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RESPIRATORY AND CARDIOVASCULAR CONTROL DURING DIVING IN BIRDS AND MAMMALS

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SUMMARY

Recent studies on freely diving birds and mammals indicate that, contrary to the classical hypothesis, the majority of dives are aerobic with minimal cardiovascular adjustments (i.e. bradycardia and selective vasoconstriction). It is postulated that during these aerobic dives the cardiovascular adjustments result from the opposing influences of exercise and the classical diving response, with the bias towards the exercise response. It is envisaged that the active muscles, as well as the brain and heart, are adequately supplied with blood to enable them to metabolize aerobically. Intense mental activity, particularly in carnivores seeking their prey, may also attenuate the classical response. Aerobic dives are usually terminated well before the oxygen stores are depleted, and another dive follows once they have been replenished. In this way a series of dives is performed.

Prolonged dives are endured as a result of a shift towards the classical response of bradycardia, presumably more intense vasoconstriction, and anaerobiosis. This may be a form of alarm response, particularly in small animals such as ducks and coypus, or it may be a means of allowing the marine birds and mammals that dive deeply for their food to engage in unusually long hunting expeditions. For those that dive under ice, it may also allow long periods of underwater exploration as well as being a safety mechanism should the animal become disoriented.

INTRODUCTION

There has recently been a shift in the views concerning the type of metabolism and the intensity of cardiovascular adjustments that occur during diving in all vertebrates, including birds and mammals. This shift is largely the result of studies on free diving animals, which do not normally exhibit the dramatic cardiovascular adjustments, i.e. a large reduction in heart rate and selective vasoconstriction, that are seen during forced submersion. Our understanding of the factors controlling the respiratory and circulatory systems during diving must therefore be reassessed.

THE DIVING RESPONSE

Many aquatic birds and mammals can survive forcible submersion for much longer than would be expected on the basis of their oxygen stores and of continued aerobic metabolism at the pre-dive rate (Scholander, 1940). It was concluded therefore that

oxygen consumption is reduced during such diving. Scholander's experiments on seals, ducks and penguins verified this conclusion and also demonstrated that the level of lactic acid in the blood increases, particularly after the animal surfaces.

Subsequent studies on forcibly submerged birds and mammals have substantiated these findings (Scholander & Irving, 1941; Andersen, 1959; Andersen, Hustvedt & Lövö, 1965; Clausen & Ersland, 1970*a*, 1971; Murphy, Zapol & Hochachka, 1980) and thus added considerable weight to the suggestion made by Irving (1934, 1939) that the way for diving endotherms to survive asphyxia is for the supply of oxygen to be reduced to all tissues and organs that can withstand oxygen lack in the short term. They can metabolize anaerobically, producing lactic acid. On the other hand, oxygen-dependent tissues, like the central nervous system and heart, must have their oxygen supply maintained (see Bryan & Jones, 1980*a*; Murphy *et al.* 1980). This, Irving contended, could be achieved by reduced perfusion of the body, except the heart and the brain.

All investigations have confirmed that during forced submersion, selective vasoconstriction occurs, so that the perfusion of tissues such as the gut, kidneys, liver and skeletal muscle decreases substantially (Johansen, 1964; Elsner *et al.* 1966*a*; Butler & Jones, 1971; Jones *et al.* 1979; Zapol *et al.* 1979). On the other hand, in ducks, blood flow to the adrenals and brain increases and coronary flow remains unchanged (Johansen, 1964; Jones *et al.* 1979), and in seals, adrenal blood flow is reduced less severely than that to other tissues, coronary flow is reduced in proportion to the reduction in cardiac work, and cerebral flow remains unchanged (Zapol *et al.* 1979). In all, there is a considerable reduction in total cardiac output which is almost directly proportional to the decline in heart rate (i.e. stroke volume changes little or decreases). This so called 'diving bradycardia' is the typical element of the cardiovascular adjustments to diving. The decline in cardiac output and increase in total peripheral resistance are so closely matched that, except perhaps for the first few seconds, there is usually little or no change in systemic arterial pressure during a forced dive.

The rate of onset of the bradycardia varies between animals (Fig. 1*a*, *b*). In some mammals, particularly the muskrat *Ondatra zibethica*, coypu *Myocastor coypus*, and seals, heart rate declines almost instantaneously upon forced submersion to 30% or less of its initial value (Scholander, 1940; Elsner, 1965; Folkow, Lisander & Öberg, 1971; Drummond & Jones, 1979). After this initial rapid decline, heart rate may continue to fall to a lower level of 10–20% of the pre-dive value. At the other extreme, the herbivorous Florida manatee *Trichechus laterostris* exhibits a slowly developing bradycardia to 50% of its pre-dive value (Scholander & Irving, 1941). Also, in the foetus of the Weddell seal *Leptonychotes weddelli*, bradycardia develops more slowly and to a lesser extent than in the mother, and there is evidence of selective vasoconstriction in response to apnoeic asphyxia of the mother (Elsner, Hammond & Parker, 1970; Liggins *et al.* 1980). However, the foetus relies heavily on the continued perfusion of the placenta (Liggins *et al.* 1980). In most aquatic birds that have been studied, forcible submersion of the head causes a progressive reduction in heart rate over a period of many seconds (Fig. 1*a*). In diving ducks the reduction is quicker than in dabbling ducks (Butler & Woakes, 1982*b*). In mallard duck *Anas platyrhynchos*, it

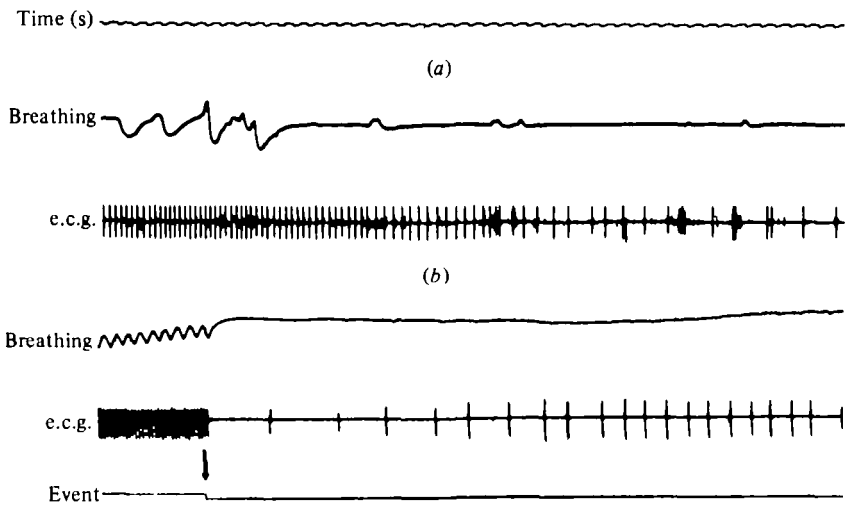

Fig. 1. Traces showing development of bradycardia in response to forcible submersion of the head of (*a*) a domestic duck, *Anas platyrhynchos*, and (*b*) a muskrat, *Ondotra zibethica*. Downward deflexion of the event marker indicates the point of submersion in each case. (Jones, 1981.)

takes 20–30 s for a 50% reduction in heart rate, whereas in the tufted duck *Aythya fuligula*, a 50% decline is reached within 5 s of forced submersion (Butler & Taylor, 1973; Butler & Woakes, 1982*b*). However, in the gentoo penguin, *Pygoscelis papua*, heart rate falls progressively (Millard, Johansen & Milsom, 1973).

The functional significance of these cardiovascular adjustments to forced submersion, in terms of oxygen conservation and the maintenance of oxidative metabolism in the brain, has been demonstrated (Irving *et al.* 1941*b*; Butler & Jones, 1971; Bryan & Jones, 1980). Thus, these adjustments, together with their metabolic consequences of reduced overall oxygen usage and anaerobic production of lactic acid, would appear to be the ideal solution to the problem of underwater survival for aquatic endotherms. However, the implementation of such adjustments may not be as intense or as common as was once thought.

Most marine mammals, and the emperor penguin, *Aptenodytes forsteri*, are periodic breathers with excessivelly long respiratory pauses between individual breaths or groups of breaths even when the animals have access to air. During inspiration, particularly in the mammals, heart rate increases whereas during the long pauses it declines quite substantially (Irving *et al.* 1963; Kanwisher & Sundnes, 1965; Kooyman, 1967; Ridgway, 1972; Lin, Matsuura & Whittow, 1972; Harrison, Ridgway & Joyce, 1972; Casson & Ronald, 1975). In some of these animals, heart rate during short spontaneous dives is no lower than during periods of breath holding in air, e.g. bottlenose dolphin, *Tursiops truncatus* (Ridgway, 1972), Weddell seal (Kooyman & Campbell, 1972), hooded and harbour seals, *Cystophora cristata* and *Phoca vitulina* respectively (Påsche & Krog, 1980). In the Weddell seal, heart rate during short (5 min) voluntary dives is approximately twice as high as that during longer (> 20 min) dives and during enforced submersion (Kooyman & Campbell, 1972). Within 30 s

of voluntary submersion, heart rate is lower during the (eventually) longer dive than during the shorter ones.

More interesting is the fact that over 97% of voluntary dives performed by Weddell seals in Antarctica are of less than 26 min duration and totally aerobic, with blood lactate not increasing substantially above its resting level (Kooyman *et al.* 1980). Only during the few longer dives does blood lactate increase and, after a dive of 61 min duration, it may take 2 h to return to its resting level. For this marine mammal it is clear that, during the vast majority of its natural dives, no anaerobiosis occurs, and it is concluded that the cardiovascular adjustments are minimal. Also, there is no evidence of vasoconstriction in the dorsal epaxial muscle of dolphins, *T. truncatus*, during trained dives of 1·5 min duration to 100 m depth (Ridgway & Howard, 1979). During brief (15 s) feeding dives there may be no bradycardia at all in the harbour seal (Jones *et al.* 1973). This may not be too surprising as no oxygen conserving adjustments would be necessary. Surprisingly, however, muskrats show a great reduction in heart rate to 17% of the resting value, within 1–2 s of spontaneous submersion, even though the dive may only last a few seconds (Drummond & Jones, 1979).

With the notable exception of the emperor penguin (Kooyman *et al.* 1971*a*), birds do not dive voluntarily for longer than 1–2 min and most birds appear to have mean dive durations of less than 1 min (Butler & Jones, 1982*a*). When diving naturally, neither pochards, *Aythya ferina*, tufted ducks (Butler, 1980; Butler & Woakes, 1976, 1979) nor cormorants, *Phalacrocorax auritus*, (Kanwisher, Gabrielsen & Kanwisher, 1981) exhibit a maintained bradycardia. In the case of the ducks (Fig. 2), heart rate falls from an elevated pre-dive rate, to its lowest value immediately upon submersion (cardiac interval may begin to increase before submersion). Heart rate then increases and reaches a steady level after 6–8 s. This steady heart rate and the heart rate recorded from diving cormorants are similar to those recorded from the birds while they are swimming on the surface of the water (Fig. 2) and are higher than the resting rates in each case. Frightening the ducks into diving causes heart rate during submersion to be higher than during voluntary dives (Butler & Woakes, 1979), whereas preventing tufted ducks surfacing from a voluntary dive causes a distinct bradycardia (Fig. 3). In free-diving penguins, heart rate is lower than in penguins exercising on water or on land, but it does not go below the resting level during dives of less than 60 s duration (Millard *et al.* 1973; Butler & Woakes, 1982*c*). In the tufted duck, oxygen usage during voluntary dives is approximately 3·5 times that recorded during periods of inactivity and is similar to that recorded at the duck's maximum sustainable swimming velocity (Woakes & Butler, 1982).

Oxygen stores are greater in diving animals than in their more terrestrial relatives (Packer *et al.* 1969; Lenfant *et al.* 1970; Keijer & Butler, 1982) and they may be enhanced just before diving by hyperventilation (see Fig. 2) and increased cardiac output (Ridgway, Scronce & Kanwisher, 1969; Kooyman *et al.* 1971*a*; Kooyman *et al.* 1973; Butler & Woakes, 1979; Craig & Påsche, 1980). It has been calculated that there is sufficient oxygen in the body of a tufted duck to allow oxidative metabolism to continue at 4 times the resting rate for 46 s (Butler & Woakes, 1982*a*). The longest reported natural dive for this species is 40 s (Dewar, 1924).

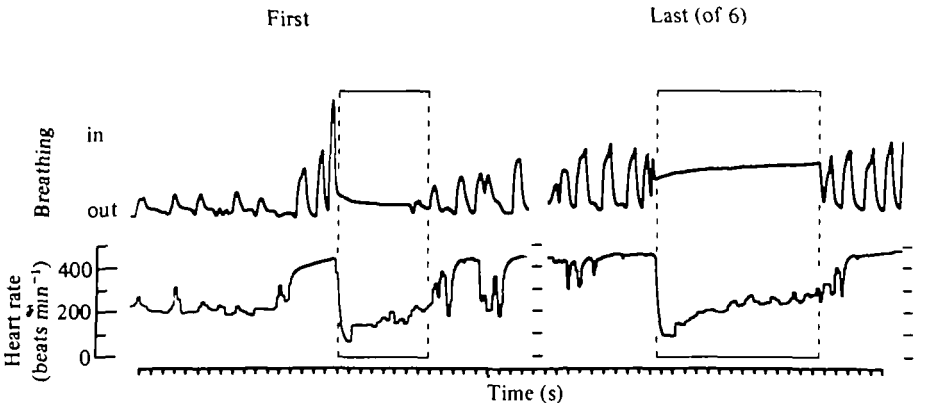

Fig. 2. Traces showing heart rate and respiratory frequency associated with the first and last spontaneous dives of a series performed by a male tufted duck, *Aythya fuligula*, on a pond 0·65 m deep. In each case the period of submersion is indicated by the dashed vertical line joined by horizontal bars. (Butler, 1980.)

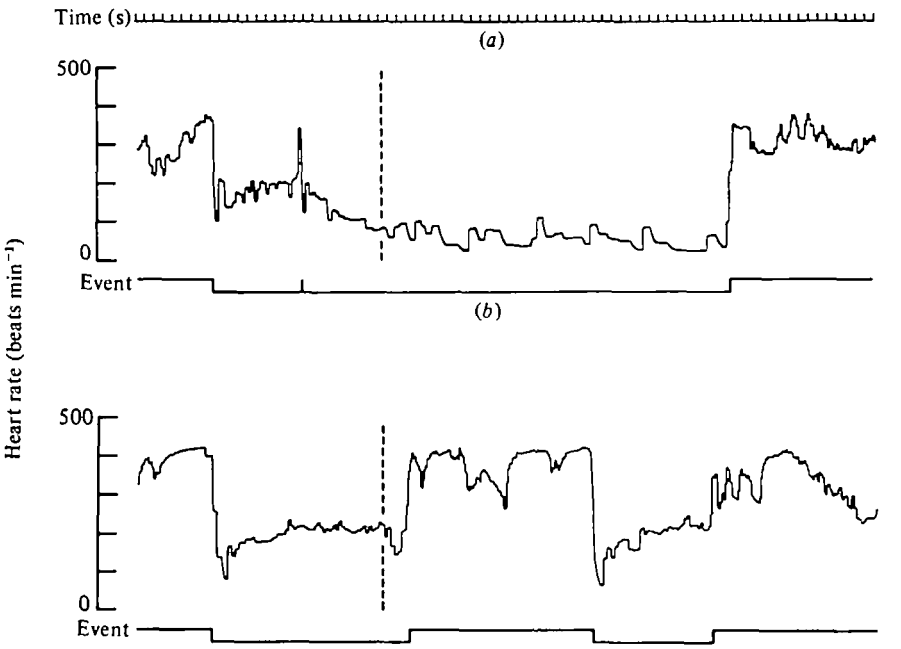

Fig. 3. Traces showing heart rate of male tufted duck, *Aythya fuligula*, (*a*) when prevented from surfacing after a spontaneous submersion, (*b*) during two unimpeded, spontaneous dives. In each case the duck was on a tank, 1·6 m deep. Downward deflexion on the event marker indicates the point of submersion, and upward deflection the point of surfacing. In (*a*) the duck began to surface at the time indicated by the vertical line on the event marker, but was prevented from gaining immediate access to air. The vertical dashed lines in (*a*) and (*b*) indicate identical times after submersion and illustrate the differences in heart rate under the two conditions (Woakes & Butler, 1982).

In summary, studies on voluntarily diving birds and mammals indicate that metabolism is usually aerobic and that bradycardia (and presumably peripheral vasoconstriction) is less than that caused by forced diving and may be completely absent. It is most likely that the active skeletal muscles are adequately perfused. However, these animals can, and sometimes do, dive for extended periods and resort to anaerobiosis with all of the accompanying cardiovascular adaptations. Although invoked infrequently under natural conditions, these adjustments must constitute a safety factor of great biological importance. The success of diving birds and mammals must, at least partly, reside in the fact that they can, if necessary, survive prolonged periods of submersion. Only by understanding the natural limits of an animal's endurance can we appreciate the full extent of biological adaptation.

CONTROL OF VENTILATION

During diving

During submersion (forced or voluntary), ventilatory activity ceases, despite the fact that some stimulatory inputs, e.g. those resulting from exercise and asphyxia, would be expected to increase during a normal dive. During natural dives the animals are exercising and it is well known that exercise in air stimulates ventilation. In terrestrial mammals, this hyperpnoea may result from a combination of direct stimulation from the motor cortex (Ledsome, 1968), or the hypothalamic locomotor region (Marshall & Timms, 1980; Eldridge, Millhorn & Waldrop, 1981), together with feedback excitation from the joints (Barron & Coote, 1973) and muscles (Coote, Hilton & Perez-Gonzalez, 1971; McCloskey & Mitchell, 1972; Mitchell, Reardon & McCloskey, 1977) of the exercising limbs. In tufted ducks swimming at maximum sustainable velocity, ventilation is approximately 3 times resting values (Woakes & Butler, 1982). Passive leg movements in decerebrate ducks cause a slight (25%) increase in ventilation (Butler & Jones, 1982*b*), but the contribution of muscle receptors is unknown. The arterial chemoreceptors also contribute to exercise hyperpnoea in dogs (Flandrois, Lacour & Eclache, 1974) and in man (Wasserman *et al.* 1975). During diving, this influence would be compounded by the progressive depletion of oxygen and accumulation of carbon dioxide, the latter of which would also stimulate the central chemoreceptors.

Despite earlier suggestions (Andersen, 1963), CO_2 does not inhibit ventilation in diving birds, although administration via the lungs may cause 'functional pulmonary vagotomy' as a result of its inhibition of CO_2-sensitive pulmonary receptors (Milsom, Jones & Gabbott, 1981) and thus cause a reduction in respiratory frequency. Nonetheless, when breathing gas mixtures which give blood gas tensions similar to those recorded at the end of 60 s forced submersion, mallard ducks increase their ventilation volume by a greater proportion than do chickens and pigeons exposed to similar changes in P_{a,O_2} and P_{a,CO_2} (Butler & Taylor, 1973, 1974). The ventilatory responses of nutria, *Myocastor coypus*, to hypoxia and hypercapnia fall within the range reported for non-diving mammals (Frankel & Ferrante, 1978). Studying respiratory sensitivity to hypoxia and hypercapnia in marine mammals is not that simple, because as well as changing tidal volume and/or respiratory frequency while breathing, they can

also alter the duration of the apnoeic period (Påsche, 1976*a*, *b*; Gallivan, 1980). Early studies on restrained seals (Robin *et al.* 1963) and manatees (Scholander & Irving, 1941) indicated that they are relatively insensitive to CO_2. However, freely diving animals exhibit a greater sensitivity to CO_2 (Påsche, 1976*a*; Gallivan, 1980), with the manatee maintaining a constant P_{A,CO_2} while $\dot{V}_E$ increases (Gallivan, 1980). When expressed on a relative basis (% increase in ventilation for a given change in CO_2), the data from harbour and hooded seals overlap with those from man (Craig & Påsche, 1980). The threshold ($P_{A,O_2} \simeq 8$ kPa) and response of harbour seals to hypoxia is similar to that for man (Craig & Påsche, 1980).

Thus the ability of diving birds and mammals to remain apnoeic while submerged is in no way the result of a dramatic reduction in the sensitivity of their receptor systems to hypercapnia or hypoxia. Although there may be a strong voluntary component to breath holding during submersion, experiments on decerebrate animals have indicated that there is also a reflex inhibition of respiratory activity (Andersen, 1963; Drummond & Jones, 1979). Submerging intact ducks with a tracheal cannula to the air only invariably causes apnoea when the water reaches the level of the glottis (Butler & Jones, 1968). However, section of the glottal branch of the IXth cranial nerve does not have any significant effect on apnoea during submersion and the ophthalmic and mandibular branches of the trigeminal nerve seem to be most important in this respect (Andersen, 1963; Bamford & Jones, 1974). The receptors themselves are sensitive to temperature (cold) or mechanical stimulation (Bamford & Jones, 1974; Leitner & Roumy, 1974*a*, *b*). The cold receptors are thought to be responsible for apnoea, and they cause not only cessation of inspiratory activity but also enhancement of expiratory activity (Leitner, Roumy & Miller, 1974). Although diving mammals close their external nares when diving, water would still, presumably, reach the glottal region during feeding. In seals, stimulation of the trigeminal and superior laryngeal nerves cause apnoea (Elsner, Angell-James & Daly, 1977), and in the muskrat, the maxillary branch of the Vth cranial nerve and the inferior laryngeal nerve are involved in the apnoea of submersion (Drummond & Jones, 1979).

These receptors inhibit ventilation not only at the beginning of submersion but also as stimulation of the chemoreceptors increases throughout the dive (Bamford & Jones, 1976*b*; Daly, Elsner & Angell-James, 1977; Elsner *et al.* 1977). In anaesthetized cats, brief chemical stimulation of the carotid body during expiration causes enhanced expiratory activity and the later in expiration the stimulus is given (provided it is not too late) the greater this effect becomes (Eldridge, 1976). In those animals which submerge upon expiration, e.g. ducks and seals, it is possible therefore that the increased chemoreceptor activity could reinforce apnoea rather than be a potential stimulus to breathing. A postural apnoea, elicited by the vestibular apparatus or muscle receptors in the neck, may be important during diving in birds (Huxley, 1913; Paton, 1913). Although manually placing the head of a restrained duck into different positions does not induce apnoea (Andersen, 1963; Butler & Jones, 1968), at the beginning of a natural dive the neck is rapidly extended and arched while the head is plunged vertically into the water (Butler & Woakes, 1982*a*). Such violent movement could induce a postural apnoea. Eventually, however, the diving bird or mammal surfaces for air and apnoea is terminated.

Recovery

There is evidence, in seals, that the duration of voluntary dives is determined by blood gas tensions (Kooyman *et al.* 1971*b*; Påsche, 1976*a*, *b*; Craig & Påsche, 1980) and, in the manatee, that CO_2 is the major factor (Gallivan, 1980). In tufted ducks, denervation of the carotid bodies extends the duration of voluntary dives by 24%, although they are still, on average, approximately half of the maximum calculated length of aerobic dives (Butler & Woakes, 1982*b*). Other factors are therefore involved in terminating a dive long before the oxygen stores are depleted. In man, it has been demonstrated that further breath-holding is possible, even though hypoxia and hypercapnia are not alleviated at the breaking point of the previous breath hold (Fowler, 1954; Honda *et al.* 1981). The absence of normal respiratory movements may lead to the stimulation of muscle, joint and tendon receptors in the respiratory muscles as they develop tension; the act of lung ventilation would temporarily remove such stimulation (Campbell *et al.* 1967). A general accumulation of excitatory state in the medullary respiratory neurones arising from the loss of respiratory movement has been discussed (Godfrey & Campbell, 1968). The return of P_{E,CO_2} to normal is necessary before the bottlenose dolphin is willing to dive again (Ridgway *et al.* 1969), so perhaps CO_2, acting directly via the central chemoreceptors and indirectly via receptors in the respiratory muscles, is the major stimulus. If so, hyperventilation before a dive would prolong dive duration not only by increasing oxygen stores (see p. 198) but also by reducing P_{a,CO_2}. However, dive duration in a number of birds appears to be related to the depth of water: the deeper the water the longer the dives (Dewar, 1924; Butler & Woakes, 1979; Butler, 1980). Also, some mammals are able to remain apnoeic despite the accumulation of large quantities of lactic acid and a decline in pH_a during long voluntary dives (Kooyman *et al.* 1980). The balance between inhibitory and excitatory influences on ventilaton during diving is clearly extremely variable.

Generally, breathing begins immediately upon surfacing, but the gray whale, *Eschrichtius robustus*, may begin to expire while the nares are still submerged (Kooyman, Norris & Gentry, 1975), i.e. before the inhibitory stimuli are removed. At the other extreme, breathing may be delayed for several seconds or even minutes after a forced dive (Irving, Scholander & Grinnell, 1941*a*; Butler & Jones, 1971), and on one occasion, after a tufted duck was prevented from surfacing from a natural dive, it did not breathe, after an initial exhalation, for 19 s (Woakes & Butler, 1982). Once breathing does begin, it is always at an elevated level and the time taken for it to return to pre-dive levels varies directly with the length of the dive, in freely diving Weddell seals (Kooyman *et al.* 1971*b*) and in forcibly submerged ducks (Lillo & Jones, 1982*b*). Presumably, following aerobic voluntary dives, hyperpnoea lasts as long as is required to replace the oxygen stores (Butler, 1980; Kooyman *et al.* 1980). The level of CO_2 in the blood may well determine when the animal is ready to dive again (Ridgway *et al.* 1969; Gallivan, 1980). After longer dives, when anaerobiosis occurs, then other factors, such as disturbances in acid/base balance and the removal of anaerobic metabolites are likely to prolong the stimulation of ventilation (Kooyman *et al.* 1971*b*).

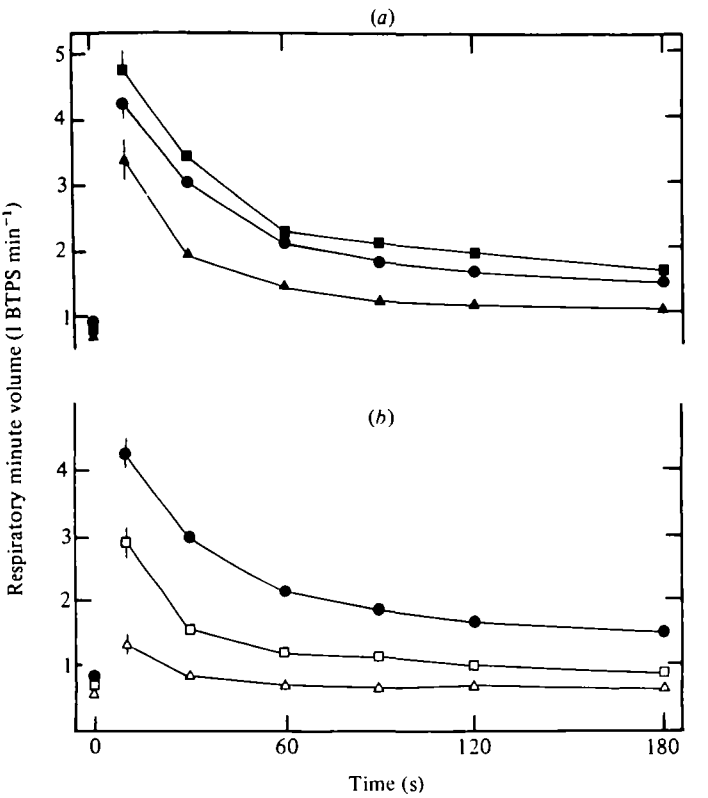

Fig. 4. Mean values of respiratory minute volume in unanaesthetized domestic ducks, *Anas platyrhynchos*, following emergence from forced submersion for 2 min and from 2 min forcible submersion while being unidirectionally ventilated (UDV) with one of two gas mixtures (*a*) before and (*b*) after chronic denervation of the carotid body chemoreceptors. ●, Two min forcible submersion with no UDV; ■, UDV with 5 % O_2; ▲, UDV with 8 % CO_2; □, UDV with 5 % O_2 and 5–8 % CO_2; △, UDV with 10 % CO_2. Pre-dive values are given at time = 0 and ±S.E. of mean are given for first set of values following emersion. (Modified from Lillo & Jones, 1982*b*).

It would be reasonable to assume that hyperpnoea upon surfacing is the result of the uninhibited stimulation of the central and peripheral chemoreceptors and of the increase in cardiac output and venous return (cf. Wasserman, Whipp & Castagna, 1974). The evidence is not that convincing. The magnitude of post-dive hypernoea in forcibly submerged ducks is not affected by artificially maintaining heart rate and blood pressure (and hence cardiac output) at low levels upon surfacing (Lillo & Jones, 1982*b*). Blood gas tensions return to normal within 20 s of surfacing after 2 min submersion in ducks, which then become hyperoxic and hypocapnic for a short while as the hyperpnoea continues (Butler & Jones, 1971; Butler & Taylor, 1973). Denervation of the carotid bodies dramatically reduces the influence of hypercapnia upon post-dive hyperpnoea in ducks, and the residual response to CO_2 is probably mediated via the central chemoreceptors. However, the effect of hypoxia and hypercapnia combined on post dive hyperpnoea is less dramatically reduced after carotid body denervation (Fig. 4). Thus, hypoxia is exerting its influence

via receptors other than those in the carotid bodies. Although the locomotory muscles may not be contracting at this stage, even in voluntarily diving animals, it could be that 'metabolic receptors' (Hník *et al.* 1969; Coote, Hilton & Perez-Gonzalez, 1971; McClosky & Mitchell, 1972) in skeletal muscle contribute to the post-dive hyperpnoea, particularly at the end of forced dives and excessively long voluntary dives, when high levels of metabolites are released from the muscles. The concentrations of the metabolites may be related to P_{a,O_2} at the end of the dive.

CONTROL OF CARDIOVASCULAR SYSTEM DURING DIVING

Efferent control

It is clear that the cardiac response to submersion is not uniform under different conditions. The reduction in heart rate (and presumably the intensity of the other cardiovascular changes) seems to be maximal during forced submersion of diving animals (except in muskrats – Drummond & Jones, 1979) and may be attained infrequently during the normal life of these animals. Whatever the magnitude of the effect, the efferent pathways will be the same.

Bilateral vagotomy or injection of atropine prevents any reduction in heart rate not only during forced submersion of birds and mammals (Van Citters *et al.* 1965; Butler & Jones, 1968, 1971; Ferrante & Opdyke, 1969; Drummond & Jones, 1979), but also during more natural dives (Murdaugh, Seabury & Mitchell, 1961; Butler & Woakes, 1982*a*). Increased parasympathetic activity is most likely the sole cause of the bradycardia during diving (Butler & Jones, 1968, 1971; Ferrante & Opdyke, 1969; although Folkow *et al.* (1967) suggest that there is also a reduction in sympathetic activity to the heart during forced submersion. The vagus also has a negative inotropic effect on the heart of ducks and coypus during forced diving (Folkow & Yonce, 1967; Ferrante & Opdyke, 1969; Folkow *et al.* 1971). This explains why, despite real increases in venous pressure (Johansen & Aakhus, 1963; Folkow *et al.* 1967), ventricular dilatation (Aakhus & Johansen, 1964; Ferrante & Opdyke, 1969; Blix & Hol, 1973) and maintained or even reduced arterial pressure, cardiac stroke volume does not increase appreciably, and may even decrease in all birds and mammals studied (Elsner, Franklin & Van Citters, 1964; Folkow *et al.* 1967; Cohn, Krog & Shannon, 1968; Folkow *et al.* 1971; Jones & Holeton, 1972; Sinnett, Kooyman & Wahrenbrock, 1978; Zapol *et al.* 1979). The one aberrant report is that of a 2·5 times increase in the duck after 20–72 s of submersion (Jones *et al.* 1979). There is general agreement that there is a marked reduction in myocardial work and hence in oxygen demand of the heart during enforced diving.

The peripheral vasoconstriction is mediated by sympathetic efferents acting via α-adrenoceptors (Djojosugito, Folkow & Yonce, 1969; Kobinger & Oda, 1969; Butler & Jones, 1971; Folkow *et al.* 1971; Andersen & Blix, 1974). Venoconstriction is thought to be important in mobilizing the venous oxygen stores during forced dives, and also presumably during prolonged voluntary dives (Djojosugito *et al.* 1969; Ronald, McCarter & Selley, 1977). It is, however, the degree of constriction on the arterial side which determines the level of perfusion to each area of the body.

In seals and in ducks the larger arteries constrict before they enter the organ or tissue they supply (Bron *et al.* 1966; Folkow, Fuxe & Sonnenschein, 1966; White, Ikeda & Elsner, 1973). Thus these larger arteries contribute approximately 20% of total resistance in the skeletal vascular bed of a duck during maximal vasoconstriction, but less than 5% in the cat. Also, these arteries are not affected by the vasodilator substances produced during muscle contractions, and overall vasoconstriction can be maintained in the muscles of ducks even during exercise (Folkow *et al.* 1966). The same authors found that the femoral artery of the duck is much more densely supplied with adrenergic nerves than that of the cat or turkey and that invaginations of the wall where the artery branches are particularly well innervated in the duck. In seals, the renal arteries can be occluded near their origin from the aorta during enforced diving and these, together with other supply arteries, have sympathetic nerves penetrating into their muscular layers, thus ensuring maximum recruitment of smooth muscle (White *et al.* 1973). The intensity of the nervous penetration into the muscular layer seems to determine the degree of closure of that particular vessel. The influence of sympathetic activity on the vascular beds of the kidney, intestine and skeletal muscle in the coypu is more pronounced than in the cat; there being no 'auto regulatory escape' in the intestinal vascular bed of the diving animal (Folkow *et al.* 1971).

Circulating catecholamines are also likely to exert an influence on the large arteries during diving. Exercise itself is known to stimulate the release of catecholamines in man (Manhem, Lecerof & Hökfelt, 1978), and in ducks the levels double after 1 min forced submersion (Huang, Sung & Huang, 1974). After 2 min submersion, humoral factors contribute up to 30% of the increase in hind-limb vascular resistance in ducks, and as the concentration of catecholamines increases exponentially as the dive progresses, so they would be expected to exert a progressively greater influence (Hudson, Jones & Pivnik, 1982). The release of catecholamines is possible because of the maintained or increased blood supply to the adrenals during forced submersions of seals (Zapol *et al.* 1979) and ducks (Jones *et al.* 1979). Whether circulation to renal, intestinal and skeletal muscular beds is sufficient during the later stages of a forced dive to maintain these substances at sufficiently high concentrations is unknown. The diameter of the pulmonary artery decreases during forced diving in ducks (Aakhus & Johansen, 1964) and resistance to flow in the pulmonary circulation of the harbour seal increases by 3 times during forced submersion (Sinnett *et al.* 1978). Such a response is not too surprising as both hypoxia and hypercapnia cause pulmonary vasoconstriction in cats and dogs (Barer, Howard & Shaw, 1970).

Several factors would tend to result in an increased coronary blood flow during forced submersion, e.g. extended diastole and decreased P_{a,O_2}. However, coronary blood flow is closely related to the work performed by, and therefore the oxygen demand of, the myocardium (Berne, 1974). The reduced cardiac work associated with diving bradycardia offsets the other factors in ducks, where coronary flow changes little during forced submersion (Jones *et al.* 1979). In seals, coronary resistance increases and flow falls during forced submersion (Zapol *et al.* 1979). This may be the result of a greater reduction in cardiac work than in ducks and the

fact that the measurements were made early in the dive when oxygen saturation of the blood was still high (Butler & Jones, 1982*a*).

There is growing evidence to suggest that reflexes from the arterial chemoreceptors are not involved in controlling cerebral blood flow (Heistad *et al.* 1976; Traystman, Fitzgerald & Loscutoff, 1978). In fact, the physiological role of the sympathetic innervation of the cerebral blood vessels appears to be of only minor significance (Traystman & Rapela, 1975); it is local factors, such as hypoxia, that play a dominant role. The cerebral vessels of ducks are much more sensitive to hypoxia and hypercapnia than those of terrestrial mammals (Grubb *et al.* 1977; Grubb, Colacino & Schmidt-Nielsen, 1978), which, no doubt, explains the 8·5 times rise in cerebral blood flow in the duck during prolonged, forced submersion. At the measured blood-gas tensions during a prolonged dive, hypercapnia is responsible for a 2-times increase and hypoxia a 6-times increase in cerebral blood flow, the effects being additive (Jones *et al.* 1979). It would be interesting to know whether the cerebral vessels of diving mammals are similar to those of other mammals or to those of ducks in their sensitivity to P_{a,O_2} and P_{a,CO_2}.

Afferent control

As with the respiratory system, there will be opposing stimuli affecting the cardiovascular system during voluntary dives. In mammals, rhythmic exercise in air causes tachycardia, increased arterial blood pressure, and a rise in blood flow to the respiratory and locomotor muscles (Fixler *et al.* 1976). The sense organs in the muscles and joints that cause hyperpnoea during exercise in air also cause slight tachycardia and a rise in arterial blood pressure (Coote *et al.* 1971; McCloskey & Mitchell, 1972; Mitchell *et al.* 1977). Also, stimulation of sites in the diencephalon causes locomotor activity, tachycardia, increased blood pressure, vasoconstriction in renal and mesenteric vascular beds, and functional hyperaemia in the working limbs (Smith, Rushmer & Lasher, 1960; Marshall & Timms, 1980). These areas could be involved in the generation of locomotion and in the integration of the accompanying cardiovascular adjustments described. Passive movements of the hind limbs of decerebrate, artificially ventilated ducks do not cause significant increases in heart rate (Butler & Jones, 1982*b*).

There is evidence to suggest that nasal and facial receptors, as well as producing apnoea, are also involved in the rapid cardiovascular changes associated with diving in some mammals. A rapid bradycardia occurs during forced submersion or nasal stimulation in artificially ventilated seals and muskrats, respectively, although it is not as intense as that during apnoeic submersion (Tanji, Weste & Dykes, 1975; Drummond & Jones, 1979). If the nasal, facial, and, in the case of the muskrat, the laryngeal receptors, are eliminated by local anaesthesia or nerve section, there is no bradycardia under these conditions (Dykes, 1974; Drummond & Jones, 1979). The rate of onset of bradycardia is slower in harbour seals during enforced apnoea in air than it is during forced submersion. After complete anaesthesia of the face, the cardiac response is similar in both situations, even if the seals have access to air via a tracheal cannula when under water (Dykes, 1974). Similarly, in the same species, the onset of bradycardia during voluntary diving is more rapid than that during

spontaneous apnoea in air (Jones *et al.* 1973). These observations indicate that immersion *per se* may have a direct effect on heart rate, via trigeminal and laryngeal receptors. A more recent report, however, states that the intensity and rate of onset of bradycardia is similar during voluntary diving and spontaneous apnoea in air in both harbour and hooded seals (Påsche & Krog, 1980), although the evidence regarding the rate of onset is not convincing. If correct, this statement does detract somewhat from the idea that trigeminal and laryngeal receptors exert a direct influence on the cardiovascular system during natural dives.

Many workers have claimed that part of the bradycardia seen during forced submersion of ducks is in direct response to stimulation of receptors on the beak or internal respiratory passages (Andersen, 1963; Feigl & Folkow, 1963; Folkow *et al.* 1967). Unlike the situation in seals and muskrats, the time-course of bradycardia in ducks is similar during natural respiratory pauses in air, forced apnoea of paralysed birds in air, and during enforced submersion (Butler & Jones, 1968; Butler & Taylor, 1973; Bamford & Jones, 1974). What is more, if ducks or geese continue to breathe spontaneously at normal levels through a tracheal cannula when the head is partially or totally submerged, there is no bradycardia (Butler & Jones, 1968; Cohn *et al.* 1968). It does not seem, therefore, that receptors on the beaks or internal respiratory passages of adult birds have any direct effect on heart rate. However, Jones, Milsom & Gabbott (1982*a*) found that if heart rate is above approximately 190 beats min^{-1}, then an initial bradycardia is apparent in ducks upon forcible submersion. Running water down an orally facing tracheal cannula in an artificially ventilated duck can also cause a reduction in heart rate and vasoconstriction (Fig. 5), but these are probably secondary effects (see later). In ducklings there does appear to be a direct effect of water immersion *per se*, causing approximately one-third of the bradycardia in response to forced submersion (Jones & Butler, 1982). The temperature of the water has no effect on the cardiac response to forced submersion of the head in birds or mammals such as seals (Andersen, 1963; Butler & Jones, 1968; Dykes, 1974). In man, however, the colder the water the greater is the bradycardia during non apnoeic face immersion. The decrease in heart rate is least at approximately 35–40 °C (Kobayasi & Ogawa, 1973).

As well as any direct effects, stimulation of nasal or facial receptors can influence the cardiovascular system indirectly by inhibiting respiratory activity. This not only removes the influences of lung ventilation, it also reduces activity in central respiratory neurones. The concept of activity in discrete central respiratory neurones 'irradiating' to cardiovascular neurones and thereby imposing a respiratory rhythm on efferent activity to the cardiovascular system has been challenged (see, for example, Koepchen, Klüssendorf & Sommer, 1981). These authors think in terms of various degrees of intracentral coupling in efferent activity to the cardiovascular system with a mean cardiovascular–respiratory rhythm (but see Spyer, 1982). Whatever the neuroanatomical and physiological basis may be, there is, in diving birds and mammals, an increase in heart rate associated with activity in central inspiratory neurones in the absence of pulmonary afferent information (Bamford & Jones, 1976*a*; Angell-James, Elsner & Daly, 1981). It is suggested that it was the inhibition of such activity in central inspiratory neurones (or maybe in cardio-respiratory

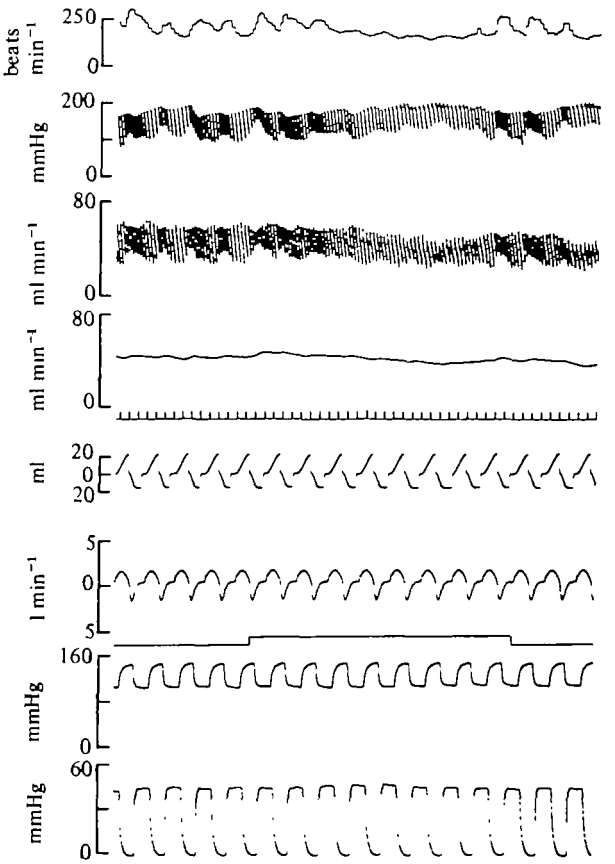

Fig. 5. The effect on heart rate, in a relaxed, artificially ventilated ♀ mallard duck, *Anas platyrhynchos*, of pouring water down an orally facing tracheal cannula. Traces, from above down: heart rate, arterial blood pressure, pulsatile flow through the femoral artery, mean flow through the femoral artery, time marker(s), respiratory tidal volume, respiratory air flow, event mark (upward deflection indicates period of water flow), tracheal P_{O_2}, tracheal P_{CO_2}. (Butler & Taylor, 1982.)

neurones – Koepchen *et al.* 1981) which eliminated the rhythmic increase in heart rate in the artificially ventilated duck when water was run over the glottis (see Fig. 5). A similar effect on heart rate, via central respiratory (or cardio-respiratory) neurones, may also occur in seals when facial receptors are stimulated upon submersion (Daly *et al.* 1977; Angell-James *et al.* 1981). Artificial lung inflation during periods of apnoea in ducks and seals causes tachycardia and a rise in arterial blood pressure, both of which are abolished following selective denervation of the lungs (Bamford & Jones, 1976*a*; Butler & Taylor, 1982) or bilateral vagotomy (Angell-James *et al.* 1981). Thus in those animals that dive upon expiration, removal of the excitatory influences of activity in central inspiratory neurones and pulmonary afferent feedback will contribute to any initial bradycardia. Sudden deflation of lungs in a paralysed muskrat certainly causes a bradycardia similar to that seen during voluntary dives

(Drummond & Jones, 1979). Presumably, during natural dives when the nostrils are closed, deflation of the lung and stimulation of receptors on the outside of the animal are the major, initial influences. There is no doubt, however, that the effects, whether direct or indirect, of stimulating nasal, facial and laryngeal receptors upon forced immersion have a more rapid and more intense effect on heart rate in seals and muskrats than they do on that in mallard or domestic ducks (see Fig. 1*a*, *b*). In ducks that dive naturally, such as tufted ducks and cormorants, these initial effects may be more important (Butler & Woakes, 1982*b*; Mangalam & Jones, 1982).

The progressive hypoxia and hypercapnia that develop during diving would tend to stimulate the central and peripheral chemoreceptors. Under the influence of certain anaesthetics, stimulation of the carotid bodies in dogs, in the absence of changes in ventilation, causes bradycardia and generalized vasoconstriction (Daly & Scott, 1958, 1962). However, if transmission through the hypothalamic defence area is not affected, then similar stimulation of the carotid bodies in cats evokes a variable change in heart rate and vasodilatation in the limbs; a response similar to that during the alerting stage of the defence reaction, in that the vasodilatation is, at least in part, cholinergic (Marshall, 1977). Stimulation of the aortic bodies causes vasoconstriction and a slight bradycardia (Angell-James & Daly, 1969) and hypercapnic stimulation of the central chemoreceptors in cats leads to vasoconstriction in the hind-limbs (Lioy, Hanna & Polosa, 1981).

Denervation or perfusion of carotid bodies with hyperoxic blood prevents much of the bradycardia in response to forced head submersion in unanaesthetized mallard ducks or their domesticated varieties (Hollenberg & Uvnäs, 1963; Jones & Purves, 1970; Lillo & Jones, 1982*a*; Jones *et al.* 1982*a*; Butler & Woakes, 1982*b*). Stimulation of the peripheral chemoreceptors accounts for 85% of the bradycardia and 67% of the increase in vascular resistance in domesticated ducks, while the central chemoreceptors, sensitive to P_{CO_2} in the blood, contribute about 30% of the total change in vascular resistance (Jones *et al.* 1982*a*). Thus, in mallards, or their domesticated varieties, peripheral and central chemoreceptors are dominant in producing the cardiovascular responses to enforced submersion. That is why these responses are relatively slow in their onset; they rely on the progressive accumulation of CO_2 and depletion of O_2. This appears not to be the case, however, in truly diving birds, such as tufted ducks or cormorants, *P. penicillatus*, where a relatively rapid initial bradycardia is unaffected by carotid body denervation or pre-breathing 100% O_2 (Butler & Woakes, 1982*b*; Mangalam & Jones, 1982). Although neither of these birds shows a maintained bradycardia during voluntary dives with respect to the rate recorded during swimming activity on the surface, denervation of the carotid bodies in tufted ducks has no effect on the initial changes in heart rate (Butler & Woakes, 1982*b*) but does lead to a higher rate than normal towards the end of long (> 20 s) natural dives (Fig. 6). Observations from one duck indicate, however, that the carotid bodies are not involved in the bradycardia that occurs when a tufted duck is prevented from surfacing from a voluntary dive (see Fig. 3*a*; Woakes & Butler, 1982).

Carotid body chemoreceptors are unimportant in initiating the diving responses in seals and muskrats (Tanji *et al.* 1975; Daly *et al.* 1977; Drummond & Jones, 1979). However, the carotid bodies do exert a profound inhibitory effect on the

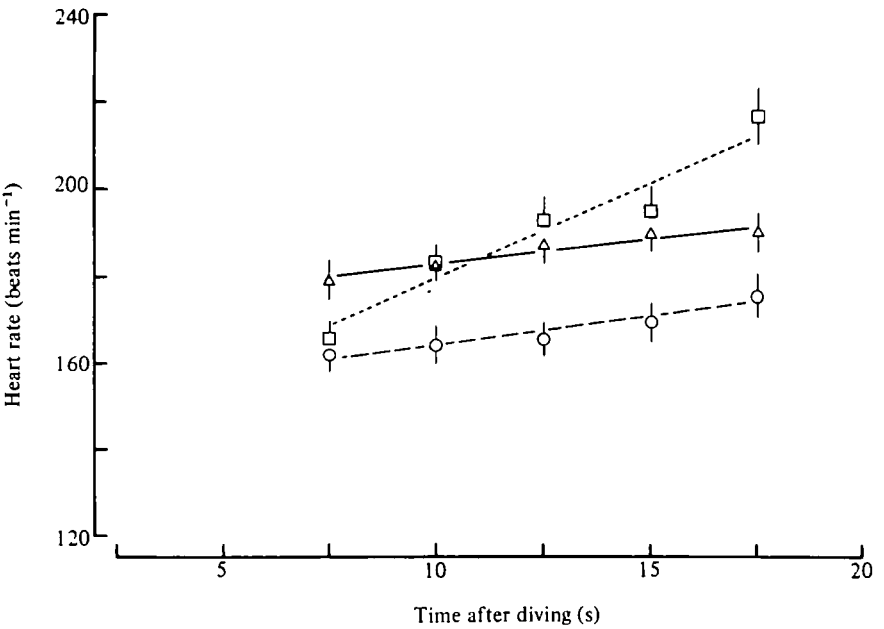

Fig. 6. Regression lines of heart rate in intact (---), sham operated (——) and carotid body denervated (------) tufted ducks, *Aythya fuligula*, during the middle portion (7·5–17·5 s) of relatively long natural dives. For intacts, 41 dives, for shams, 23 dives and for denervates 45 dives. Equations describing regression lines: $y = 151 + (1{\cdot}33 \pm 0{\cdot}55)\,x$ for intacts, $y = 171 + (1{\cdot}17 \pm 0{\cdot}54)\,x$ for shams and $y = 136(4{\cdot}33 \pm 0{\cdot}95)\,x$ for denervates. Mean values (±S.E. of mean) of heart rate at the sample points are also given: intact (○), sham operated (△), denervated (□). (From Butler & Woakes, 1982*b*.)

heart of anaesthetized seals as the dive progresses (Daly *et al.* 1977). On the other hand, artificial lung ventilation with air, at a level similar to that recorded in resting, restrained animals, does not abolish the bradycardia in forcibly submerged, unanaesthetized seals (Tanji *et al.* 1975). Taking into account the effect of lung ventilation itself on heart rate (Angell-James *et al.* 1981), it is clear that the chemoreceptors were not contributing much to the bradycardia at all under these conditions. Thus the importance of the peripheral chemoreceptors during forced dives in seals is uncertain. There are no data on their importance during voluntary dives. In the foetus, the onset of bradycardia may occur in the absence of measurable changes in foetal blood gas tensions (Liggins *et al.* 1980). Whether or not interaction occurs in anaesthetized seals between nasal reflexes and chemoreceptor reflexes, with respect to their effect on heart rate (see Elsner *et al.* 1977) rather depends on the base line chosen for comparison (Butler & Jones, 1982*a*).

With changes in cardiac output and vasoconstriction occurring in various vascular beds during diving, baroreceptors may play an important role in maintaining arterial blood pressure by themselves influencing cardiac activity and peripheral resistance. It has been concluded that a barostatic reflex is involved in the reduction in heart rate during forced submersion in ducks, although some authors consider this role to be a minor one (Kobinger & Oda, 1969; Butler & Jones, 1971) while

others consider it to be of greater importance (Andersen & Blix, 1974). The effect of denervation of the baroreceptors on the cardiovascular response to forced submersion depends upon whether the operation is acute or chronic (Jones, 1973; Lillo & Jones, 1982*a*; Jones *et al.* 1982*a*, *b*). Nonetheless, bradycardia and increases in peripheral resistance do occur in each case, so that baroreceptors are not essential for these changes. As a result of a series of elegant experiments involving stimulation of the central cut end of a baroreceptor nerve and perfusion of the central and peripheral chemoreceptors in ducks, Jones and his colleagues conclude that the baroreceptors are not necessary for the maintenance of arterial pressure during forced diving. They may maintain it 'sensibly constant during a dive' and they contribute some 10–15% to the bradycardia seen after 60–90 s of forced submersion (Jones *et al.* 1982*a*, *b*). In seals, the barostatic reflex is more sensitive during submersion, probably as a result of a shift of the curve to the left, i.e. resetting (Angell-James, Daly & Elsner, 1978). These authors suggest that the resetting is the result of interaction between inputs from the baroreceptors, nasal receptors and chemoreceptors. Drummond & Jones (1979) were unable to demonstrate that baroreceptors are involved in the diving bradycardia in muskrats. Despite an earlier suggestion to the contrary (Blix, Wennergren & Folkow, 1976), Jones, Milsom & West (1980) demonstrated quite convincingly that receptors in the left ventricle play no part in the cardiac chronotropic response to forced submersion in ducks.

The inhibitory effects on the heart of nasal stimulation and of brief stimulation of carotid baroreceptors or chemoreceptors are only apparent in anaesthetized dogs during expiration (Haymet & McCloskey, 1975; Gandevia, McCloskey & Potter, 1978*a*, *b*). Central inspiratory neurone activity or rapid lung inflation can each independently prevent bradycardia being elicited by any of the above stimuli. The blocking of these cardioinhibitory influences will not be present in seals and ducks during submersion, since they dive on expiration. Sea lions, cetaceans and penguins, however, dive on inspiration, and although maintained lung inflation has no effect, prolonged activity in the central inspiratory neurones may inhibit full expression of the diving reflexes in these animals (Gandevia *et al.* 1978*a*). This could explain why, in the California sea lion, heart rate decreases relatively slowly during trained head submersion (Elsner *et al.* 1964) and why heart rate is slightly lower, during the expiration that follows a voluntary apnoea in air, than it is at the end of apnoea itself (Lin *et al.* 1972). Heart rate also declines slowly in the dolphin, *Tursiops truncatus*, during voluntary dives (Irving *et al.* 1941*a*), although in *T. gilli*, bradycardia is immediate at the beginning of longer, trained dives (Elsner, Kenney & Burgess, 1966*b*). In addition, forcibly submerged penguins often exhibit a bradycardia only when they have exhaled some air (Butler & Woakes, 1982*c*). In the anaesthetized seal the ventilatory changes in response to selective stimulation of the carotid bodies with hypercapnic hypoxic blood are insufficient to prevent a substantial slowing of the heart (Elsner *et al.* 1977), whereas spontaneous breaths are able to alleviate much of the bradycardia during experimental diving (Angell-James *et al.* 1981). Clearly, the interaction between these various stimuli and their eventual effect on the cardiovascular is complex and may depend very much on the intensity of each stimulus.

Recovery

As may be the case with lung ventilation, the recovery of the cardiovascular system at the end of a dive does not result merely from the removal of inhibitory and commencement of excitatory sensory stimuli. Heart rate may increase as the animal prepares to surface from forced or voluntary dives (Irving *et al.* 1941*a*; Murdaugh *et al.* 1961; Kooyman & Campbell, 1972; Jones *et al.* 1973; Millard *et al.* 1973; Butler & Woakes, 1979, 1982*a*), but quickly returns to the diving value if surfacing and breathing do not actually occur (see Fig. 3*a*; Murdaugh *et al.* 1961; Butler & Woakes, 1979). With most forced dives the complete recovery process does not occur until the first breath is taken, when there is tachycardia and vasodilatation (Scholander, 1940; Elsner *et al.* 1966*a*; Butler & Jones, 1968, 1971; Butler & Taylor, 1973; Bamford & Jones, 1976*a*).

The tachycardia in ducks is the result of a reduction of vagal tone and, according to most authors, is unaffected by β-adrenoceptor blockade (Butler & Jones, 1968, 1971; Kobinger & Oda, 1969; Butler & Taylor, 1973). However, Folkow *et al.* (1967) found that the recovery tachycardia and rise in cardiac stroke volume are diminished after β-adrenoceptor blockade and therefore suggest that sympathetic activity to the heart increases at this time. Sympathetic β-receptors could be involved in increasing cardiac stroke volume (cf Folkow *et al.* 1967) and/or they could be involved in the vasodilatation upon emergence (Butler & Jones, 1971). In the absence of other influences, the marked vasodilatation in skeletal muscle immediately upon surfacing could be the result of stimulation of the carotid bodies evoking a defence-type reaction, thus including an element of sympathetic cholinergic activity (cf. Hilton & Joels, 1965; Marshall, 1977). Such an involvement, however, is not evident from the results of Butler & Jones (1971) working on ducks, and a reduction in activity in sympathetic constrictor fibres may be the major factor. The high levels of circulating catecholamines that may be present at the end of a forced dive (Hudson *et al.* 1982) would be expected to have a constrictor effect, so perhaps the α-adrenoceptors are inhibited. On the other hand, a neurogenic, non-cholinergic, non-adrenergic vasodilator mechanism (McGregor, 1979) may operate in ducks during the recovery period.

It is difficult to identify the separate effects on the cardiovascular system of activity in central inspiratory neurones and lung inflation when an animal surfaces from a dive. In ducks and seals, artificial inflation of the lungs during forced dives causes a large increase in heart rate (Bamford & Jones, 1976*a*; Angell-James *et al.* 1981; Butler & Taylor, 1982). This cardioacceleration is abolished in ducks by denervation of the lungs or by increasing CO_2 concentration in the inflating gas to 8–10% (Bamford & Jones, 1976*a*; Butler & Taylor, 1982). Thus in birds the receptors are CO_2-sensitive. In seals, inflation with a hypoxic hypercapnic gas mixture still causes cardioacceleration during face immersion (Angell-James *et al.* 1981). The reason why, in ducks, heart rate upon recovery from a forced dive is greater than that when breathing a hypercapnic hypoxic gas mixture, even though ventilation volume is similar in both cases (see Butler & Taylor, 1973), may be that the CO_2 receptors are inhibited in the latter case but become suddenly active at the end of a dive when

air is breathed. Certainly, the increase in heart rate upon surfacing from a forced dive is slower in ducks with denervated lungs than it is in intact animals (Bamford & Jones, 1976*a*). Activity in central inspiratory neurones is known to cause cardiac acceleration in ducks (Bamford & Jones, 1976*a*), while such an effect is highly likely in seals (Angell-James *et al.* 1981).

The immediate alleviation of chemoreceptor drive is not essential for the recovery tachycardia upon commencement of breathing in seals (Scholander, 1940; Daly *et al.* 1977), although bradycardia returns if the anoxic conditions are maintained (Scholander, 1940). The return of blood gases to normal is necessary for the long term recovery from a dive. When diving voluntarily, both birds and mammals probably only remain at the surface long enough to replace their oxygen stores. The sooner this is achieved the sooner they can dive again, so there may be a voluntary component to the hyperpnoea, and hence to the increased circulation, between dives.

CONCLUDING REMARKS

The differences in the cardiovascular responses to forced and voluntary dives in the same animal, indicate that the reflexes that have been discussed so far may be modified in the brain (see Thomas & Calaresu, 1973; Coote *et al.* 1979, for experiments on cats). Decreases in heart rate before submersion and increases before surfacing may involve some form of associative learning (Jones *et al.* 1973). Unlike Rey (1971), Gabbott, Jones & Campbell (1982) were able, by repeated submersion, eventually forcibly to dive ducks without their showing a bradycardia. The apparent lack of a bradycardia in ducks and cormorants during voluntary dives could be the result of a similar process involving conditioning or habituation. Preliminary data from tufted ducks indicate however that there may be a bradycardia during voluntary dives. For one particular bird, oxygen uptake during diving was 0·46 ml s^{-1} (3·4 times resting) and the mean, stable heart rate was 126 beats min^{-1}. When swimming at the surface and consuming oxygen at 0·46 ml s^{-1}, its heart rate was 174 beats min^{-1} (Woakes & Butler, 1982). Thus the cardiovascular adjustments during voluntary diving may result from the opposing influences of exercise and the classical diving response (cf. Millard *et al.* 1973), with the bias towards the exercise response. This would explain the higher heart rate in tufted ducks towards the end of voluntary dives after denervation of the carotid bodies (Butler & Woakes, 1982*b*). It is envisaged, therefore, that during aerobic voluntary dives, blood flow increases to the exercising muscles and is reduced to the non-exercising muscles, kidneys and intestine (cf. Mitchell, Remensnyder & Sarnoff, 1963; Fixler *et al.* 1976; Hohimer & Smith, 1979). The intensity of vasoconstriction may be greater during diving than during exercise in air, thus leaving proportionately more of the slightly reduced (presumably) cardiac output for the contracting muscles and the brain. Ducks have a high resting blood flow to their skeletal muscles, compared with cats and dogs, even though their weight-related oxygen uptakes are similar (Folkow *et al.* 1966; Grubb, 1981). This gives a significant venous reserve for use during exercise. The reduced heart rate during diving, compared with exercise in air, may therefore relate to inactive respiratory muscles, slightly greater

vasoconstriction, and increased $a - v_{O_2}$ content difference. If, in the above example the rest of the body did consume less oxygen during diving, then the legs must have used more during diving than during swimming on the surface, as total oxygen uptake was the same in each case. Few data are available on the energetics of swimming in marine birds and mammals, but from those that are, oxygen uptake during diving is 1·6 times resting in the harbour seal in the laboratory (Craig & Påsche, 1980) and may actually be less than the resting value in Weddell seals (Kooyman *et al.* 1973) and king penguins (Butler & Jones, 1982*a*) in Antarctica. Thus a similar argument could apply. The fact that blood flow to the hind limb decreases when a penguin dives (Millard *et al.* 1973) does not detract from this idea, for the penguin swims with its flippers! Also, penguins and the marine mammals are specifically adapted for locomotion in water, so they may be very efficient under such conditions.

During prolonged voluntary dives, or if prevented from surfacing, seals and ducks exhibit more profound bradycardia, similar to that seen during forced submersion. In seals, the duration of the dive may be adjusted to the cardiovascular changes made early in the dive (Jones *et al.* 1973) or the animal may prepare for a particular dive duration (Kooyman & Campbell, 1973). Ducks prevented from surfacing have to adjust to an extended dive duration just as they are about to surface and they can do this, apparently, without information from their carotid body chemoreceptors (Woakes & Butler, 1982). These ducks (and presumably seals during unusually long dives) continue to exercise, so there is a shift in the balance between the exercise and diving-type responses under these circumstances.

Immediately upon diving voluntarily, trigeminal reflexes, the withdrawal of central inspiratory activity and pulmonary afferent information could augment any cardiovascular adjustments initiated centrally in diving mammals, particularly those that dive on expiration. This is not likely to be the case in naturally diving ducks, where there is a large reduction in heart rate from its elevated pre-dive level. No such large instantaneous reduction is seen during forced submersion, voluntary head ducking or dabbling (Butler & Woakes, 1979), so in ducks diving voluntarily this response may be totally central in its origin. It is extremely unlikely that the hypothalamic area (area A), which was electrically stimulated by Folkow & Rubinstein (1965), is involved in this response as they obtained a slowly developing bradycardia. This area could be involved, however, in the cardiac response seen when a voluntarily diving duck is prevented from surfacing (Fig. 3*a*). Stimulation in the periventricular area of the anterior hypothalamus of the northern elephant seal, *Mirounga angustirostris*, does cause cardiovascular adjustments that are similar to those seen during diving (Van Citters *et al.* 1965). Unfortunately there are no other such studies on diving animals.

It has been suggested that the classical 'diving reflex' may be important if an animal dives when alarmed (Feigl & Folkow, 1963) and the cardiac changes seen in tufted ducks prevented from surfacing from a voluntary dive tend to support this idea. However, ducks that have never been submerged before show no bradycardia during a forced dive after breathing 100% O_2 (Gabbott *et al.* 1982). The more familiar pattern of cardiovascular changes associated with the alerting stage of the defence-alarm reaction when breathing, i.e. vasodilatation in skeletal muscle and vasoconstriction in the kidneys and splanchnic circulation (Hilton, 1982),

be elicited in man by emotional stress such as mental arithmetic (Brod *et al.* 1959). Mental arithmetic also attenuates the bradycardia induced by face immersion in man (Ross & Steptoe, 1980). The mental effort involved in searching for food, especially in carnivores, may have similar effects on the cardiovascular system during diving. This would enhance the exercise-type response.

Although experiments on animals that have been forcibly submerged give important information on the possible nervous pathways involved in the respiratory and cardiovascular adjustments during diving, there is no doubt that we need more data from animals diving under natural or near natural conditions. Most dives performed by aquatic birds and mammals are probably for food and are almost always aerobic. If penguins or the smaller aquatic mammals are being chased by their natural predators (leopard seals, killer whales) then it is doubtful if remaining submerged would afford much protection for most of them. A quick breath of air is probably always possible, especially for those animals that naturally leap out of water in a porpoise-like action. Penguins and pinnipeds would, in fact, be safer on land (or ice). Animals, such as the Weddell seal, which can dive deeply, could use such a tactic to escape from killer whales, which tend to perform more shallow dives. The exceptionally long dives that have been recorded for Weddell seals could be the result of prolonged searches for food, long periods of exploration in new territory, or of disorientation. The latter is a greater possibility when the seals dive under ice in the darkness of the winter months. Coypus, on the other hand, spend no longer than 20–30 s when under water feeding, but if alarmed they can lie immobile, completely submerged with their legs outstretched, for several minutes (Gosling, 1977). It is tempting to suggest that when feeding the cardiovascular adjustments are minimal, but during 'alarm' dives, of whatever duration, the cardiovascular changes are immediate and intense (cf. voluntary dives of the muskrat, Drummond & Jones, 1979). Only when we are aware of the full repertoire of the physiological responses to diving, and under which circumstances each response is displayed, can we begin to understand the mechanisms by which the responses may be controlled.

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