by existing evidence. An alternative possibility is that the lack of oxygen has a direct effect on the heart itself. Clarke, Eggleton, Eggleton, Gaddie & Stewart (1938) have described the effect of asphyxia on the isolated ventricle of the frog as a decrease in the strength rather than in the frequency of contraction, although some of their records do show a
fall in frequency. At the moment therefore it is difficult to decide how the response to oxygen lack is mediated. What does seem certain is that the influence of the breathing movements on heart rate must be an effect mediated by the nervous system. It is difficult to imagine a mechanism other than a nervous one which would be effective. The experiments on vagotomized animals show that, in some cases, sudden changes in heart rate seen before vagotomy cannot be produced after the operation. In addition, preliminary experiments using the adrenaline antagonist, yohimbine hydrochloride, indicate that the sympathetic nervous system may be involved in the quick recovery of heart rate when breathing begins after submersion.

**SUMMARY**

1. The ability of the frog to remain submerged declines as the oxygen concentration in the water falls or the carbon dioxide content rises. The critical oxygen concentration appears to be about 5 mg./l. and the critical carbon dioxide concentration 100 mg./l. at temperatures around 10° C.

2. Submergence results in a decrease in heart rate which develops over a period of 15-30 min. but which disappears immediately the animal surfaces and breathes. The bradycardia is accentuated by oxygen lack or carbon dioxide excess.

3. During submergence the heart is influenced by two main factors, the shortage of oxygen and the cessation of breathing movements, both of which contribute to the decrease in rate. The former can still affect rate after vagotomy. The connexion between breathing and heart rate is dependent on the nervous system, though the detailed pathway is not worked out.

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**REFERENCES**


Factors influencing submergence and heart rate in the frog


