CONTROL OF HEART RATE DURING DIVING IN DUCKS

by

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ABSTRACT

Forced submergence of redhead ducks (*Aythya americana*) caused heart rate to fall from 100 ± 3 beats•min⁻¹ (mean ± s.e.m., N = 12) to a stable rate of 35 ± 4 beats•min⁻¹ (N = 12) within 5 seconds after submergence. Bradycardia was unaffected by breathing oxygen before the dive or by denervation of the baroreceptors, but was virtually eliminated by local anaesthesia of the narial region. When freely diving on a man-made pond, heart rate of redhead ducks and lesser scaup (*A. affinis*) two seconds after submergence was positively correlated with the pre-dive rate (r² = 0.71). Breathing oxygen before the dive and denervation of baroreceptors had little effect on this relationship. Chasing to induce submergence caused a slight enhancement of bradycardia, heart rate during the dive being about 10% lower than after a voluntary dive. Local anaesthesia of the narial region inhibited voluntary diving, but heart rates in chase-induced dives after nasal blockade were significantly higher (10-30%) than those obtained from untreated ducks in chase-induced dives.

Dive heart rate, at 2-5 seconds submergence, was linearly related to the logarithm of the pre-dive rate for all voluntary and forced dives as well as dabbles. Even the heart rate which occurred 2-5 seconds after being trapped under water as a function of the rate immediately before trapping fitted this relationship. The function was described by the
equation $Y = -451 + 246 \log X$, where $Y = \text{dive (or trapped)}$ and $X = \text{pre-dive (or pre-trap) heart rate (r}^2 = 0.98)$. The relationship was unaltered by β-blockade with propranolol. Data from stimulation of the cut distal ends of vagal and cardiac sympathetic nerves suggest that a similar increase in vagal activity occurs on submergence in all of these dives. The first cardiac interval in voluntary dives represents a lower heart rate, indicating a higher level of vagal activity.

When dabbling ducks (*Anas platyrhynchos*) dabble, heart rate at two seconds submergence is little changed from the pre-dabble rate. When these birds dive, however, heart rate at two seconds submergence is about 250 beats·min$^{-1}$, regardless of the pre-dive rate. Bilateral denervation of arterial baroreceptors significantly altered the dive:pre-dive relationship.

These results have shown that nasal receptors are responsible for bradycardia in diving ducks when forcibly submerged, but that nasal receptors contribute little to the change in heart rate when ducks dive voluntarily. The results also suggest that there is a psychogenic modulation of the heart rate in voluntary dives which influences the pre-dive rather than the dive heart rate. Finally, dabbling ducks differ from diving ducks in their response to forced and voluntary diving. Chemoreceptors are responsible for the majority of the response in forced dives, and baroreceptors provide primary control in voluntary dives.
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GENERAL INTRODUCTION

Diving animals subject themselves to periods of asphyxia during which many homeostatic processes are disrupted. Sustained interruption of a process essential for internal homeostasis will inevitably lead to physiological dysfunction, yet diving animals can survive prolonged periods of asphyxia that would kill many of their more strictly terrestrial analogues. Homeothermic animals are especially susceptible to the danger of oxygen deprivation because of the high energy cost for maintenance of cell function.

As early as the mid-nineteenth century, scientists became interested in diving physiology. In 1870, Paul Bert discovered that ducks experienced a fall in heart rate when forcibly submerged in water. He also showed that ducks could survive submersion asphyxia for up to 20 minutes, whereas chickens died within 3 minutes after submersion. Richet (1899) calculated the oxygen stores of a 1.5 kg duck and deduced that a concomitant reduction in metabolism was necessary for the duck to survive more than 3 minutes underwater. In 1909, Burne, working on the walrus, suggested a redistribution of blood away from the "vegetative organs" during submersion. Scholander (1940) demonstrated that lactic acid concentration was greater in skeletal muscle than in blood during a dive, but blood lactate increased substantially upon surfacing. Thus, it was shown that muscle perfusion was
inhibited and that the muscles were metabolizing anaerobically.

Cardiovascular Adjustments to Forced Submergence

Since these early observations, bradycardia during enforced submersion has been observed in many air-breathing vertebrates including the frog (Jones and Shelton, 1964), snake (Johansen, 1959), muskrat (Drummond and Jones, 1979), vole (Clausen, 1964), fur seal (Irving et al., 1963), porpoise (Irving et al., 1941), hippopotamus (Elsner, 1966), cormorant (Mangalam and Jones, 1984), and duck (Butler and Jones, 1971). In fact, submersion bradycardia appears to be a universal trait among air-breathing vertebrates including man.

Accompanying a fall in heart rate is a reduction of cardiac output. Clearly, stroke volume could not increase sufficiently to compensate for the observed decrease in heart rate. The sea lion (Elsner et al., 1964) and duck (Jones and Holeton, 1972) showed no or little change in stroke volume even in the face of extreme bradycardia.

If adequate perfusion of the brain is to take place, it is essential that arterial blood pressure be maintained. Irving et al., (1942) measured the blood pressure of a seal during experimental dives and showed that, despite an intense slowing of the heart blood pressure remained unchanged. Jones and West (1978) stated that "mean arterial blood pressure is seldom more than 20% changed from the pre-dive level." To
maintain blood pressure in the face of reduced cardiac output, peripheral resistance must increase. Ducks (Jones et al., 1979) and muskrats (Jones et al., 1982) that have been forced to dive experienced a large reduction in the percent of cardiac output flowing to the kidneys, liver, muscles, spleen and small intestine (and lungs in the duck) while flow increased to the brain, heart and, to a lesser extent, the eyes. These cardiovascular adjustments are collectively referred to as the diving response and have been touted as oxygen conserving compensations.

Cardiovascular Adjustments in Freely Diving Animals

Scholander (1940) showed that a seal submerged but free to raise its head and breathe failed to show the expected bradycardia. Extensive research aimed at freely diving animals has had to wait the technological advances which brought about the miniaturization of electronic components. Unfortunately, devices small enough to monitor physiological variables of small animals, are not yet available and many studies require that animals be confined in small tanks while wired to external equipment. For this reason, data from naturally or freely diving animals is not nearly so extensive as that from forcibly submerged animals.

Bradycardia is highly variable in animals trained to dive. Sea lions trained to dive on command had a higher heart rate in these dives than during restrained dives (Elsner
et al., 1964), but dolphins showed a more intense bradycardia after training (Elsner et al., 1966). The Weddell seal (Kooyman and Campbell, 1972) exhibited a mild bradycardia during short dives (<20 minutes), but during longer dives the bradycardia was more intense. More interesting is the fact that within 30 seconds following submersion the heart rate was lower in the eventually longer dives than in the shorter ones. The muskrat always showed an extreme bradycardia, but the first cardiac interval immediately following submergence was more protracted in longer dives than in shorter ones (Drummond and Jones, 1979). Cormorants diving naturally showed no bradycardia (Kanwisher et al., 1981), and the heart rate of freely diving tufted ducks increased just before the dive, but during the dive, did not fall below that while swimming on the surface and was considerably higher than at rest (Butler and Woakes, 1982a).

Kooyman et al. (1980) reported that blood lactate of Weddell seals did not increase significantly following dives of less than 20 minutes, indicating that muscle and organ perfusion was maintained; however, blood lactate rose dramatically following dives of more than 20 minutes. Millard et al. (1973) observed a reduction in blood flow to the leg of a naturally-diving penguin, but as Butler and Jones (1982) pointed out, this may reflect a redistribution of blood away from non-active tissues because the penguin swims with its wings. Kooyman et al. (1971) reported seeing an emperor
penguin freely bleeding from a wound at its wing tip for nearly 2 minutes during a dive.

**Efferent Control of the Cardiovascular Adjustments**

The rapid onset of cardiovascular responses to forced submergence or free diving suggests that they are controlled by neural mechanisms. In birds and mammals, the pacemaker is innervated by cardiac (sympathetic) and vagal (parasympathetic) nerves (Burnstock, 1969). Bilateral vagotomy of the muskrat (Drummond and Jones, 1979) and injection of atropine in the duck (Butler and Jones, 1971) completely abolished the bradycardia associated with submergence, indicating that heart slowing during enforced submersion is governed by the vagus nerve. Butler and Woakes (1982a) showed that the reduction in heart rate from the pre-dive tachycardia of freely-diving ducks was nullified following injection of atropine, and they proposed that the pre- and post-dive tachycardia are brought about by inhibition of vagal tone. Butler and Jones (1971) demonstrated that the post-dive tachycardia of forcibly submerged ducks was unaffected by sympathetic (β) blockade; however, Folkow *et al.* (1967) found that the post-dive tachycardia was attenuated following β-receptor blockade. The nature of the sympathetic influence on the pre- and post-dive tachycardia in freely diving ducks is unknown. It has been suggested by Kanwisher *et al.* (1981) that cardiac rhythm is under volitional control.
Butler and Woakes (1982a) further suggest that the pre-dive tachycardia and hyperventilation serve to increase the oxygen stores by saturating the arterial blood and decreasing the a-v $O_2$ difference. It is tempting to assume that the animal initiates these cardiac adjustments to mitigate the stress that would otherwise lessen its ability to complete the dive successfully, but no such evidence exists.

Peripheral vasoconstriction is under the control of the sympathetic nervous system acting through $\alpha$-adrenoceptors (Butler and Jones, 1971); whereas, vasodilation is brought about by stimulation of $\beta_2$-adrenoceptors (Hillman and Lundvall, 1981) or by metabolic dilators (Folkow and Neil, 1971). During periods of metabolic activity certain organs have increased blood flow (Bevegard and Shepherd, 1967). Ducks exercising on a treadmill underwent a 25% reduction of vascular resistance (Grubb, 1982), presumably to augment perfusion of the working muscles. It seems likely that animals swimming under water should also require muscle perfusion. Folkow et al. (1966) demonstrated that metabolic dilator substances could not abate neurogenic vasoconstriction of the hindlimb of the duck, but the cat experienced reactive hyperaemia during muscular work even with strong sympathetic stimulation. Anatomical studies by these authors showed more dense adrenergic innervation of the large arteries in the hindlimbs of ducks than of cats. If the animal is to perfuse its muscles during a dive, it is clear that neurogenic
constriction must be inhibited, but it is unclear whether muscle perfusion is necessary for a 20 second dive by ducks.

Afferent Control of the Cardiovascular Adjustments

Questions concerning the sensory mechanisms which bring about the dive response have been the subject of investigation for more than a century. These studies have centered on (1) facial, nasal, laryngeal and glottal receptors, (2) central and peripheral chemoreceptors and (3) arterial baroreceptors. Andersen and Blix (1974) considered the barostatic reflex to be of major importance; however, Jones (1973) and Lillo and Jones (1982) demonstrated that bradycardia and peripheral vasoconstriction develop in barodenervated ducks, and Drummond and Jones (1979) could show no involvement of the baroreceptors in the dive response of muskrats. It is probable that in ducks the baroreceptors are responsible for maintaining blood pressure in the face of large changes in cardiac output and vascular resistance once these adjustments are established in the dive, but do not contribute much to the development of the dive response per se in forced dives.

"The progressive hypoxia and hypercapnia that develop during diving would tend to stimulate the central and peripheral chemoreceptors" (Butler, 1982). The role of arterial chemoreceptors in freely diving animals is uncertain, but during forcible submersion of domestic ducks, denervation of the carotid bodies prevented much of the bradycardia (Jones
and Purves, 1970; Lillo and Jones, 1982); moreover, ducks breathing an hyperoxic gas mixture before submersion displayed little or no bradycardia during enforced submersion (Mangalam and Jones, 1984). Carotid body denervation had no effect on the initiation and depth of bradycardia from the pre-dive levels of freely diving tufted ducks (Butler and Woakes, 1982b), and Butler (1982) reported that the carotid bodies were not involved in the bradycardia that developed when a tufted duck was prevented from surfacing. Arterial chemoreceptors are not essential in initiating the diving response in seals (Daly et al., 1977) and muskrats (Drummond and Jones, 1979); however, if the chemoreceptors were stimulated in forcibly submerged seals, heart slowing was considerably augmented (Elsner et al., 1977; Daly et al., 1977).

In many birds and mammals, the gradual changes in blood gases following submersion could not account for the rapid onset of cardiovascular adjustments. Tufted ducks (Butler and Woakes, 1982a) showed an immediate increase (about two-fold) in the cardiac interval after a voluntary dive, and the cardiac interval of muskrats was expanded from approximately 0.2 seconds to more than 1 second immediately after an unrestrained dive (Drummond and Jones, 1979). When the nasal, facial and laryngeal receptors of muskrats were extirpated by nerve section, there was no submersion bradycardia (Drummond and Jones, 1979). Furthermore, local anaesthesia of the face
of artificially ventilated harbor seals completely abolished
the bradycardia associated with submersion (Dykes, 1974).
Angell-James and Daly (1972) showed that bradycardia and
peripheral vasoconstriction could be elicited in dogs by
stimulation of the nasal passages with water.

It is clear from these observations that the control of
cardiovascular adjustments in freely diving animals is
uncertain. Moreover, the complex responses brought about by
reflex interactions have not been investigated in freely
diving animals.
SECTION 1

The Heart Rate Response and its Control during Restrained and Unrestrained Diving by Diving Ducks

INTRODUCTION

The cardiac response to diving shown by diving ducks submerging voluntarily is very different from that obtained by force diving the same ducks in the laboratory (Butler and Woakes, 1979; 1982a&b). In voluntary diving, heart rate increases before the first dive in a series, and on, or even just before, submersion there is a transient bradycardia. Heart rate then increases in the first few seconds of the dive to a steady rate, which is often quite similar to that obtained in ducks resting quietly on the surface (Butler and Woakes, 1979; 1982a). This contrasts with forced dives in which heart rate falls progressively, usually to very low levels (Butler and Woakes, 1976; 1979; 1982b).

An "immersion reflex" has been suggested to play an important role in divers, although its existence has never been unequivocally demonstrated in birds (Butler and Woakes, 1982a; Mangalam and Jones, 1984).

When a bird or mammal dives, if it is to remain aerobic, it must sustain itself on the oxygen stored at the moment of submersion. That aerobic metabolism is preferable to anaerobic metabolism has been demonstrated by Kooymans et al. (1980). They pointed out that clearing anaerobic metabolites
takes considerably longer than simply replenishing the oxygen stores, and the dive-pause ratio falls off rapidly as the animal begins to rely on anaerobiosis.

If terminating the dive before relying heavily on anaerobic metabolism is adaptive, then it seems likely that diving animals are capable of sensing \( \text{PaO}_2 \) either continuously or at some threshold level. Butler and Woakes (1982b) reported that dive times of tufted ducks (\textit{Aythya fuligula}) whose carotid bodies had been denervated were significantly longer than those of intact animals. When Butler and Woakes (1982b) forcibly submerged tufted ducks, they wrote that the "cardiac response to forcible submersion of the head of tufted ducks was, surprisingly, not greatly affected by denervation of the carotid bodies."

The difference in cardiac responses to submergence in voluntary and forced diving has led to a controversy about the basic concept of a "diving response." It has been proposed that in forced dives the response is a product of, or is accentuated by, fear or stress (Kanwisher et al., 1981; Kanwisher and Gabrielsen, 1985), while in voluntary dives, there is not so much a diving bradycardia as a tachycardia before the dive (Butler and Woakes, 1982a&b). These ideas have been promulgated despite claims that "calm" or "relaxed" ducks respond better (deeper bradycardia) in forced dives (Irving et al., 1941; Folkow et al., 1967), and because some birds submerging voluntarily show little heart rate change.
before, during or even after the dive (Butler and Woakes, 1984; Gabrielsen, 1985).

Nevertheless, the central nervous system probably plays an important role in modulating diving responses, but no one has attempted to study the role of psychogenic and reflexogenic mechanisms in initiating diving responses of diving birds, and to determine how one influences the other. One attempt has been made to quantify the extent of the psychogenic modulation of the diving response in dabbling ducks (Blix, 1985), but this approach was literary rather than experimental. An experimental approach to this problem is difficult. As a start, comparing the diving response in voluntary and forced dives in the same group of animals under a range of conditions, might clarify any relationship between heart rate responses in both types of dive. It is important, however, that any relationship be supported by our knowledge of both the afferent and efferent neural mechanisms involved in the response. To this end, results of a limited investigation of the efferent control of the heart in the duck are reported.

This study was undertaken to investigate the nature of "immersion reflexes" in diving ducks, and an attempt was made to measure their contribution to the cardiac response in restrained and free dives. An attempt was also made to determine whether diving ducks are capable of adjusting dive
time and heart rate in response to altered levels of inspired oxygen, and to examine the role of the baroreceptors in cardiac control during forced and voluntary diving.
Heart rate response to restrained diving

Six redhead ducks (*Aythya americana*) were used to test the role of nasal receptors in the initiation of the diving response. Heart rate was monitored using needle electrodes placed subcutaneously on the left side of the abdomen and the right shoulder. The ducks were forcibly submerged by gently lowering the head into a container of water. This manoeuvre was done carefully to prevent the animals struggling on submersion. Each duck was submerged three times, and a mean heart rate was calculated for that animal. Xylocaine (Lidocaine USP, Astra Pharmaceuticals Canada Ltd., Mississauga, Ontario) in aerosol form was administered into the nares for local anaesthesia of narial receptors. To minimize the possibility of anaesthetizing the glottis, loosely packed cotton wool was placed over the glottis to shield it from the spray. A slip of the cotton protruded out of the corner of the mouth, and the beak was taped closed to prevent the duck swallowing the cotton. Fifteen minutes after Xylocaine application the cotton wool was removed and another 5 minutes allowed before the first of a series of three dives on each animal was performed. Two hours later, after recovery from the application of Xylocaine, the birds were again tested for a diving response.
Another six redhead ducks were tested to determine the effect of high arterial oxygen tension on the initiation of the diving response. A plastic bag was placed over the duck's head, and either air or 100% oxygen was passed through the bag for at least 3 minutes before the animal was submerged. As above, three dives per animal for each condition were recorded.

To elevate the heart rate of ducks before a forced dive, one redhead duck was encouraged to run on a treadmill and 2 others were chased along a corridor. Immediately after exercise ceased, the duck's head was forcibly submerged into a beaker of water for 15 seconds.

Heart rate was determined from the cardiac interval taken at 1, 2, 5, 10, and 15 seconds in the dive. Dive duration was varied to minimize the effect of conditioning (Gabbott and Jones, in preparation), but all dives lasted at least 15 seconds. The data were analyzed using analysis of variance, and significance was set at P < 0.05. All values from this series of experiments are given as means ± S.E.M. and N = the number of animals contributing to the mean.

Heart rate response to free diving

To test the response to voluntary diving, heart rate was obtained telemetrically using EKG transmitters (Narco Biosystems, Downsviow, Ontario, Canada). The transmitter was sterilized with benzalkonium chloride (Zephiran, 1:750,
Winthrop Laboratories, Aurora, Ontario). A midline incision was made in the skin and body wall over the abdomen after anaesthetizing the area by injection of Xylocaine. Bipolar loop electrodes were placed on the pericardium and the transmitter was put in the peritoneal cavity. The peritoneal cavity was then closed with surgical silk. After surgery, 125 mg of Ampicillin (Penbritin, Ayerst Laboratory, Montreal, Quebec) was administered I.M. and the birds were allowed one day to recover before being used in any experiments. The EKG signal was received on a Narco FM-Biotelemetry receiver, stored on magnetic tape, and displayed on a pen recorder. On playback, heart rates were either determined from measurement of the cardiac intervals or by using a cardiotachometer. The ducks were placed on a pond deep enough to allow them to dive for food. This pond was man-made and had a surface area of 3 x 5.5 m. The bottom of the pond was tapered so that the depth of the water ranged from 0.3 - 1.7 m. A platform (2.0 x 1.5 m) and a Plexiglas enclosure (1.0 x 1.5 x 0.8 m high) were placed at the shallow end of the pond. The enclosure had a vertically sliding door to seal it sufficiently so that oxygen levels in the enclosure could be altered. The surface of the water outside the enclosure was covered with netting stretched over wooden frames. Each frame was 1 m². The frames floated on the water and prevented the birds from surfacing anywhere but within the enclosure; however, the birds could lift the netting to take a breath if it became necessary. At the
corner of the pond farthest from the enclosure was a feeding station with a chute through which food could be dropped into a receptacle at the bottom. Nitrogen or oxygen was infused (20 l·min⁻¹) into one side of the enclosure and removed from the opposite side. A fan was mounted inside the box to mix the air. Gas from the box was led to a Beckman F₃ paramagnetic oxygen analyzer which was calibrated with nitrogen and air. The 50% level of oxygen was estimated from flow rate and enclosure volume and should not be taken as precise. The value does, however, represent a high concentration of inspired oxygen.

Most voluntary dives occurred in the period after food was delivered into the feeding chute. To reduce the sympathetic contribution to the increase in heart rate before a voluntary dive, 1.5 mg·kg⁻¹ of propranolol was injected into the muscles of three redhead ducks, and the animals were immediately returned to the pond. Xylocaine blockage of the nasal area was performed as described for restrained animals, and a short, forced dive was done to confirm the efficacy of the blockade before the animals were put back onto the pond. Ducks were reluctant to dive after their nasal area had been blocked with Xylocaine. So few voluntary dives were obtained that the birds had to be "chased" to make them submerge (Butler and Woakes, 1979). This was usually done by banging on the lid of the enclosure with a stick or waggling a net at them.
Pre-dive heart rate was reduced by non-pharmacological means by encouraging 3 redhead ducks to dabble for food. The ducks were placed singly on a 25 cm deep pond with food on the bottom which the ducks reached by dabbling. To simulate the "forced" dive response by unrestrained ducks, 2 redheads were presented with a 1 l beaker of water on the floating platform at the end of the pond. They voluntarily submerged their heads in the beaker to obtain food which covered the bottom.

Four redhead ducks and four lesser scaup diving voluntarily were prohibited from surfacing by lowering a vertical panel at the only point with access to the surface just before the bird was to surface. The panel was removed usually after an additional 10 seconds of diving; however, on one occasion each duck was forced to remain under water until it had fatigued.

Three redhead ducks (two male and one female) were used to test the effect of baroreceptor denervation on the submersion response. All surgery was performed under general anaesthesia (20mg/kg sodium pentobarbital, Somnotol, MTC Pharmaceuticals, Mississauga, Ontario). Entry to the thoracic cavity was gained through the interclavicular air sac. The left baroreceptor nerve was sectioned approximately 1.5 cm distally from the nodose ganglion, and the right nerve was sectioned near the pulmonary vein at a point where the nerve turns ventrally and medially toward the heart. After denervation, the air sac was closed with surgical silk and the
skin was sutured over the repaired air sac. The ducks were allowed one week to recover and then placed on the diving tank. The effectiveness of denervation was tested by injecting 25µg of phenylephrine into the brachial vein while monitoring blood pressure from the brachial artery and noting the presence or absence of a fall in heart rate. Two of the barodenervated redhead ducks were forcibly submerged using the procedure described above, except only three dives per duck were used.

Heart rate was determined from measuring the number of beats over the second before submersion, from the cardiac interval actually at or even just before submergence (1st cardiac interval), and from cardiac intervals after the heart rate stabilized, but not later than 5 seconds after submergence. Data were analyzed by plotting the relationship between pre-dive heart rate and the heart rate which occurred on submersion, or that occurring in the first 2-5 sec diving. Regression analysis was performed on the data using a curve fitting program. When animals were trapped under water, the heart rate was measured one second before and 2-5 seconds after the partition was closed. The significance of any difference (at P < 0.05) between data from chase-induced dives by untreated and Xylocaine blocked ducks was assessed from the statistics for linear regressions on both sets of data. In this series of experiments n = the number of observations and N = the number of animals used.
The effects of various afferent neural inputs on the diving responses in voluntary dives, especially those when animals were prohibited from surfacing, were investigated in eight birds. Baroreceptors were denervated in three redhead ducks, and diving responses were investigated 3 weeks later. Four ducks (2 redhead and 2 scaup) were allowed to breathe oxygen before unrestrained dives by passing pure oxygen through the enclosure, to reduce arterial chemoreceptor stimulation in the dive. Nasal receptors were anaesthetized in 2 redheads using Xylocaine.

Control of heart rate by vagal and cardiac sympathetic nerves

To study the properties of the efferent neural pathway, stimulating electrodes were implanted bilaterally on the cut peripheral ends of the cardiac and vagal nerves of three young White Pekin ducks (*Anas platyrhynchos*; average body mass 1 kg). Ducks were anaesthetized by intramuscular injection of pentobarbital and the sternum was divided in the mid-line to expose the central cardiovascular area. Cardiac and vagal nerves were identified as they coursed towards the heart and were sectioned 1-2 cm from the heart. Loop electrodes, similar to those described by Jones et al. (1982), were threaded onto the distal cut ends of the nerves. The electrodes were connected, one pair from each pair of nerves, to two stimulators via stimulus isolation units (Grass Model PSIU6D; Grass Instruments Corporation, Quincy, Mass., U.S.A.).
The vagus nerves were stimulated using a constant current of 0.5 mA, and the cardiac sympathetic nerves were stimulated at a constant current of 1.0 mA. Increasing vagal current to 1.0 mA caused no further change in heart rate at any stimulation frequency. The stimulus duration was 2.5 ms for both sets of nerves. Heart rates were recorded over a wide range of stimulation frequencies of each pair of nerves (although the frequency of stimulation was always the same for both vagal or both cardiac nerves). These heart rates were plotted against stimulation frequencies of vagal and sympathetic nerves using the method of presentation of Levy and Zieske (1960). At the end of these experiments animals were killed with an overdose of pentobarbital administered intravenously.

In this study, forced-dive refers to submersion of the head only while the animal is under restraint, voluntary face immersion refers to submersion of the head only performed voluntarily by the duck to retrieve food from the bottom of a beaker filled with water, dabbling is the act of upending in the water with head and thorax submerged, voluntary dives are those involving underwater swimming initiated by the animal, chase-induced dives are those in which the animal dived to escape an apparently threatening situation, and trapped-dive refers to the part of the dive following blockade of the only area with access to the surface.
RESULTS

Heart rate response to restrained diving

Heart rate of restrained redhead ducks in the laboratory was between 90 and 110 beats·min⁻¹ (100 ± 3 beats·min⁻¹; N=12). On submergence, heart rate fell rapidly and progressively to a stable rate of 35 ± 4 beats·min⁻¹ (N=12) within 5 seconds (fig. 1a & 1b). Bradycardia occurred rapidly (fig. 2) and was unaffected by breathing oxygen before the dive (fig. 1a), but application of Xylocaine to the narial region virtually eliminated diving bradycardia (fig. 1b & 2). After Xylocaine, heart rate fell to only 80% of the pre-dive rate 15 seconds after submersion (fig. 1b).

The first cardiac interval was usually the shortest in the dive (fig. 2). The duck running on a treadmill had a pre-dive heart rate of over 300 beats·min⁻¹, and heart rate fell on forced submergence, but not as low as in restrained animals (fig. 3). Ducks which were chased and then caught and dived had extremely high pre-dive heart rates (over 400 beats·min⁻¹). Heart rate fell immediately and remained stable for 2-5 seconds after which the rate began falling gradually but quickly over the remainder of the 15 second dive. Only the first 5 second period was used in the present analysis (fig. 3). Unfortunately, it was not possible to obtain reliable estimates of the first cardiac interval in dives involving exercising animals and the regression line shown in
Figure 1. Heart rate response to submersion in diving ducks (mean ± 1 S.E.M.). (a) Response of animals breathing oxygen (open circles) or air (solid circles) before submersion. (b) Response of animals after narial anaesthetization (open circles) compared with untreated animals (solid circles). These animals were breathing room air before submersion, N = 6.
Figure 2. Traces showing heart rates of restrained ducks before, during, and after forced submergence. In the upper trace the duck had breathed air before submergence. The lower trace shows the cardiac response to submergence after the application of Xylocaine to the nares. 'Down' refers to the time at which the beak entered the water and 'up' refers to the time the animal surfaced.
Figure 3. The relationship between pre-dive and dive heart rate taken at the time the heart rate stabilized but before 5 seconds submergence in a restrained dive. The solid circles represent data taken while the animals were immobilized and had not exercised. The open circles represent data taken after the duck was exercised on a treadmill (the two points at 300 beats•min⁻¹ pre-dive) or after being chased (above 400 beats•min⁻¹ pre-dive). The short line with a slope greater than one was drawn from the regression of the first cardiac interval for restrained dives excluding those following exercise.
figure 3 pertains only to forced dives in the laboratory when the animal was strapped to the table.

Both of the barodenervated ducks showed a strong diving response when forcibly submerged. One of the ducks produced dive heart rates in the range of those of intact ducks (dive heart rates at 5 seconds submergence were 28, 32 and 39 beats•min\(^{-1}\)), even when pre-dive heart rate was high (160 to 200 beats•min\(^{-1}\)). The other duck, however, showed a much stronger bradycardia. Immediately upon submergence, an extremely long cardiac interval occurred (the first or second interval was either 24, 26 or 28 seconds long, corresponding to slightly more than 2 beats•min\(^{-1}\)).

**Heart rate response to free diving**

Heart rates varied greatly during voluntary diving and dabbling, but there was a consistent pattern to the responses. Dives were usually performed in a series, and heart rate increased to at least 300 beats•min\(^{-1}\) before the first dive and continued to do so in the pauses between dives so that after 5 or 10 dives, heart rate approached 500 beats•min\(^{-1}\). Before and during diving, heart rates of redhead ducks were always higher (e.g. fig. 4a) than when dabbling (fig. 4b) or retrieving food from the beaker of water (fig. 4c), but in all three situations, heart rate dropped immediately upon submergence. The first cardiac interval in voluntary dives was usually the longest, and heart rates fell to between 100
Figure 4. EKG traces of a redhead duck a) diving voluntarily, b) dabbling and c) immersing its head into a bucket of water to retrieve food. The downward arrow is the approximate point of submergence, and the upward arrow is the point at which the duck surfaced.
a) Diving

b) Dabbling

Voluntary Face Immersion

5 sec
and 140 beats·min⁻¹ (fig. 5). Heart rate at the first cardiac interval was positively correlated with pre-dive heart rate (fig. 5). Heart rate after 2-5 seconds submergence was more strongly correlated with pre-dive heart rate (fig. 6a) and was significantly above that occurring at the first cardiac interval at all pre-dive rates over 250 beats·min⁻¹ (fig. 5 & 6a). β-blockade prevented pre-dive heart rate from exceeding 300 beats·min⁻¹ even in a series of dives, but on submergence both the first cardiac interval and heart rate after 2 and before 5 seconds fell in the same range as those obtained from untreated ducks with low pre-dive heart rates (i.e. before voluntary face immersion or dabling).

In chase-induced dives, the length of the first cardiac interval was highly variable, and was usually shorter than in voluntary dives (e.g. fig. 7b). In one duck, chasing had no apparent effect on dive heart rates compared with those in voluntary dives. In another duck, however, chasing caused a more pronounced fall in heart rate than in voluntary dives. Dive heart rates after chasing were 10% below those in voluntary dives, and this difference was significant. The regression lines describing the relationship between dive and pre-dive heart rates in chase-induced dives in 3 ducks were compared with those obtained from the same animals after blockade of the nasal area with Xylocaine. In all three cases, the elevations but not the slopes of these regression lines were significantly different, with the heart rates from
Figure 5. The relationship between the pre-dive heart rate and the first cardiac interval on submergence. The broken line is the regression on these data. Combined data from four ducks after β-blockade (open circles) and untreated (closed circles) In this and all other similar figures the line through the origin represents the line of identity.
\[ Y = 68 + 0.16 \pm 0.05X \pm 18 \]
Figure 6. Relationship between pre-dive and dive heart rate in dives by unrestrained ducks. The line passing through the origin represents equal pre-dive and dive heart rates. (a) Combined data from 5 ducks diving voluntarily (solid triangles), β-blocked with propranolol (open triangles), untreated. (b) Data from chase-induced dives by a single female redhead after local anaesthesia of the nares (solid circles), untreated (open circles). Data for propranolol blocked dives for this duck are not identified by use of a separate symbol but are included in the regression analysis. The broken line is the regression line for voluntary dives by this animal with no nasal blockade. The regression equations include the 95% confidence limits of the slope and the standard error of estimate.
Figure 7. EKG trace obtained from unrestrained ducks submerging voluntarily. (a) A diving duck diving voluntarily and (b) a chase-induced dive. The downward pointing arrow indicates the approximate time at which the beak entered the water, and the arrow pointing up indicates the approximate time the animal surfaced.
A) B)

Athyra americana
Xylocaine treated ducks being above those from untreated animals. In the duck shown in figure 6b, dive heart rate was elevated after Xylocaine blockade by 15 to 30% depending on the pre-dive heart rate. In contrast, in another duck, the elevation in dive heart rate was 10%, regardless of pre-dive heart rate. The third duck showed an elevation in heart rate of 20 to 25% over a much more restricted range of pre-dive heart rates. The data were not combined, and figure 6b is included to demonstrate that chasing and nasal blockade account for a small portion of the dive response even in that animal where these factors had the greatest effect. A few voluntary dives were obtained from two of the ducks after nasal blockade, and these indicated less elevation in dive heart rates compared with voluntary dives before nasal blockade to those obtained in chase-induced dives.

Changing the level of oxygen in the air breathed before diving affected dive duration (fig. 8a), but had no effect on heart rate after 2-5 seconds submergence (fig. 8b). Dive duration increased as the concentration of oxygen in the inspired air increased. The increase was linear at oxygen levels between 10 and 15%, with a coefficient of determination ($r^2$) of 0.98 (fig. 8a). At oxygen levels below 10%, all diving ceased, even if food were withheld for one day before the trial. At oxygen levels above 15%, dive time increased more slowly with an increase in oxygen concentration (fig. 8a); however, no correlation occurred between the dive heart
Figure 8. (a) Relationship between inspired oxygen and dive time in voluntary dives. The numbers in parentheses above or below the points are n and N. (b) The range of heart rates obtained between 2 and 5 seconds into voluntary dives when the animals breathed air, low or high oxygen concentrations before diving. The bar at 21% oxygen represents the range of heart rates of 42 dives from 5 ducks with the greatest density of points occurring at 190 beats per minute.
rate and the level of oxygen breathed before the dive, and the relationship between dive and pre-dive heart rate remained unchanged (fig. 9a). Dive heart rates after breathing 50% oxygen were in the same range as those obtained after breathing air or 13-16% oxygen (fig. 8b & 9a).

Baroreceptor denervation caused a small but significant depression of dive heart rate compared with that of intact animals (fig. 9b), heart rate being 5 to 20% lower than intact animals.

Preventing access to the surface caused a pronounced fall in heart rate when the duck returned to surface in the enclosure at the end of a voluntary dive. Heart rate fell immediately after the entrance to the chamber was blocked and this new rate was either maintained for the rest of the enforced submergence or slowly increased (fig. 10b). Heart rate two seconds after blockade was correlated with the rate immediately before blockade, and although the heart rates of restrained ducks before and after submergence were often lower than those of unrestrained ducks before and after access to the surface was prohibited, there was considerable overlap of the dive:pre-dive relationship (fig. 3 & 11). This overlap in the relationship was unaffected by β-blockade, baroreceptor denervation, or anaesthetization of nasal receptors with Xylocaine. In two scaup, breathing 100% O₂ before the dive had no effect, whereas in another two scaup and one redhead whose baroreceptors were denervated, dive heart rate was not
Figure 9. (a) The relationship between pre-dive and dive heart rate after breathing high or low levels of oxygen. The solid circles represent dives after breathing 14-16% oxygen. The open circles are from dives after breathing air containing less than 14% oxygen, and the solid triangles after breathing air containing 50% oxygen. The line through the data is the regression line from figure 6a. (b) The relationship between pre-dive and dive heart rate after bilateral denervation of the baroreceptors. The solid circles are from non-β-blocked dives, and the open circles are from dives following β-blockade with propranolol. The broken line is from figure 6a, and the solid line is the regression of these data.
Figure 10. EKG traces of a redhead duck a) forcibly submerged while under restraint, b) prohibited from surfacing during a free dive and c) trapped as in b, but the duck had breathed oxygen before the dive. The point marked "turn" is the point at which the duck turned to return to the enclosure.
A) Forced Dive
   Restrainted

B) Forced Dive
   Unrestrained

C) Forced Dive
   Unrestrained
   After Oxygen

Dive Surface

Forced Dive
Prohibited

Turn

10 sec
Figure 11. The relationship between pre- and "trapped" heart rates when surfacing was prohibited during a dive. The solid circles represent points for intact animals having breathed air before the dive. The open circles represent points taken from ducks that had breathed oxygen before the dive. The solid triangles are from barodenervated ducks, and the open triangles are from ducks whose nares had been anaesthetized with Xylocaine. The solid line is the regression of the first cardiac interval from voluntary dives (figure 5), and the broken line represents the stabilized rate at two seconds submergence (figure 6a).
stable (fig. 11). In these birds, heart rate rose suddenly soon after the birds turned to return to the enclosure (fig. 10c) and when surfacing was prevented heart rate fell but not as low as before. In fact, heart rates after blockade fell into the range of those in voluntary dives made from similar starting heart rates (fig. 11).

With each duck, once after breathing air and once after breathing oxygen before the dive, the entrance to the enclosure was not re-opened. Animals swam around the pond although wing propulsion replaced leg propulsion after 40 sec or so under water. Eventually all activity ceased and the ducks floated up under the netting, covering the pond, and breathed. Heart rate remained low throughout these manoeuvres except when the breath was taken. The total period spent under water until activity ceased was around 60 seconds and this was extended by 10 seconds, on average, if the ducks had breathed oxygen before the dive.

Control of heart rate by vagal and cardiac sympathetic nerves

Heart rate was 283 ± 28(S.D.; n=12) beats·min⁻¹ after bilateral section of the vagal and cardiac nerves. Interestingly, this was also the heart rate observed in 2 redhead ducks after pharmacological blockade of cardiac and vagal nerves (propranolol, 1.5 mg·kg⁻¹ and atropine, 2.5 mg·kg⁻¹). Bilateral stimulation of the vagal nerves resulted in a rapid fall in heart rate, a stable rate usually being
achieved within 1 or 2 seconds (fig. 12). Restoration of pre-stimulation heart rate was equally rapid when stimulation was stopped. In contrast, heart rate only rose slowly in response to bilateral stimulation of the cardiac nerves. Usually it took 30 seconds or so for heart rate to stabilize in response to the maximum stimulus frequency used (8 Hz). When stimulation stopped heart rate fell slowly and reached pre-stimulation levels within 20 to 30 seconds. Heart rates versus stimulation frequencies of vagal and sympathetic nerves are shown in figure 13, and the surface in figure 13 describes all possible heart rates that can be produced by any combination of sympathetic and vagal stimulation. The stimulation frequencies used may not reflect the activity on the nerves of an intact animal, but the shape of the curves will not be altered, and the heart rate at 40 Hz vagal and 8 Hz sympathetic stimulation represent the cardiac response to maximal activity of these nerves.
Figure 12. The heart rate response to bilateral stimulation of the distal cut ends of the a) vagus and b) cardiac sympathetic nerves. The horizontal bar represents the duration of the stimulus.
Figure 13. The relationship of heart rate to bilateral stimulation of the distal cut ends of the vagus and cardiac sympathetic nerves. The figure was drawn through the points by approximation, not by regression. See the text for an explanation of points A, B, C, D and E.
DISCUSSION

The present results have revealed some interesting insights about the variability in cardiac responses in forced and voluntary dives by free and restrained ducks. The main question is whether any genuine relationships exist between heart rate in all these types of dives, and whether these relationships are supported by our knowledge of the efferent and afferent neural mechanisms affecting cardiac control.

Heart rate fell immediately on forcible submergence, and giving the duck 100% oxygen to breathe before submergence had no effect on the bradycardia in the first 15 seconds of the dive, nor did denervation of the baroreceptors. Butler and Woakes (1982b) had shown that carotid body denervation did not alter the onset of bradycardia in tufted ducks, when forcibly submerged.

Anaesthetization of the narial region in diving ducks completely eliminated diving bradycardia in forced dives, performed with care; however, if the animal struggled, heart rate was lower than that just before the struggle. Diving mammals, such as seals and muskrats, also show a rapid heart rate response to forced diving which is eliminated by neurotomy or anaesthesia of the facial or narial region (Dykes, 1974; Drummond and Jones, 1979). Hence, it appears that rapid bradycardia in forced dives is likely to be associated with a "nasal reflex" (Jones, 1981).

Diving ducks showed marked changes in heart rate during
every voluntary and chase-induced dive, even after breathing air with 50% oxygen in the pre-dive period. This confirms the observation of Butler and Woakes (1982b) that after carotid body denervation heart slowing in voluntary dives by tufted ducks was little changed from that of intact animals in the early part of submergence. That is, information from carotid bodies is not ignored in longer dives. Tufted ducks with their carotid bodies denervated have a mean dive duration 3 seconds longer than intact ducks, and a maximum dive time 6 seconds above that of intact animals (Butler and Woakes, 1982b). Furthermore, heart rate at the end of a dive is higher in ducks with denervated carotid bodies than in intact ducks (Butler and Woakes, 1982b). In this study, dive time increased by 2 seconds following oxygen loading. It is likely that the dive is terminated not by oxygen shortage, but probably for behavioral reasons.

In free dives, unlike in restrained dives, nasal blockade with Xylocaine did not greatly affect heart rate after 2-5 seconds submergence. Stimulation of nasal receptors in free diving appeared to cause between 10 and 30% of the heart rate adjustment. Heart rate fell lower in chase-induced than voluntary dives in two of the three ducks, but the results suggest that the contribution from nasal receptors is similar in both voluntary and chase-induced submersions. Denervation of the baroreceptors caused an apparent, although slight, enhancement of the dive response, but these three ducks were
not tested before denervation; therefore, it is not certain where the dive heart rates would fall if these ducks had intact baroreceptors. Furthermore, the data on heart rates obtained in voluntary dives after breathing various levels of oxygen confirm that chemoreceptors have little effect on heart rate adjustments up to 5 seconds after submergence. Consequently, nasal receptors, baroreceptors and chemoreceptors combined only account for a minor component of the initial cardiac responses to voluntary submersion.

There is no doubt that in free dives, other inputs predominate in causing the cardiac responses and are not displayed in forced dives. This supports claims of "anticipation" of the dive response. Butler and Woakes' (1976) original claim for "anticipation" was compromised somewhat by their earlier statement that "the initial bradycardia occurs just as the animal dives and not before" (Woakes and Butler, 1975). More recent data, however, linking cine films of submersion behavior to telemetric recordings of heart rate, have shown that lengthening of the cardiac interval occurs before the nasal area contacts the water (Butler and Woakes, 1982a).

Regions of figure 13 which appear to pertain to intact animals can be identified on the surface describing heart rates resulting from combinations of vagal and sympathetic stimulation. For instance, maximal sympathetic activity in the absence of vagal stimulation gave heart rates of 500
beats·min⁻¹ (point A in figure 13), which was the highest rate observed before voluntary dives. β-blockade with propranolol gave heart rates around 300 beats·min⁻¹ just before voluntary dives, and this is indicated by the point B on figure 13. Finally, before restrained dives, the heart rate was around 100 beats·min⁻¹, and since β-blockade did not lower heart rate further (tested in two ducks), cardiac efferent control in these animals is likely to be described by point C or lower on figure 13. If sympathetic activity does not decrease in a dive (it cannot when pre-dive rates are represented by B & C), then the relative increase of vagal activity required to cause the diving heart rates observed must be similar in voluntary dives, represented by points A and B, and in forced dives represented by point C. Specifically, an increase in vagal activity of about 20 Hz will give the dive heart rates observed in voluntary dives, with and without propranolol, and in forced dives.

In light of the above suggestion, the cardiac responses in all dives, which, might represent 20 Hz vagal activity, were re-investigated. Pre-dive heart rate was plotted against dive heart rate in (1) voluntary dives and dabbles before and after propranolol, (2) forced dives by restrained animals at rest and after exercise and (3) trapped dives by unrestrained animals. Regression analyses were performed on these data and relationships were established for each group and all groups combined. Individually, and in combination, a single linear
relationship could be fitted to a plot of dive heart rate against the logarithm of pre-dive heart rate (fig. 14). Of course, a diminution in sympathetic activity in dives would confound this argument, especially when voluntary dives are made from high heart rates. However, Butler and Woakes (1982a) showed that even when pre-dive heart rate was as high as 400 beats·min⁻¹ in ducks diving after atropine, no change in heart rate occurred, implying that sympathetic cardiac nerve activity is unaltered in dives.

A 20 Hz increase in vagal activity in forced dives by restrained ducks will result in maximal vagal activity. In voluntary dives, the first cardiac interval is usually the longest and could also represent the heart rate at maximal vagal activity, although there may not be sufficient time for full expression of the bradycardia because as can be seen in figure 12, neither the first nor second cardiac interval following vagal stimulation is the longest that was achieved for that stimulation frequency. Nevertheless, it might be expected that these points would be related on a plot of dive:pre-dive heart rate, but it is difficult to predict what form the relationship would take. This relationship, however, could be similar to that describing the influence of sympathetic activity on heart rate at maximal vagal activity (fig. 13). This relationship is illustrated by the curve DE in figure 13.
Figure 14. The relationship of the logarithm of pre-dive (or pre-trapped) heart rate and dive (or trapped) heart rate for all categories. The solid circles represent restrained dives including those following exercise. The open circles represent heart rates for all trapped dives. The solid triangles are from all voluntary dives including β-blocked dives, and the open triangles are from dabbles and voluntary face immersions.
It seems unlikely that a single afferent mechanism could provide the necessary vagal activity in all types of dive if only because vagal activity may be maximal, although declining rapidly, at the start of all voluntary submergences whether dives or dabbles. Surprisingly, Butler and Woakes (1979) claimed that the tufted duck showed no heart rate changes when freely dabbling; yet, 9 out of 10 of the dabbling episodes seen in their figure 5a are associated with rapid changes in heart rate, and in three cases, these changes are greater than 100 beats·min⁻¹. Some of the heart rates associated with dabbling and emerging in the tufted duck resemble those depicted in figure 4(b) of this study for a redhead duck dabbling voluntarily. The termination of hyperventilation at the onset of a dive or dabble (Butler and Woakes, 1976; 1979) could enhance vagal outflow and reinforce an increase in vagal activity concomitant with the onset of submergence, giving a prolonged initial cardiac interval. There is no pre-dive tachycardia or hyperventilation in forced dives, and in these dives, heart rate declines rapidly and progressively. The description of the first cardiac interval in free and forced dives by two separate linear regression equations seems entirely appropriate.

It seems plausible for a common afferent mechanism to produce a similar level of vagal activity in all types of dive. Unfortunately, no support for such a mechanism can be derived from this investigation of the role of afferent
reflexogenic neural mechanisms in the diving response which showed that all of the cardiac response in restrained dives could be attributed to nasal receptors, but nasal receptors account for only between 10 and 30% of the response observed in voluntary or chased-induced dives. Hence, another mechanism must be involved in voluntary dives, but this cannot be peripheral chemoreceptors because breathing elevated or reduced levels of oxygen in the air before diving had no effect on the dive:pre-dive heart rate relationship up to 5 seconds after submergence. So, although chemoreceptors undoubtedly influence heart rate in longer dives (Butler and Woakes, 1982b), any influence is not expressed in the first 5 seconds of a dive.

The animals' response to being trapped is of considerable interest. As soon as the duck became aware that it was not going to be allowed to surface, heart rate fell to levels which would have been seen in either a forced or voluntary dive depending on the heart rate immediately before being trapped. A similar response was described by Butler and Woakes (1982a) in tufted ducks, although they described it as "progressive", as opposed to the sudden fall seen in these diving ducks. This then may be an expression of the "classical" dive response in unrestrained diving, but the mechanisms involved in generating this response are unknown. If the response to being trapped were to conserve oxygen, thereby prolonging dive time, then these animals certainly
should be able to extend dive time beyond 60 seconds, especially in light of that Dewar's (1924) report that a duck of similar size (Aythya marila) had a maximum dive time of 49 seconds in the wild. Even after oxygen loading, redhead ducks could extend dive time by only 10 seconds. It seems likely that anaerobic byproducts accumulating in the muscles having reduced blood flow are limiting dive time, not total oxygen stores.

The absence of any major reflexogenic influences on heart rate in voluntary dives suggests that psychogenic influences are much more likely to be expressed in free than in restrained dives in diving ducks. For instance, psychogenic influences affect dive heart rate both initially, and after 2 to 5 seconds submergence, through their influence on the pre-dive rate. Anticipation of the dive response, described in voluntary dives by Butler and Woakes (1976, 1982a), also implies a profound psychogenic influence. Finally, that heart rate can immediately drop when the normal diving pattern is disrupted, in unrestrained animals, suggests that integration occurs well above the brainstem level in free dives. These conclusions conflict with those obtained from studying restrained and free head submersions by dabbling ducks (Kanwisher et al., 1981; Blix, 1985), in that psychogenic influences affect the former and not the latter. Obviously this conflict will only be resolved by further research.
SECTION 2

The Heart Rate Response to Diving and Dabbling in a Non-diving Duck

INTRODUCTION

Ducks are commonly divided into two groups, divers and dabblers, but all dabbling ducks dive when they are very young, and even adult dabbling ducks dive for food far more frequently than is generally acknowledged. Foraging dives by mallards (*Anas platyrhynchos*), black ducks (*A. rubripes*), African black ducks (*A. sparsa*), pintails (*A. acuta*), Bahama pintails (*A. bahamensis*), shoveler (*Spatula clypeata*), New Zealand brown ducks (*A. aucklandica chlorotis*), gadwalls (*A. strepera*), cape teal (*A. capensis*), gray teal (*A. gibberifrons*), wood ducks (*Aix sponsa*) and mandarin ducks (*A. galericulata*) have been observed (Bourget and Chapdelaine, 1982; Chapman et al., 1959; Dean, 1950; Kear and Johnsgard, 1968; Kutz, 1940; Mylne, 1954). They have been reported diving to depths greater than 3 meters (Kutz, 1940), having mean dive times of approximately 5 seconds (Bourget and Chapdelaine, 1982; Dean, 1950), and a maximum dive time of 10 seconds (Bourget and Chapdelaine, 1982; Chapman et al., 1959). Bourget and Chapdelaine (1982) suggested that dabbling ducks spending the winter in northerly regions may be forced to dive for food because the shallow water areas are more susceptible to freezing over during cold spells.
The cardiac response to forcible submergence of a restrained dabbling duck is characterized by a gradual slowing of the heart and an increase of peripheral resistance (Butler and Jones, 1982). This response is virtually eliminated by sectioning the carotid body nerve (Jones and Purves, 1970), indicating that it is brought on by the progressive hypoxemia and hypercapnemia associated with the breath-hold dive. Diving ducks, however, develop an immediate bradycardia when forcibly submerged, and although the bradycardia is not affected by carotid body denervation (Butler and Woakes, 1982b), it is eliminated by anaesthetizing the nasal receptors with a local anaesthetic (figure 1, section 1).

Mangalam and Jones (1984) and Jones et al. (1982) showed that breathing 100% oxygen before a dive greatly reduced the cardiac response to enforced submergence of the Pekin duck. When pre-dive heart rate is lower than 150–190 beats min⁻¹, virtually all of the bradycardia in dabblers during forced dives, results from stimulation of peripheral arterial chemoreceptors (Jones & Purves, 1970; Jones et al., 1982).

An immersion reflex has been claimed to exist in dabbling ducks, which is more obvious the higher the pre-dive heart rate (Andersen 1963a,b,c; Feigl & Folkow 1963; Folkow et al., 1967; Rey 1971; Butler & Jones, 1982; Blix & Folkow, 1984; Blix, 1985). However, Jones et al. (1982) suggested that the initial rapid fall in heart rate in dabblers may not necessarily mirror a similar change in cardiac output. In
fact, cardiac output is most likely to be unchanged in this period. Furthermore, they also showed that there was a strong relationship between changes in heart rate in the first few seconds of submergence (y) and the pre-dive heart rate (x). The relationship was expressed by the formula \( y = x - 188 \) for pre-dive heart rates above 188 beats \( \text{min}^{-1} \), so if pre-dive heart rate were below 188 beats \( \text{min}^{-1} \), there was no change in heart rate early in the dive. This or a similar relationship also appears to apply to the Canada goose during voluntary dabbles (Kanwisher et al., 1981). Heart rate fell rapidly, from rates in the range of 230-290 beats \( \text{min}^{-1} \) before submergence, to a rate in the range of 140 to 150 beats \( \text{min}^{-1} \). Therefore, when heart rates are very high, the potential exists for rapid cardiac adjustments on submergence even though a specific "immersion reflex" may not exist.

This study was undertaken to investigate the nature of the cardiac adjustments in dabbling ducks (\textit{A. platyrhynchos}) dabbling and diving voluntarily, and the underlying mechanisms which bring about these adjustments.
METHODS AND MATERIALS

Two male mallards and one female Pekin duck (Anas platyrhynchos) were used to record the cardiac response to voluntary dabbling. Five mallards (2 male and 3 female) were used in the voluntary diving observations.

An EKG transmitter (Narco Biosystems, Downsview, Ontario) was placed in the peritoneal cavity as described in the previous sections. All surgery was performed under local anaesthesia (2% Xylocaine, Astra Pharmaceuticals, Mississauga, Ontario). The birds were allowed one day to recover before being placed on the water. For dabbling, the ducks were placed in a 0.8 X 2.8m Fiberglas tank with water 0.3m deep. Food was scattered on the surface and quickly sank. The EKG signal was received on an FM-1100-7 biotelemetry receiver (Narco Biosystems), recorded on magnetic audio tape and displayed on a pen recorder. Only dabbles lasting longer than 3 seconds were used in data analysis. This was done to ensure that the heart rate was not changing at the 2 second mark as the result of any activity associated with surfacing.

For diving, four of the mallards were placed one at a time in a 0.8 X 1.8m Fiberglas tank with water 1.0m deep. Food was scattered on the surface and quickly sank. The condition of the birds was monitored, and if a bird would not dive, it was removed before excessive weight loss. For the first week, the birds ate only that food which could be reached before the food sank. If the bird did not dive after
one week, a small amount of food was placed on the platform daily. The amount of food was kept small to maintain the health of the animal and the hunger drive. The duck was removed from the tank if diving had not begun by the end of the second week.

The duck's behavior was recorded on a Canon VR-40 VHS video recorder using a JVC GX-N4 camera. Once diving had begun, an EKG transmitter was implanted, and the signal was placed on one of the audio tracks of the video recorder. The video tape was reviewed at high speed. When a dive was observed, the tape was played at normal speed, and the audio signal was displayed on a pen recorder.

To test the effect of sympathetic influences on the pre-dive and dive heart rates, a β-blocker was used, but because the diving behavior is easily disturbed, and the effects of this disturbance can last for several hours, a β-blocker having a long half-life was required. Propranolol has a half-life of 2 hours in mammals, whereas nadolol (Corgard, Squibb Montreal, Quebec), a non-selective β-blocker, has a half-life of 24 hours in mammals (Gilman et al., 1980). One of the mallards was given 4 mg of Nadolol orally on the first day followed by 2 mg daily for 2 additional days. The duck was then observed for another week without β-blockade after which the drug was again administered using the same protocol. Only dives lasting longer than 4 seconds were used to avoid changes in heart rate associated with surfacing. The
transmitters were removed after data collection.

The three mallards and one additional mallard were used to study the effect of baroreceptor denervation on the cardiac response to voluntary diving. Barodenervation was done using the procedure described in section 1. Diving usually resumed within a week after barodenervation. The EKG transmitter was then implanted using the same procedure as for intact ducks. Chronic barodenervation is usually accompanied by high heart rates (Jones et al., 1983), and nadolol was used to increase the range of pre-dive heart rates in this group. One of the ducks was used as a sham. The nerves were exposed and a loop of thread was placed around each nerve. One end of each thread was anchored to the skin high in the neck after the wound was closed. After heart rate data were collected for this condition, the duck was again anaesthetized, and the thread was slowly pulled through the wound sectioning the nerves. The duck was returned to the tank on the following day and data collection resumed. The effectiveness of denervation was tested in each duck by injecting 25µg of phenylephrine into the brachial vein and noting the presence or absence of a fall in heart rate. The animals were then killed with a lethal dose of sodium pentobarbital and baroreceptor nerve section checked post mortem. One of the four mallards underwent a bradycardia following administration of the vasoconstrictive drug. The post mortem inspection revealed that only the left baroreceptor nerve was sectioned.
This animal is reviewed separately.

Pre-dive heart rate was determined one second before submergence, and dive heart rate was determined two seconds after submergence. The data are shown as least squares regressions with the 95% confidence limits of the slope and the standard error of estimate. The slopes were tested for significance (P < 0.05) against slopes of either 0 or 1 using the t-statistic.
RESULTS

Heart rate response during dabbling

When dabbling voluntarily, heart rate of mallards did not change noticeably, although the slope of the regression line is significantly different from unity (fig. 15 & 16a). The large changes were rare, and were not always related to the moment of submergence or surfacing. The mean duration of submersion was 3 ± 1 (S.D.) seconds with a maximum of 9 seconds; however, the large majority of dabbles were less than 3 seconds, and were therefore not used in the analysis.

Heart rate response during diving

The time required to make a return trip to the bottom of the diving tank was approximately 2 seconds. Dive time increased during the first week of diving from 2 seconds to 6 seconds, and the combined mean of all ducks after the first week was 6.2 ± 2.1 seconds with a maximum dive time of 11.6 seconds.

When voluntarily diving, the ducks showed little or no anticipatory increase in heart rate before the dive even if the pre-dive heart rate was low (fig. 16b). The heart rate was lowest before the first of a series of dives, and increased progressively between subsequent dives. This increase was positively correlated with dive duration, and negatively correlated with the length of the intervening
Figure 15. The relationship between pre-submergence and submersion heart rate during voluntary dabbling when submersion time exceeded three seconds. The broken line represents the regression of these data. In this and all other similar figures, the line passing through the origin represents the line of identity.
DIVE HEART RATE (min$^{-1}$)

PRE-DIVE HEART RATE (min$^{-1}$)

$Y = 23 + 0.90 \pm 0.08X \pm 22$
Figure 16. EKG and heart rate traces from mallards a) dabbling and b) diving. The broken line at 250 beats·min$^{-1}$ represents the typical heart rate at two seconds into the dive. There is no typical heart rate for dabbles.
pauses. The range of pre-dive heart rates can be seen in figure 17a. In one duck, the mean dive time was 7.7 ± 1.2 seconds, the dive:pause ratio was one, and pre-dive heart rate rapidly increased to 500 beats•min⁻¹ during a series of dives. The duck represented by the data in figure 18 (solid circles) had a low dive:pause ratio (<<1). The heart rate of this duck immediately after a long dive (>9 seconds) was usually above 400 beats•min⁻¹, but the duck rested at the surface for more than 10 seconds and heart rate decreased, consequently the rate before the next dive was never higher than 350 beats•min⁻¹.

The heart rate in the early stages of the dive was nearly the same regardless of the pre-dive rate. The combined data from 3 mallards (50 dives each) show that dive heart rate was approximately 250 beats•min⁻¹ (fig. 17a). When the data for each animal were analyzed separately, the regression lines of 2 of the 3 ducks were not significantly different from zero, but the third as well as the non-β-blocked dives of the duck shown in figure 18 were significantly different from zero. The regression lines of all ducks, however, cross the line of identity. Obviously heart rate increased in dives when the pre-dive heart rate was low, and decreased in dives when the pre-dive heart rate was high (fig. 16b, 17a & 18). Following β-blockade with nadolol, not only was the pre-dive heart rate low, but the dive heart rate was significantly depressed
Figure 17. The relationship between pre-dive and dive heart rate during a) voluntary dives of three intact mallards (solid circles) and one sham operated mallard (open circles), and b) voluntary dives of barodenervated ducks without β-blockade (solid circles) and following β-blockade with nadolol. The solid lines are regressions of the data, and the broken line is the regression of the data after adding 30 beats·min⁻¹ to the dive heart rate of β-blocked dives (see text for an explanation).
Figure 18. The relationship between pre-dive and dive heart rate from one mallard intact (solid circles), after β-blockade with nadolol (open circles) and after denervation of the left baroreceptor nerve only (solid triangles). These data are from the same bird and are not included in the previous figures.
compared with dives in which sympathetic activity was not antagonized (fig. 18).

Baroreceptor denervation altered the relationship between pre-dive and dive heart rate (fig. 17b). The regression line now approaches, although is significantly different from, the line of identity (fig. 17b). The duck that had only one baroreceptor nerve sectioned (fig. 18) gave a response between that of intact ducks and those ducks whose baroreceptor nerves were sectioned bilaterally (fig. 17b).
DISCUSSION

Mallards appear to be regulating heart rate during the early stages of voluntary dives, with the regulated rate being approximately 250 beats\textsuperscript{-1} min\textsuperscript{-1}. This cardiac response to voluntary diving is nearly eliminated by bilaterally sectioning the baroreceptor nerves, but not by unilateral sectioning. Information about cardiac rhythm is carried in the baroreceptor nerves (Arndt et al., 1977), but there is no evidence that animals use this information to adjust heart rate. In any event, the change in heart rate must be vagally-mediated because the cardiac sympathetic nerves are incapable of bringing about rapid changes in heart rate (figure 12, section 1); therefore, not only is the amount of change of vagal activity variable, but the direction of change also depends on the pre-dive rate.

When propranolol was given to diving ducks, dive heart rate fell to values in the same range as that of non-β-blocked dives having similar pre-dive heart rates (figures 5 & 6a, section 1); however, when given nadolol, dive heart rate was approximately 30 beats\textsuperscript{-1} min\textsuperscript{-1} lower than when given propranolol (personal observations). Figure 18 also shows dive heart rates approximately 30 beats\textsuperscript{-1} min\textsuperscript{-1} lower after administration of nadolol than would be expected if sympathetic antagonism were the only effect of this drug. This calls into question the points representing β-blockade in figure 17b and the slope of the regression. A new regression can be generated by
adding 30 beats min⁻¹ to dive heart rates of β-blocked dives (indicated by the broken line of fig. 17b). Both of the slopes are significantly different from one. Obviously, other factors are responsible for the remainder of the cardiac adjustments to submergence (e.g. those relating to exercise or the cessation of breathing).

As with mallards, penguins also showed no anticipatory tachycardia before the dive, but unlike mallards, penguins showed little or no cardiac adjustments to voluntary submergence (Butler and Woakes, 1984). If the pre-dive tachycardia seen in all diving ducks thus far examined is in anticipation of a high level of exercise, it certainly cannot explain the absence of such a tachycardia in mallards because dabbling ducks are more buoyant than diving ducks (Dehner, 1946 – in King, 1966), and have to work harder to dive. Two of the five ducks used in the present study seemed unable to submerge fully without the aid of the wings, but once under water, they switched to leg propulsion.

The cardiac response to voluntary dabbling exhibited by the mallard is puzzling. On the one hand, arterial chemoreceptors are responsible for the cardiovascular adjustments to forced diving, and it is surprising that little or no cardiac adjustment occurred during short periods of head submersion. On the other hand, however, the evidence presented by Jones et al. (1982) showed clearly that, when the duck's heart rate was high just before a forced dive, heart
rate fell rapidly to approximately 188 beats·min⁻¹. The rapid changes in heart rate that occur on forced submergence do not appear when the duck freely dabbles. Ventilation was not recorded, but if minute ventilation is similar when pre-dive heart rate is high in forced and voluntary head submergence, then the rapid bradycardia may not be reflexogenic. Since that study, more data have been analyzed, and the equation is now y = 1.01 x - 146. Evidence from voluntarily dabbling geese (Kanwisher et al., 1981) and Pekin ducks (Gabrielsen, 1985) indicates changes in heart rate to approximately 150 beats·min⁻¹, with heart rates of 200-300 beats·min⁻¹ during the short breathing period. Observations from this study reveal some change in heart rate during the dabble, but these were seen when the period of submersion was short (less than 3 seconds), and when the dabbles occurred in rapid succession (surface time usually less than 2 seconds). In any event, heart rate did not fall to 150 beats·min⁻¹ with any regularity during these dabbles, and as can be seen from the last trace of figure 16a, the presence of a bradycardia was unpredictable, and the rate of fall of cardiac frequency was variable.

It is clear from this study, and from other studies of voluntary diving in birds that few generalizations can be made regarding the cardiovascular adjustments to submergence. Moreover, our knowledge of the adjustments to submergence is limited to heart rate. Hypotheses about other aspects of the
cardiovascular system are based on assumptions, such as the maintenance of arterial pressure. If arterial pressure is being maintained during the dive, it is likely that the system is responding to and correcting for changes in pressure brought about by alterations in total peripheral resistance, and since mallards are leg-propelled divers, the change in total resistance may be largely the result of modifications in hindlimb perfusion. There is no evidence, however, for or against arterial pressure being maintained. If not, heart rate itself is the likely regulated variable.
GENERAL DISCUSSION

Diving birds foraging in nature spend a considerable amount of time under water. Pedroli (1982) stated that tufted ducks may spend as much as 5.9 hours out of 24 under water, and that nearly all of this foraging activity takes place within a 14 hour period that spans night time. Siegfried (1974) reported that female scaups spent 5.5 hours under water during a 12 hour period between 0800 and 2000. This represents 42.1 and 45.8% for tufted ducks and scaups respectively of the total foraging period being spent under water. Diving birds, however, do not dive continually in the foraging period. Long pauses occur among bouts of diving. In a series of dives interrupted by no more than 30-second pauses (a diving bout), it is clear that the animals are spending far more time under water than at the surface. A lesser scaup (Aythya affinis) performed a series of 42 dives over a period of 20 minutes during which 72% (14.4 minutes) of the time was spent under water (unpublished observations). Most diving ducks spend about 63% of a diving bout under water (i.e. a dive-pause ratio of 1.7); however oldsquaw (Clangula hyemalis) spend more than 80% of their diving bouts under water (Dewar, 1924). It is not certain how long the bird can maintain this high dive-pause ratio.

In an effort to understand the behavior of the lesser scaup, a Log-Survivor plot (Fagan and Young, 1978) was prepared from over 200 dives and pauses of 17 birds diving in
the wild (fig. 19). The abscissa represents the duration of the event, and the ordinate displays the logarithm of the percent number of events greater than a given duration. This function allows predictions because the slope of any segment of the function is proportional to the probability that the event will terminate. The curve shows that the probability that a dive will terminate in less than 10 seconds is near zero, but the probability is very high above 20 seconds. There is nothing especially revealing in this curve; however, the figure for between-dive pauses shows some interesting characteristics. The greatest probability that a pause will end and a dive begin occurs between 6 and 10 seconds, after which the probability decreases and is near zero above 30 seconds. The specifics of this figure do not apply to all diving birds, and will not even apply to the same species if that bird is diving in very deep water 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. Dive-pause ratios less than one were recorded for lesser scaup and redhead ducks on the man-made pond used in this study. It is likely that the cardiovascular adjustments observed in this study reflect the adjustments occurring in naturally diving ducks in the wild; however, observations on dive-pause ratios underscore the need to record from animals diving in a more natural setting.
Figure 19. Log-Survivor plot of 202 dives and pauses of 17 lesser scaup diving in the wild. The ordinate shows the logarithm of the percent number of dives greater than the time shown on the abscissa.
Plasma lactic acid concentration of tufted ducks swimming on the surface increased by more than two-fold when swimming velocity increased from 0.3 to 0.7 m·sec\(^{-1}\) (Woakes and Butler, 1986). The underwater velocity of redhead ducks in this study was 0.8 m·sec\(^{-1}\), and if lactic acid increases when swimming at these velocities even while breathing, then it is likely that lactic acid will also increase during a dive. The short pauses may not be sufficient to clear this anaerobic byproduct, 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. To extend this study will require sampling arterial blood, allowing us to estimate the influence of plasma lactate on the duration of a diving bout.

Circumstantial evidence from section 2 suggests that muscle oxygen stores may not maintain contraction for more than 30 seconds if ducks are prevented from gaining access to the surface at the end of a voluntary dive. In fact, leg propulsion was succeeded by an even shorter period of wing propulsion before all locomotor activity ceased and the animal floated passively to the surface. Heart rates telemetered from these animals suggest that cardiovascular adjustments similar to those observed in forced dives (i.e. bradycardia and redistribution of blood flow) may occur when the animal
becomes aware of its plight. If so, then muscle oxygen stores are not effective in prolonging muscle activity in submerged ducks. In a preliminary study, the tibialis anterior muscle of Pekin ducks was electrically stimulated and isometric tension measured. The muscles of the hindlimb of the duck maintained their strength of contraction for less than 30 seconds and completely fatigued in about 90 seconds after muscle blood flow was occluded (fig. 20). In forced dives, contraction strength of the hindlimb decreased with a similar time course once extreme bradycardia occurred (fig. 20), but contraction strength was unaltered for 45 minutes while the animal was breathing.

Diving ducks differ from dabbling ducks in their response to both forced and voluntary diving. When forcibly submerged, dabbling ducks respond to a fall in arterial oxygen tension by slowing heart rate and increasing peripheral resistance, whereas diving ducks reduce heart rate before any change in arterial oxygen tension occurs. This "immersion response" may be adaptive in the wild because oxygen conservation could extend dive time; however, nasal receptor stimulation caused only a small change in the dive response of freely diving redhead ducks. Furthermore, when trapped under water, diving ducks showed an intense bradycardia, yet dive time was not greatly increased. The pre-dive tachycardia and hyperventilation, seen when diving ducks voluntarily dive, will increase oxygen stores thereby maximizing aerobic dive
Figure 20. Traces of the isometric tension, in arbitrary units, developed when the tibialis anterior is stimulated at 3 pulses per second. A decrease in contraction strength when the ischiatic artery is occluded can be seen within 10 seconds following occlusion. Arterial blood pressure is included for the dive as an indication of cardiovascular performance in the dive. The spikes seen in the tension trace of the dive represents struggles.
time as suggested by Butler and Woakes (1982a). The bradycardia and probable redistribution of blood flow (Heieis and Jones, personal communication) occurring on submergence would then aid in conserving oxygen for the exercising muscles.

Baroreceptor denervation does not alter the cardiac response in diving ducks diving voluntarily, so if arterial pressure is being maintained in intact ducks, then total peripheral resistance must be the controlled variable. During voluntary diving, diving ducks appear to control heart rate by producing a constant increase in vagal activity. In contrast, dabbling ducks diving voluntarily appear to regulate heart rate using their baroreceptors, but it is more likely that arterial pressure is being maintained. In this case, dabbling ducks may be controlling peripheral flow, and adjusting heart rate accordingly to regulate blood pressure. This explanation, however, hinges on assumptions that arterial pressure is being maintained and that stroke volume is constant. Jones et al. (1983) reported that stimulation of the proximal cut ends of the baroreceptors during submersion of a force-dived Pekin duck caused little change in peripheral resistance. The reason is unclear, but it may be that the baroreflex is incapable of overriding the intense vasoconstriction brought about by chemoreceptor activation, and may not reflect a central inhibition of the baroreflex. Chemoreceptors will not be activated above resting levels at
the moment of submersion, and the baroreflex is probably fully operative and could regulate arterial pressure through changes in peripheral resistance or cardiac output. Clearly, further research is necessary to uncover the role of baroreceptors in voluntary diving of both groups of ducks.

It is traditional to report the heart rate response to submersion as means and standard errors in the manner presented in figure 1 (section 1). This presentation, however, suggests that there is a typical pre-dive and dive heart rate. This is an acceptable conclusion when there is little variability in pre-dive and dive heart rates among dives, but it is clear from this study that some information regarding the heart rate response to diving is lost using this form of analysis. It is also common in the diving literature to report either percent change or difference in heart rate. A common mechanism, however, can lead to differences in percent change or absolute fall in heart rate depending on the initial value. Clearly, an understanding of the contributions of cardiac sympathetic and vagal nerves is necessary for a complete analysis of the heart rate response to forced and voluntary diving.
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