Forced and voluntary diving in ducks: cardiovascular adjustments and their control


Department of Zoology, University of British Columbia, Vancouver, B.C., Canada V6T 2A9

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Diving ducks submerge voluntarily for less than 1 min yet, in forced dives in the laboratory, redhead ducks can endure at least 8 min underwater. This is much longer than a dabbling duck of the same body mass can endure and is a result of the quicker onset of oxygen-conserving cardiovascular responses in divers. Oxygen conservation during forced dives is indicated by a profound bradycardia as blood flow is restricted to cerebral and central cardiovascular areas. In voluntary dives, on the other hand, heart rate is frequently above resting rates, and blood flow is preferentially directed to the working muscles of the hind limbs. Profound bradycardia only occurs in unrestrained ducks when they are trapped underwater. Since leg movements cease within 30 s after ducks are trapped, blood flow must at that time be directed away from the working muscles, as in the "classical" oxygen-conserving dive response. Cardiovascular adjustments to forced diving are caused by stimulation of nasal receptors in diving ducks. In dabblers, arterial chemoreceptor stimulation is crucial to the response, although an intact baro-static system may be necessary for development of profound bradycardia. Baroreceptors are essential for the cardiac response observed when dabblers are trained to dive for food, although neither baro-, chemo-, nor naso-receptors appear to have much to do with the cardiac adjustments to voluntary submergence in diving ducks. Nevertheless, in divers, cardiac adjustments to dabbling and forced, voluntary, and trapped dives are linearly related on a plot of dive (trapped) against the logarithm of pre-dive (pretrap) heart rate. This relationship is due to a similar increase in vagal activity, of some 50% of maximum, in all types of diving manoeuvres. Phychogenic factors, long thought to be important in cardiac responses to forced diving, would appear to underpin this relationship.

Introduction

Voluntary diving and dabbling

Voluntary submergence by diving ducks requires a considerable amount of energy to overcome the forces of drag and buoyancy. In a 0.5-kg diving duck, the buoyant force is about 1 N and is some 3-4 times greater than the subsurface drag force (at swimming speeds of 0.5-0.6 m s⁻¹). When ducks are at least three body surface diameters below the air-water interface, the subsurface drag force is about twice that of animals swimming on the surface at speeds of 0.5-0.6 m s⁻¹ (M. R. A. Heieis, D. R. Jones, and R. W. Blake, manuscript in preparation). Consequently, the bird expends most of its energy during submergence overcoming the effects of buoyancy.

Diving ducks spend a large amount of time underwater. Pedroli (1982) found that tufted ducks (Aytha fuligula) spent as much as 5.9 out of 24 h under water, and that nearly all of this foraging activity took place within a 14-h period. Siegfried (1974) reported that lesser scaup (Aytha affinis) spent 5.5 h underwater during a 12-h period. Therefore, in these two aythylids, between 40 and 45% of the total foraging period was spent underwater, representing 20-25% of the day. In a field study, we observed a lesser scaup perform a series of 42 dives over a period of 20 min, during which 72% (14.4 min) of the time was spent underwater. The mean dive time was 20 s and the mean pause between dives was 8 s. It is not certain how long the bird can maintain this high dive-pause ratio. A series of dives was terminated by a long pause that often lasted.
several minutes. Plasma concentration of lactic acid in tufted ducks swimming on the surface increases more than twofold when swimming velocity increases from 0.3 to 0.7 m·s⁻¹ (Woakes and Butler 1986). The underwater velocity of lesser scaup is around 0.6–0.8 m·s⁻¹ on artificial ponds, and if lactic acid increases when they are swimming at these velocities, even while breathing, it is likely that lactic acid also increases during a dive. The short interdive pauses may not be sufficient to clear this anaerobic by-product, thereby causing a slow accumulation of lactic acid during the diving bout. This may explain the termination of the diving bout; of course, there may well be other reasons for terminating diving, such as satiation, or to allow time for the bird to restore feather condition by preening.

Diving behaviour of lesser scaup diving on a natural pond of unknown depth has been analysed quantitatively using a log–survivor plot (Fig. 1; technique described by Fagen and Young 1978). Predictions can be made from a log–survivor plot because the slope of any segment of the function is proportional to the probability that the event will terminate. Figure 1 shows that for lesser scaup the probability that a dive will end in less than 10 s is nearly zero, but the probability of a dive ending is very high for dives lasting longer than 20 s. The greatest probability that a pause will end and a dive begin occurs between 6 and 10 s, after which the probability decreases and is nearly zero above 30 s. The specifics of this figure do not apply to all diving birds, and do not apply even within a single species if a particular bird is diving in water of a different depth because, as Dewar (1924) showed, dive times and dive—pause ratios are positively correlated with the depth of the water in which the bird is diving.

Dabbling ducks are also aquatic feeders, but usually restrict themselves to shallow areas where they can reach the bottom by tilting their bodies so that only the head and thorax are under water. Szijj (1965) reported that, during dabbling, mallards (Anas platyrhynchos) remained submerged for 4.2–5.8 s, and northern pintails (Anas acuta) remained submerged for 4.8–6.2 s. Our own observations of dabbling mallards on a man-made pond gave average submergence times of 3 s with a maximum underwater time of 9 s (Furilla and Jones 1987b). Dabbling ducks have been observed actually diving for food in deep water (for a review see Furilla and Jones 1987b), and we have trained mallards to dive for food on a man-made pond with water 1 m deep. Mean dive time was 6 s with a maximum dive time of 12 s (Furilla and Jones 1987b). Dive—pause
FIG. 3. Blood flow distribution at rest (left panel) and during a voluntary dive (right panel) in a lesser scaup (Aythya affinis) determined by trapping of macro-aggregated albumen (40 μm diameter), labelled with technetium-99m, in the capillaries. Posterior views from gamma-camera scans. In both panels the head is uppermost (M. R. A. Heieis and D. R. Jones, manuscript in preparation).

ratios varied among individuals, but were usually less than 1. Because our mallards were diving on a man-made pond, we cannot say with certainty that the dive–pause ratios are representative of the behaviour of mallards diving in water 1 m deep in the wild.

Maximum underwater endurance

Diving ducks usually remain underwater voluntarily for less than 1 min, but what is the maximum time that these animals could remain underwater if the need arose? A 700-g diving duck (Aythya americana) was forced to dive for 8 min and another, of unknown but similar body mass, was forcibly submerged for 6 min. These animals were not removed from the water because of any sign of physiological stress, but because the experiment had been concluded, and it is our opinion that these birds could have remained underwater well beyond these times. In a recent study of the influence of body mass on dive time of Pekin ducks, Hudson and Jones (1986) presented the relationship \[ t_d = 6.6 M_b^{0.64} \], where \( t_d \) is dive time in minutes and \( M_b \) is body mass in kilograms. Therefore the maximum dive time of a 700-g Pekin duck would be 5.3 min if the same relationship held for divers as for dabblers. What could allow diving ducks to remain underwater longer than dabbling ducks of equal body mass?

The cardiovascular adjustments to forced submergence occur gradually in dabbling ducks, taking from 30 to 60 s after submersion for full development. Butler and Jones (1971) showed that if the heart-rate response to submersion was eliminated by injection of atropine, then in the first 30 s of the dive, the fall in arterial oxygen tension (\( P_{aO_2} \)) was virtually identical with that in untreated ducks. Pekin ducks habituated to show no bradycardia during a forced dive and naive ducks showing strong bradycardia have identical \( P_{aO_2} \) after 40 s submergence (Gabbott and Jones 1987). In contrast to that of dabblers, the heart-rate response to forced submersion of a diving duck is rapid, with maximum bradycardia occurring within 2–5 s of submersion. Local anaesthetic (Xylocaine) sprayed into the nasal passages of diving ducks virtually eliminates diving bradycardia (Furilla and Jones 1986). In two redhead ducks, \( P_{aO_2} \) at 40 s submergence was significantly lower in the absence of diving bradycardia than in the same animals when their heart rate fell during submergence (Table 1). In fact, in a single untreated duck, \( P_{aO_2} \) was higher after 2 min submergence (50.8 mmHg, 1 mmHg = 133.3 Pa) than any value seen after 40 s in nonbradycardic dives. Therefore diving ducks are capable of reducing the rate of oxygen depletion earlier in the dive than dabbling ducks are, which prolongs underwater endurance.

Cardiovascular responses to diving

Cardiovascular adjustments to voluntary and forced diving

The heart rate response to voluntary diving in diving ducks is characterized by an increase beginning 3 or 4 s before the dive with a decrease occurring abruptly at or slightly before head immersion (Butler and Woakes 1979; Furilla and Jones 1987a). This pattern occurs in redheads (A. americana), whether they are diving, dabbling, or even voluntarily sub-
merging their heads into beakers of water to retrieve food (Furilla and Jones 1987a). Predive and dive heart rates during dabbling, however, are usually lower than during diving. The lowest diving heart rate is established with the first cardiac interval of the dive and heart rate then rises either to remain stable for the rest of the dive or even to increase somewhat towards the end. However, if access to the surface is denied after the bird has begun its ascent, there is an immediate reduction in heart rate which may approach rates seen in a forced dive at the same time after submersion (Stephenson et al. 1986; Furilla and Jones 1987a).

Dabbling ducks, when dabbling voluntarily, usually show little or no heart rate adjustments associated with head immersion (Furilla and Jones 1987b). When they are diving, however, their heart rate at 2 s submersion is around 250 beats/min (Fig. 2A). Therefore, not only is the degree of change in heart rate variable but the direction of change is dependent on the predive rate (Fig. 2; Furilla and Jones 1987b). Dabbling ducks differ further from diving ducks in that no predive “anticipatory” tachycardia occurs.

The pattern of blood flow in diving ducks during voluntary submersion is also quite different from that seen during forced laboratory dives. The blood flow pattern during a voluntary dive by a lesser scaup is compared with flow at rest in Fig. 3. The lesser scaup was trained to wear a light-activated infusion backpack. After the training period, an arterial cannula was inserted into the brachial artery until the tip lay just outside the aortic valves. The infusion pump chamber was filled with macro-aggregated albumin (MAA) labeled with technetium-99m. MAA has an average particle size of approximately 40 μm and becomes trapped in the capillary circulation so that, after injection, blood flow distribution can be visualized using a gamma camera. MAA is broken down rapidly in the body and as the half life of 99mTc is 6 h, blood flow distribution can be determined in the same animal performing a range of behaviours over several days. In a voluntary dive, blood flow, represented by the level of darkness of the image in Fig. 3, was increased in the hind limb area while it was reduced in the area of the large pectoral muscles compared with flow distribution in the duck at rest. In contrast, during forcible submersion, blood flow is largely restricted to central cardiovascular and cerebral regions (Jones et al. 1979; M. R. A. Heieis and D. R. Jones, manuscript in preparation).

Increased blood flow to the hind limbs in voluntary dives appears necessary to maintain the high energy output of the leg muscles during submersion. Behavioural evidence suggests that muscle oxygen stores may not maintain muscle contrac-

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**Table 1. Arterial oxygen tension (P_{O_2}, mmHg) before and after 40 s forced submersion in two redhead ducks (Aythya americana)**

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<th>With bradycardia</th>
<th>With reduced or no bradycardia</th>
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<td>Predive</td>
<td>During dive</td>
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<tr>
<td>Duck 1</td>
<td>100.7</td>
<td>62.8</td>
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<td>Duck 2</td>
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**Note:** Four dives were done with each duck. Diving bradycardia was reduced or eliminated by spraying local anaesthetic on the nasal area. Dives with bradycardia were done before and after recovery from nasal anaesthesia. The predive value was taken before the first of each pair of dives. Mean P_{O_2} with bradycardia = 60.2 mmHg ± 1.9 (SD); mean P_{O_2} with reduced or no bradycardia = 45.8 mmHg ± 6.0 (SD).

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**Fig. 4.** Traces of isometric tension, in arbitrary units, developed when the tibialis anterior muscle was stimulated electrically at 3 pulses/s. (A) The effect on contraction strength of occluding the ischiatic artery. (B) The effect of a forced dive on contraction strength. Arterial blood pressure indicates cardiovascular performance in the dive and the arrow shows onset of profound bradycardia which is accompanied by a marked restriction in hind limb blood flow. The spikes seen on the tension trace in the dive were caused by struggles.

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**Control of cardiovascular adjustments to submergence**

In dabbling ducks, such as the mallard, the cardiovascular response to forced diving is inhibited by breathing oxygen before the dive but this is not the case in diving ducks (Furilla and Jones 1986). Furthermore, denervation of carotid bodies, the blood oxygen chemosensors, prevents most of the forced dive bradycardia in dabblers (Jones and Purves 1970; Lillo and...
Jones 1982; Jones et al. 1982) but not in diving ducks (Butler and Woakes 1982a). Bradycardia in forced dives is prevented in diving ducks by anaesthetising nasal receptors with a local anaesthetic agent (Furilla and Jones 1986). Interestingly, neither nasal receptors nor carotid body chemoreceptors appear to contribute to the heart rate changes that occur at the start of voluntary submersions in diving ducks (Butler and Woakes 1982a; Furilla and Jones 1987a) although dive times are longer and heart rates higher at the end of voluntary dives by diving ducks after carotid body denervation (Butler and Woakes 1982a).

Mean arterial blood pressure falls about 20% from predive levels in forced dives by dabbling ducks (Butler and Jones 1971). However, when the onset of bradycardia is rapid or heart rate fluctuates considerably in the dive, mean arterial pressure may vary somewhat more than this (Fig. 5). Since blood pressure is the product of cardiac output and total peripheral resistance, its maintenance in dives implies an important role for baroreceptors in the response. However, despite a number of investigations in dabbling ducks, the role of baroreceptors in effecting diving responses remains controversial.

Andersen and Blix (1974) proposed, on the basis of pharmacological evidence, that the baroreceptors were largely responsible for the development of diving bradycardia in ducks. In their scheme, chemoreceptor-mediated increases in peripheral resistance produce a rise in blood pressure, which then provokes bradycardia via the baroreflex. If this is the case, denervation of the arterial baroreceptors should prevent diving bradycardia. In fact, chronically barodenervated ducks, diving some weeks after deafferentation, showed the same degree of bradycardia as intact animals (Fig. 6). However, peripheral resistance in the chronically barodenervated ducks increased by only half the amount that it did in intact ducks and blood pressure fell markedly during the dive (Jones 1973). On the other hand, acutely barodenervated animals, diving within 1–2 days of the surgical denervation procedure, showed less bradycardia than intact animals (Fig. 6; F. M. Smith and D. R. Jones, manuscript in preparation). Acute barodenervation elevated predive heart rate to almost twice the predive level of intact ducks, while the end-of-dive heart rate was only 30 beats/min below the predive rate in intact animals. However, the peripheral vasoconstrictor response was unaffected by acute barodenervation and only a slight dive hypotension resulted, unlike the situation seen in the chronically barodenervated animals. Clearly there is a change in the peripheral resistance response to diving, as well as an adaptation of the dive bradycardia, after a period of time without baroreceptor input. Whether these changes are due to a central neural reorganization of the dive response after denervation, or to peripheral effector-related effects, is not yet certain. Nevertheless, the change in diving response with time after barodenervation makes it very difficult to establish the precise role of these receptors in modulating the cardiovascular adjustments to diving in intact ducks (Fig. 6).

Interestingly, arterial baroreceptors play an important role in the cardiac adjustments to voluntary submergence by dabbling ducks (mallards) trained to dive, rather than dabble, for food. When mallards dabble, their heart rate does not change from the predive rate, but when they dive, heart rate is adjusted to about 250 beats/min, regardless of predive heart rate, even though dives and dabbles may be of the same duration. Chronic denervation of arterial baroreceptors prevents any cardiac adjustments to diving although diving ability seems to be unimpaired (Fig. 2; Furilla and Jones 1987b). Hence, the utility of these cardiovascular adjustments to voluntary diving by the mallard remains to be established. On the other hand, chronic baroreceptor denervation has no effect on cardiac adjustments to either forced or voluntary submergence in diving ducks (Furilla and Jones 1987a).

Recently the suggestion has been made that the cardiac response evoked by forced submersion is actually a component of the defence reaction (Smith et al. 1974; Smith and Woodruff 1980; Kanwisher et al. 1981; Smith and Tobey 1983). The basis of this contention lies in the similarity of cardiac responses to submersion and to fear. Instead of cardiac acceleration, more typical of the “flight or fight” behaviour in the defence reaction, data obtained from cornered or restrained animals may often show cardiac deceleration as part of defensive behaviour. Hence the supposition is that bradycardia in forced submersion has been wrongly attributed to a unique function for conserving oxygen stores during prolonged submersion. This explanation, however, neglects certain observations: (i) animals lacking chemoreceptor or nasal receptor activation do not demonstrate any cardiac changes with forced submersion (Jones and Purves 1970; Furilla and Jones 1986; Gabbott and Jones 1985); (ii) ducks with all brain tissue above the mesencephalon extirpated, including the hypothalamus, show exactly the same responses to forced submersion as do intact ducks (Andersen 1963; Gabbott and Jones 1985). Clearly, activation of these receptors is more important for inducing cardiac change than is the perception of danger by
Fig. 6. Mean arterial blood pressure (MAP), heart rate (HR), and hind limb vascular resistance (HLVR) before and during forced dives of 2 to 2.5 min duration in intact (A), and in acutely (B) and chronically barodenervated (C) dabbling ducks (Anas platyrhynchos) (pru, peripheral resistance units). The intact and acutely barodenervated duck data are from F. M. Smith and D. R. Jones (manuscript in preparation) and the chronically barodenervated duck data are from Jones (1973).

"cognitive" levels of the brain. A reflexogenic element in the response to head immersion exists that is not necessarily incorporated into the complex behavioural patterns that constitute the fear response. Nevertheless, it could be argued that during submersion, chemoreceptor-driven cardiovascular changes are caused by activation of defence reaction output pathways.

Undoubtedly, higher centres within the central nervous system (CNS) have an influence over heart rate which is demonstrated, for instance, in the "anticipatory" decrease in heart rate before voluntary dives begin and the increase in rate before the animal surfaces at the end of a dive (Butler and Woakes 1979). Whether or not this control is volitional or conditioned is not known, especially in view of the demonstration by several researchers that cardiac responses to submersion can be habituated (in ducks: Gabrielsen 1985; Gabbott and Jones 1985) and classically conditioned (in seals: Ridgway et al. 1975; in ducks: G.R.J. Gabbott and D. R. Jones, unpublished observations). Nevertheless, it is well documented how pervasive is the extent of higher CNS control. As long ago as the 1940s, when studies were performed by Irving and Scholander, it was clear that the disposition and nervous state of animals was extremely important for full development of forced diving responses (Irving et al. 1941; Folkow et al. 1967).

The relationship between dive and predive heart rate in diving ducks

The cardiovascular responses to forced submersion are caused by stimulation of chemoreceptors in dabbling ducks and nasal receptors in divers. Chronic barodenervation disrupts cardiac adjustments to voluntary diving in mallards while naso-, chemo-, or baro-receptors appear to make no contribution to heart rate changes in divers. Hence, it seems almost impossible for there to be any relationship between heart rates in forced and voluntary diving (Fig. 7). Nevertheless, it is
obvious that in diving ducks there is a relationship between dive heart rate after 2–5 s of submergence and heart rate in the immediate predive period in both forced and voluntary dives. The higher the predive heart rate, the higher the dive heart rate (Fig. 7). In forced dives this is exemplified by dive heart rates in the range of 200–300 beats/min when ducks are exercised to raise predive rates to 400–500 beats/min (Fig. 8). In contrast, when diving ducks dabble both predive and dive heart rates are low (Fig. 8). Furthermore, heart rates usually seen only in forced dives by restrained animals can be evoked if voluntarily diving ducks are prevented from regaining access to the surface. A scatter plot of all dive and predive heart rates shows that a linear relationship can be established between dive heart rate after 2 to 5 s of submergence and the logarithm of predive heart rate (Fig. 8; Furilla and Jones 1987a). Furthermore, heart rates just after ducks are trapped underwater compared with the rates just before they are trapped also fit this relationship (Fig. 8).

The underlying physiological cause of this relationship must lie with effector (i.e., sympathetic and parasympathetic innervation of the heart) rather than afferent (naso-, chemo-, and baro-receptor) neurogenic influences on heart rate. Efferent control of the heart in ducks has been investigated by implanting stimulating electrodes, bilaterally, on cut distal ends of the vagi and cardiac sympathetic nerves. Vagal stimulation results in rapid heart rate changes while sympathetic stimulation causes much slower effects; thus it is doubtful if changes in sympathetic activity have any marked affect on heart rate up to 5 s after submergence. Heart rates versus normalized stimulation frequencies of vagal and sympathetic nerves are shown in Fig. 9; the surface in Fig. 9 illustrates all heart rates that can be produced by any combination of sympathetic and vagal stimulation. Regions that appear to pertain to intact animals can be identified on this surface. For instance, maximal sympathetic activation in the absence of vagal stimulation gives heart rates of 500 beats/min (point A in Fig. 9), which is the highest rate observed before voluntary dives. β-Blockade with propranolol yields heart rates around 300 beats/min just before voluntary dives (point B). Finally, before forced dives heart rate is between 90 and 140 beats/min and since β-blockade does not lower heart rate further (tested in two ducks), cardiac efferent control in these animals is probably described by the region around point C in Fig. 9. If sympathetic activity does not decrease in a dive (it cannot when predive rates are represented by B and C), the relative increase of vagal activity required to cause the diving heart rates observed in voluntary and forced dives appears to be similar. Specifically, an increase in vagal activity of about half maximum gives the dive heart rates observed between 2 and 5 s in voluntary dives, with and without propranolol, and in forced dives. Further, heart rates in all other dabbles and dives, including heart rate changes when ducks are trapped underwater compared with pretrapped rates, appear to result from a similar increase in vagal activity. The curvilinear relationship between sympathetic and vagal activity and heart rate, alone or in combination (Fig. 9), can be linearized by taking the logarithm of the predive heart rate in the dive:predive heart rate relationship. Furthermore, taking the logarithm of heart rates occurring before electrical stimulation of the vagi and plotting these against heart rates resulting from any increase in stimulation of 50% of maximum gives points that also fit the line describing the dive:predive heart rate relationship (Furilla and Jones 1987a).

Obviously it is the predive heart rate that sets diving heart rate and, at least for heart rates in excess of 300 beats/min, the slope of the dive:predive heart rate relationship is solely attributable to the influence of increasing sympathetic activity on predive heart rate. Even so, what initiates the increase in
Fig. 10. Effect of cycles of lung inflation and deflation with 25 mL of air or 25 mL of air + 5% CO₂ on heart rate during forced submergence of a redhead duck (*Aythya americana*). Upper trace: heart rate meter output; middle trace: intratracheal pressure (I.T.P.), up represents lung inflation; lower trace: electrocardiogram (ECG). Time bar applies to all traces. Cardioacceleration during lung inflation is caused by stimulation of intrapulmonary chemoreceptors which are inhibited by 5% CO₂ in the airway.

Fig. 11. Rapid fluctuations in heart rate of a redhead (*Aythya americana*) correlated with inhalation and exhalation after a struggle caused by pinching the web (pinch indicated by the horizontal bar under, and struggle by noise on, the ECG trace). Upper trace, intratracheal pressure, down represents inhalation; middle trace, heart rate meter output; lower trace, electrocardiogram (ECG). Time bar applies to all traces.

Vagal activity at the start of, or even just before, a dive is a mystery. In forced dives by resting animals bradycardia results from maximal vagal activation and even in voluntary dives vagal activity must be much higher at the initiation of the dive because the first cardiac interval usually represents the lowest heart rate of the dive. In forced dives maximal vagal activity can be provoked by stimulation of nasal receptors (Furilla and Jones 1986) but this cannot be the case in voluntary dives because the initial diving cardiac interval starts to be prolonged before the nasal area enters the water (Butler and Woakes...
Some seconds before the start of a voluntary dive ducks hyperventilate and exhale just before submersion (Fig. 7; Butler and Woakes 1976). Since cardiorespiratory interactions are well established in dabbling ducks (Bamford and Jones 1976) and also seem to hold for divers (Fig. 10), it is tempting to invoke these interactions to explain cardiac responses in voluntary dives. However, these interactions are usually associated with sinus arrhythmia, a waxing and waning of heart rate with inhalation and exhalation, respectively, yet this is never apparent in the immediate predive period. Further, exhalation just before submergence provokes an instantaneous fall in heart rate, not reminiscent of heart rate changes in sinus arrhythmia. In fact, the only times we have consistently observed instantaneous falls in heart rate of the order of 200 beats/min during exhalation in restrained animals is after heart rate has increased because of some form of arousal, such as a struggle or application of an extraneous stimulus that the animal perceives as threatening (Fig. 11). Hence, the initial pre-dive tachycardia and hyperventilation and the immediate increase in vagal activity may be, in part, an arousal response that influences both cardiac and respiratory controllers. The setting of dive heart rate in the first 2–5 s of the dive may represent the influence of habituation or conditioning of the initial increase in vagal activity. In any event, these ideas imply a pronounced psychogenic contribution in voluntary dives while it is our contention that the psychogenic contribution in forced dives is minimal.

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