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THE CONTROL OF THE CARDIOVASCULAR ADJUSTMENTS TO DIVING IN BIRDS AND MAMMALS

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

INTRODUCTION

Forced submergence of diving birds and mammals evokes apnoea (in the expiratory position), bradycardia, and an increase in flow resistance in many systemic vascular beds so that, in spite of a large fall in cardiac output, arterial blood pressure is maintained (Elsner et al., 1966; Jones & Johansen, 1972; Angell James & Daly, 1972). In many systemic vascular beds flow resistance increases 8-10 times whereas, in contrast, flow resistance of the pulmonary circulation only increases by 1.5 to 3 times in ducks and seals (Sinnet et al., 1978; Jones, 1981). In both birds and mammals the fall in cardiac output is largely due to bradycardia although a slight fall in cardiac contractility may also occur (Folkow et al., 1967; Folkow & Yonce, 1967; Ferrante and Opdyke, 1969). These cardiovascular changes during diving result in cardiac output being redistributed so that the proportion received by oxygen dependent organs, such as the heart and brain, is maintained or increased while blood supply to those tissues which can withstand periods of anoxia is shut off (Blix et al., 1976; Jones et al., 1979; Zapol et al., 1979). Since oxygen stores are rationed for oxygen dependent tissues, which make up only a small proportion of the total body mass, aerobic metabolism during diving is greatly reduced. Anaerobic energy production notwithstanding, there can be no doubt that the ability to survive prolonged submergence depends almost totally on the cardiovascular adjustments during diving. This has been appreciated since the end of the last century (Richet, 1899) and confirmed on numerous occasions since then.

EFFECTOR PATHWAYS

In diving, the effector response is mediated by the parasympathetic and sympathetic branches of the autonomic nervous system. In addition, circulating catecholamines may make a contribution to the peripheral vascular response (Allison & Powis, 1971, 1976; Huang et al., 1974; Jones et al., 1981b). The heart is exposed to greatly increased vagal parasympathetic activity in dives, resulting in both negative chronotropic and inotropic effects (Folkow et al., 1967; Butler & Jones, 1968; Ferrante & Opdyke, 1969; Jones & Holeton, 1972; Finley et al., 1979). Also, a number of claims have been made that cardiac sympathetic activity is reduced during diving (Folkow et al., 1967; Lin, 1974) although not all investigators agree (Butler & Jones, 1968, 1971;

Ferrante and Opdyke, 1969). It may be that differences between tissue and blood levels of hypoxia in experiments cited above have contributed to this disagreement. For example, in rabbits, tissue hypoxia or severe arterial hypoxia causes an increase while mild arterial hypoxia causes a decrease in cardiac sympathetic activity (Iriki & Kozawa, 1975). In any event, the extreme bradycardia is testament to the dominance of the parasympathetic pathway on cardiac activity.

The increase in systemic flow resistance during diving is due to the activity of the sympathetic nervous system. Pharmacological blockade of adrenergic α-receptors or of neuronal transmitter release prevents effective redistribution of cardiac output (Kobinger & Oda, 1968; Butler & Jones, 1971; Andersen & Blix, 1974; Blix et al., 1975). After sympathetic inhibition, blood pressure falls with the developing bradycardia and underwater survival time is greatly reduced (Kobinger & Oda, 1968; Andersen & Blix, 1974). In both diving birds and mammals, the vascular beds of skeletal muscle, kidney and intestine are far more sensitive to a given level of vasoconstrictor nerve fibre activiation than are these beds in non-divers (Folkow et al., 1966; Folkow et al., 1971). Furthermore, in ducks, coypus and seals, vasoconstriction involves the larger precapillary resistance vessels which are remote from the actions of the vasodilator metabolites that accumulate in the tissues during submergence (Irving et al., 1942; Folkow et al., 1966; Folkow et al., 1971). Both the density of innervation and degree of penetration of vasoconstrictor nerve fibres, even in large arteries (1 mm diameter), is much greater in diving than non-diving animals (Folkow et al., 1966; White et al., 1973), suggesting that maximal 'recruitment' of smooth muscle would be achieved by the more intimate association of vasocontrictor terminals and medial muscle fibres. In non-divers sympathetic nerve fibres are restricted to the adventitia of large arteries (White et al., 1973).

SENSORY PATHWAYS

Activation of a number of diverse groups of sensory receptors causes cardiovascular reflexes, either as a primary or secondary effect, which have at least some aspects of the diving reflexes. Undoubtedly, more than one group of receptors will be activated during submergence so it is possible that "undesirable" components of certain cardiovascular reflexes, i.e. hypertensive barostatic vasodilation, could be overruled by interaction between inputs (Korner, 1971). This rationale has led to suggestions that the afferent nervous pathway of diving reflexes may involve some or all of the following receptors or reflexes; central and peripheral chemoreceptors, baroreceptors, left ventricular receptors, "nasal" receptors, cessation of activity from central respiratory neurones and pulmonary receptors, and vestibular reflexes (Huxley, 1913a & b; Jones & Purves, 1970; Angell James & Daly, 1972; Lin et al., 1972; Jones, 1973; Daly & Angell James, 1975; Bamford & Jones, 1976; Blix et al. 1976; Angell James, Daly & Elsner, 1978; Drummond & Jones, 1979; Jones et al., 1980; Millard, 1980; Jones et al., 1981c & d). Finally, similarities between diving and cerebral ischaemic reflexes have been noted (Dampney et al., 1979; Kumada et al., 1979).

Obviously, there is much less agreement concerning sensory than motor pathways of the diving responses; another complication is that the initial and later phases of the diving responses may be controlled by different receptors. What there is no doubt about is that the

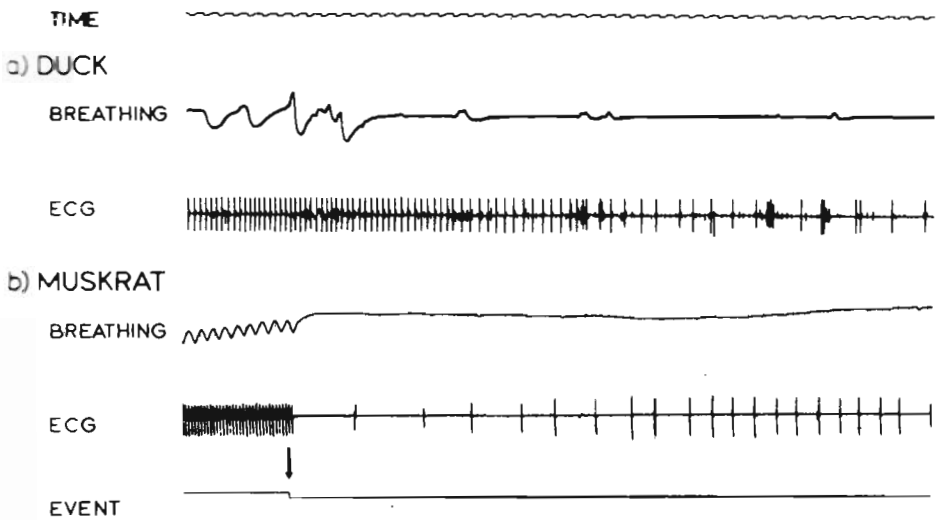

Figure 1. Traces showing slow (a, duck) and rapid (b, muskrat) development of diving bradycardia. Time, seconds, applies to all traces. Event, indicates submergence at the downward deflection, emphasised by the arrow, and applies to all traces.

time course of initiation of the diving reflexes is very different in different species (Fig. 1). In ducks, bradycardia develops rather slowly, especially if surface heart rates are of the order of 130-150 beats.min^{-1}, and full cardiac retardation may not occur for 60 sec or so (Fig. 1). On the other hand, seals and muskrats show extremely rapid development of bradycardia, full development occurs within one second of submergence (Fig. 1) (Dykes, 1974; Drummond & Jones, 1979). Increases in peripheral resistance must accompany bradycardia since arterial blood pressure is maintained, although that is not to say that the same receptors need be responsible for the total cardiovascular adjustment. The rapidly developed diving reflexes seem little affected by continuing ventilation during dives (Tanji et al., 1975; Drummond & Jones, 1979) whereas artificial ventilation of submerged ducks prevents diving responses (Reite et al., 1963).

The fast and slow adjustments are probably generated by different receptor groups. The rapid adjustment is primarily caused by stimulation of "nasal" receptors innervated by the V and X cranial nerves (Huxley, 1913a & b; Andersen, 1963; Angell James & Daly, 1969; Tchobroutsky et al., 1969; Dykes, 1974; Tanji et al., 1975; Drummond & Jones, 1979) while the slow adjustment results primarily from stimulation of arterial chemoreceptors by the progressively more hypoxic and hypercapnic blood (Daly & Scott, 1958; Jones & Purves, 1970; Angell James & Daly, 1972; Jones & West, 1978). Variations between the balance of nasal and chemoreceptor inputs, or even inputs from other receptors, will certainly occur between species and perhaps even between individuals of the same species during submergence (Table 1). In deep diving birds, i.e. cormorants, lung and therefore blood oxygen tensions will increase with

depth rendering a chemoreceptor based reflex inoperative in the early stages of a dive. However, cormorants display prompt bradycardia in forced dives (Jones & Mangalam, 1981). Input from, or interactions between, diverse receptor groups will modulate the character of the diving adjustments and add to the complexity of any attempted description of the response in terms of reflexology (Table I) (Elsner et al., 1977; Jones et al., 1981 c & d).

Table 1. Experimental manoeuvres (tests) causing bradycardia in muskrats (Ondatra zibethica). Mean values of heart rate are shown before (control) and at the time shown during the tests described in the column at the left. Also the heart rate during a test and the mean value obtained 1-2 s after the start of dives by restrained unanaesthetised animals are compared and sifnificant differences are noted. X, difference between means is significant ($P > 0.05\%$). 0, difference not significant ($P > 0.05\%$). n = number of observations. Table derived from data given in Drummond and Jones, 1979.

	Control	Test	Time	Significant difference	Notes
Unrestrained dive	310 ± 3 (n = 102)	54 ± 3 (n = 102)	1-2 s	X	All dives
		38 ± 2 (n = 37)	1-2 s	X	Dive lasting > 5 s
		73 ± 3 (n = 50)	1-2 s	0	Dive lasting < 4 s
Restrained dive	266 ± 3 (n = 66)	78 ± 4 (n = 60)	1 s	-	
Lung deflation	268 ± 7 (n = 23)	59 ± 4 (n = 23)	1-2 s	X	Paralysed
Nasal water flow 32 ml min^{-1}	277 ± 5 (n = 34)	20 ± 2 (n = 34)	1 s	X	Anaesthetised
	277 ± 8 (n = 16)	24 ± 3 (n = 16)	1 s	X	Anaesthetised, cf saline flow
Nasal saline flow	273 ± 9 (n = 12)	38 ± 3 (n = 12)	1 s	X	Apnoeic, cf with water flow group
Nasal water flow 32 ml min^{-1} Lungs' deafferented	195 ± 21 (n = 10)	48.5 ± 2 (n = 9)	1 s	X	Paralysed
Forced dive under anaesthesia	281 ± 5 (n = 56)	84 ± (n = 56)	1 s	0	non-apnoeic
Water on nose	292 ± 6 (n = 6)	76 ± 2 (n = 6)	1 s	0	Paralysed Ventilated with air
Hypoxia	277 ± 11 (n = 6)	76 ± 7 (n = 6)	when Pa_{O_2} = 4 kPa	0	Paralysed Ventilated with N_2+5% CO_2
Adrenaline Injection	253 ± 6 (n = 6)	90 ± 12 (n = 6)	maximum bradycardia	0	Paralysed Ventilated with air

COMPARISON BETWEEN FORCED AND VOLUNTARY DIVES

Virtually all experiments studying nervous control of cardiovascular resonses to diving have been done on conscious restrained animals so the contribution of 'emotional' stress, habituation or conditioning to the response is a further complicating factor. In many species the cardiac response to forced submergence is essentially similar to that observed in voluntary dives whereas in others there are marked differences (Eliassen, 1960; Elsner, 1965; Kooyman & Campbell, 1972; Jones et al., 1973; Ridgway et al., 1975; Drummond & Jones, 1979; Butler & Woakes, 1979). Both birds and mammals show reductions in heart rate before voluntary submergence and increases before voluntary emergence (Jones et al., 1973; Casson & Ronald, 1975; Butler & Woakes, 1979). In some short voluntary dives, seals may exhibit tachycardia not bradycardia and on short voluntary dives in birds maximum diving bradycardia is often obtained immediately upon submergence, heart rate then increases (Jones et al., 1973; Butler & Woakes, 1979). Cardiovascular responses to forced submergence are unaffected by thalamic decerebration (Huxley, 1913a; Andersen, 1963) but the difference between responses to voluntary and forced submergence implies that higher nervous centres are involved in naturally diving animals. Certainly, it is possible to condition or train diving birds and mammals to foster or inhibit bradycardia on submergence (Elsner, 1965; Ridgway et al., 1975; Jones et al., 1981a).

The exciting area for future research is to take the laboratory type of experimentation into the voluntary diving arena, so that the role of the central nervous system in modulating diving responses can be elucidated.

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