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CARDIAC OUTPUT OF DUCKS DURING DIVING

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Abstract—1. Blood flow in one pulmonary artery has been measured, using an electromagnetic flowmeter, in lightly restrained ducks before, during and after submergence.

2. This method gave values for cardiac output at rest (control) of 393·9 ± 53·8 ml/min (± S.E.M.) and stroke volume of 2·2 ± 0·33 ml.

3. Stroke volume increased slightly but not significantly during diving and after 2 min submergence cardiac output was 25·9 ± 5·4 per cent of control and heart rate 21·1 ± 2·1 per cent of control.

4. Heart rate and cardiac output increased sharply on emergence and after 10 sec were 136·7 ± 12·2 and 141·4 ± 7·8 per cent of control values respectively.

5. The present results confirm that the intense bradycardia that occurs during diving is accompanied by a reduction in cardiac output of the same order of magnitude as the fall in heart rate.

INTRODUCTION

THERE is little doubt that the profound bradycardia that accompanies submergence in diving animals must be accompanied by a reduction in cardiac output. From recordings of heart rate and blood pressure Eliassen (1960) concluded that in birds cardiac output decreases in proportion to the degree of bradycardia. From an indirect study of ventricular energy release Johansen & Aakhus (1963) deduced that stroke volume was virtually unchanged during diving and furthermore, from angiocardiographic studies, suggested that end-diastolic volume was not the only factor controlling stroke volume in ducks (Aakhus & Johansen, 1964). However, Folkow *et al.* (1967) found both a decrease in cardiac output and stroke volume in ducks. The fall in stroke volume of some 15–20 per cent of control occurred in spite of an increase in central venous pressure.

Elsner *et al.* (1964) directly measured stroke flows in the pulmonary artery of a sea lion and found that cardiac output varied in close accordance with the heart rate, as stroke volume was unchanged. Murdaugh *et al.* (1966) using the dye dilution technique and diving durations of up to 6 min recorded a mean decrease in cardiac output of 88 per cent of the predive value in seals with no consistent variations in stroke volume. The only other available measurements of cardiac output during diving are those reported for the frog by Shelton & Jones (1965). In frogs during submergence stroke volume decreases somewhat more proportionately than the reduction in heart rate and cardiac output falls to one-half to one-fifth of the surface value.

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Of all these recordings only those of Elsner *et al.* (1964) has been obtained directly with the minimum of attendant surgical interference. Consequently the aim of the present experiments was to implant electromagnetic blood flow probes on one pulmonary artery of the duck and record blood flow in the artery before, during and after submergence, after complete recovery from the operation, the latter process requiring from 14 to 30 days.

MATERIALS AND METHODS

Experiments were attempted on nine White Pekin Ducks (*Anas sp.*) varying in weight from 1·7 to 2·3 kg (average weight 2·1 kg) although good recordings of pulmonary artery blood flow were only obtained from six of the animals.

Blood flow was measured in the left pulmonary artery by a Biotronix BL 610 pulsed-logic electromagnetic flowmeter (Biotronix Laboratory Inc., Maryland) set to a frequency response of 50 Hz. The phase lag and amplitude distortion of the blood flow recording system was checked using a mechanical pump similar to that described by Taylor (1957). Over the range of frequencies tested (0·5–10 Hz) phase lag was 4·67°/Hz and amplitude distortion was negligible (less than 1 per cent over the full range). The output of the Biotronix flowmeter was displayed on either a Beckman RS Dynograph or a Techni-Rite 722 pen recorder. The former writes on curvilinear and the latter on rectilinear co-ordinates. The pulmonary artery was exposed after division of the sternum in the mid-line (Jones & Purves, 1970). An extra-corporeal blood flow transducer was placed around the vessel and secured by ligatures to surrounding tissue so that traction on the vessel was eliminated. The cuff-probes were 4–6 mm in dia. and were a snug fit on the blood vessel. In two ducks zero flow was obtained by use of a pneumatic cuff downstream of the flow probe (Jones, 1970) whereas in others zero flow was assessed from the flow baseline reached during the diving bradycardia. Both methods gave the same assessment of zero flow. The wound was closed, as described previously (Jones & Purves, 1970), and ducks were given penicillin (35,000 i.u./kg) for 4 days after the operation.

Ducks were used in experiments some 2–4 weeks after the operation when they were about 10 weeks old. At the termination of the experiments ducks were anaesthetized by an intravenous injection of Urethane and sacrificed by bleeding. The animal was reopened and a segment of pulmonary artery, with the cuff-probe still in position, was removed. The probes were calibrated in the usual manner using the duck's own blood (Jones, 1970).

The minimum amount of restraint was used both before and during the diving procedures. Ties were placed around the legs of the duck to hold them to an operating table, no other body restraints were used. Ducks were encouraged to dive by holding the head submerged in a beaker of water, it was often possible to release the head after the onset of the diving bradycardia and the animal would surface of its own volition some time later. In this manner dives in excess of 2 min were routinely achieved. Each duck was submerged, on average, three times in a 5-hr period.

Stroke volumes were obtained by integrating the area under the flow curve either by planimetry or by tracing the curves onto quality tracing paper, cutting them out and weighing them. This weight was compared with a standard weight representing a known flow. At least ten flow pulses were analysed in this manner before and after the dive whereas during submergence only two or three pulses were analysed either side of the desired time period. The results were analysed statistically and 5 per cent was considered as the fiducial limit of significance. In the text and tables all values are given $\pm$ S.E. of the mean.

RESULTS

Average cardiac output in all ducks at rest was $393{\cdot}9 \pm 53{\cdot}8$ ml/min or about 187·5 ml/kg per min. On a weight basis these cardiac outputs are lower than those

recorded by the dye dilution technique (Sturkie, 1966; Folkow *et al.*, 1967; Jones & Holeton, unpublished). The average resting heart rate was 186·2 ± 7·06 beats/min so stroke volume was 2·2 ± 0·33 ml.

On average stroke volume increased slightly during the dive but none of the changes were significantly different from the control value (resting duck). The maximum stroke volume was achieved after 60 sec submergence when it was 135 ± 16·4 per cent of control (Table 1). Due to the marked fall in heart rate cardiac output decreased to 58·4 ± 5·3 per cent of control after 20 sec submergence (Table 1). No further significant changes in either cardiac output or heart rate occurred until 60 sec submergence when both were significantly below the levels established after 20 sec underwater (Table 1). After 120 sec submergence cardiac output was 25·9 ± 5·4 per cent of control and heart rate 21·1 ± 2·1 per cent. Due to the slight increase in stroke volume which accompanied submergence the proportional reduction in cardiac output was not as great as that in heart rate.

On emergence both heart rate and cardiac output increased sharply and after 10 sec the former was 136·7 ± 12·2 per cent of control and the latter 141·4 ± 7·8 per cent of control. Both heart rate and cardiac output remained above control levels for at least 2 min after emergence (Table 1). There were no significant changes in

TABLE 1—CHANGES IN CARDIAC OUTPUT, HEART RATE AND STROKE VOLUME DURING DIVING AND RECOVERY. AVERAGE VALUES FOR ALL DUCKS.

	Cardiac output	Heart rate	Stroke volume
Predive	393·9 ± 53·8 ml/min	186·2 ± 7·06 beats/min	2·2 ± 0·33 ml
Dive 20 sec	58·4 ± 5·3%	53·3 ± 5·03%	115·0 ± 11·9%
30 sec	52·0 ± 7·6%	43·2 ± 4·5%	113·2 ± 8·9%
40 sec	53·4 ± 9·6%	41·9 ± 5%	120·4 ± 10·6%
60 sec	37·1 ± 6·2%	26·1 ± 3%	135·1 ± 16·4%
120 sec	25·9 ± 5·4%	21·1 ± 2·1%	116·9 ± 16·6%
Recovery 10 sec	141·4 ± 7·8%	136·7 ± 12·2%	114·2 ± 9·9%
60 sec	136·0 ± 7%	142·6 ± 14%	107·7 ± 9·6%
120 sec	125·9 ± 5·4%	125·8 ± 10·9%	108·0 ± 8·5%

The predive value is expressed in conventional units and all subsequent figures are percentages of this value. (No. of observations = 20.)

stroke volume during the recovery period either from control or stroke volumes prevailing during the dive.

There was considerable variation between individuals in stroke volumes during the dive. Two animals showed increases in stroke volume which in some cases approached 175 per cent of control after 2 min submergence. Three of the remaining four animals exhibited moderate increases in stroke volume (5–30 per cent) whereas the other consistently showed a decrease in stroke volume of the order of 20–30 per cent from control. There was no relationship between the

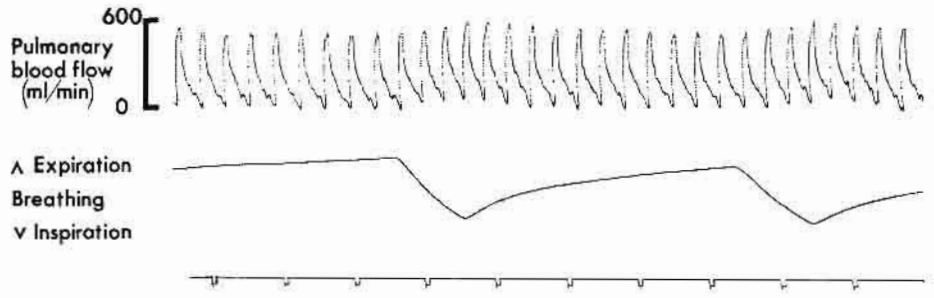

FIG. 1. The effect of increases in heart rate during inspiration on blood flow in the pulmonary artery. Upper trace, pulmonary blood flow; middle trace, breathing; lower trace, time (sec).

variations in stroke volume between individuals and any variations in heart rate. The lowest cardiac outputs were recorded from animals which consented to dive for periods in excess of 2 min, in two cases cardiac outputs of 12 per cent of control occurred. During recovery four of the animals showed maximal increases in stroke volume from 125 to 175 per cent of control but in these the tachycardia was not marked. In the other two ducks there was a pronounced tachycardia (173–202 per cent of control) and stroke volume was reduced below the pre-dive levels by as much as 33 per cent.

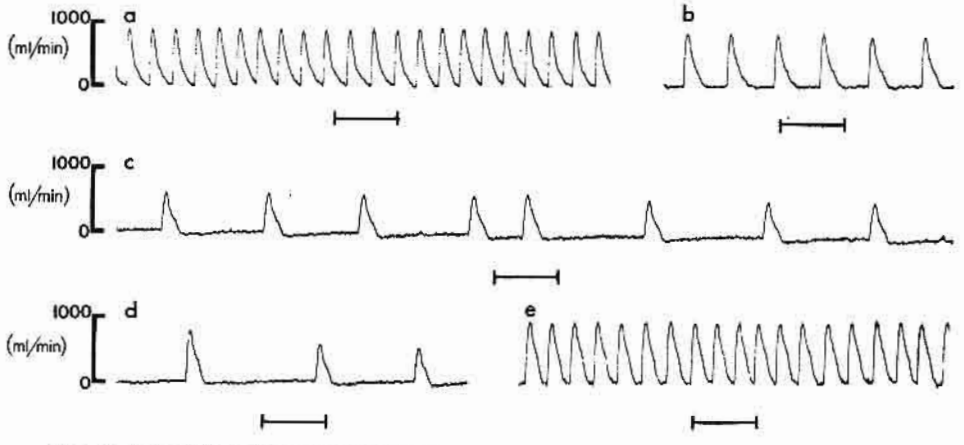

FIG. 2. Blood flow in the pulmonary artery before, during and after submergence. a, Pre-dive; b, dive—30 sec; c, dive—90 sec; d, dive—120 sec; e, recovery—20 sec. The marker under each trace indicates 1 sec.

In resting animals, after generation of peak flow, the flow decreased exponentially to zero (Fig. 2), though at high heart rates the decline was often interrupted by the next cardiac contraction before zero flow was reached. In some animals an inflexion occurred on the falling flow trace which probably represented closure of

the valves on the pulmonary outflow tract. Animals which exhibited sinus arrhythmia showed a marked interaction between the breathing cycle and flow in the pulmonary circuit in that heart rate and blood flow both increased during inspiration (Fig. 1). During diving the falling portion of the pulmonary flow trace often showed a more pronounced inflexion and in some animals changed in shape (Fig. 2). This contour of the descending limb of the flow curve was retained during the early period of recovery but flow seldom declined to zero during diastole, the pulsatile component was usually superimposed on a steady flow component.

DISCUSSION

The present results confirm that the intense bradycardia that occurs during diving is accompanied by a reduction in cardiac output of the same order of magnitude as the slowing of heart rate. In this respect the present experiments are in line with observations (Elsner *et al.*, 1964; Murdaugh *et al.*, 1966; Folkow *et al.*, 1967; Cohn *et al.*, 1968) made on diving ducks, geese, seals and sea lion. During recovery from a dive cardiac output increased to around 140 per cent of the control value after 1 min and this order of change has been recorded in geese and seals after short dives (Murdaugh *et al.*, 1966; Cohn *et al.*, 1968). However, it should be pointed out that in some ducks much larger increases in cardiac output have been recorded during recovery from short dives (Folkow *et al.*, 1967).

On average stroke volume increased slightly during diving (15–35 per cent) a result similar to that obtained in geese by Cohn *et al.* (1968). However Folkow *et al.* (1967) report that stroke volumes moderately decreased (10–25 per cent) in their ducks during diving, although in the present experiments only one duck consistently displayed a fall in stroke volume during submergence. Several factors interact to control stroke volume in the normal animal being (1) the preload, which determines the heart muscle fibre diastolic length; (2) the afterload, which is closely related to the intramyocardial systolic tension; and (3) the ionotropic state of the myocardium (Braunwald, 1971). At any given ionotropic state increases in the preload will tend to elevate cardiac output, in accordance with the Starling concept, whereas increases in the afterload will reduce cardiac output. During diving there is a marked increase in venous pressure (Folkow *et al.*, 1967) and in end-diastolic volume of the ventricle (Aakhus & Johansen, 1964) both of which will combine to increase the preload and therefore the stroke volume. Butler & Jones (1971) found that mean arterial pressure fell during a 2-min dive by some 25 mm Hg due to decreases in systolic and diastolic pressure to 95 and 71 per cent respectively. This fall in afterload will likewise tend to increase stroke volume. Consequently without any changes in contractility of the myocardium stroke volume can be expected to increase during submergence.

Obviously changes in load on the ventricle will not be absolutely consistent from animal to animal so that variations in the pattern of change in stroke volume must occur, but, it seems doubtful that the variations will be of such magnitude as to cause a fall in stroke volume without there being a change in myocardial contractility during submergence. In this respect Folkow & Yonce (1967) have

shown that the contractility of the heart ventricles in diving ducks is powerfully depressed by stimulation of the vagal nerves, which form the efferent pathway for the cardiac chronotropic response (Butler & Jones, 1968). Furthermore, Sommer & Johnson (1969) have located specific granules in the ventricles and atria of frog and chicken hearts which are apparently similar to those found only in mammalian atria (Jamieson & Palade, 1964). Sommer & Johnson (1969) suggest that these granules play some role in the sensitivity of cardiac muscle to acetylcholine since presence of these granules is correlated with a negative ionotropic response to the action of acetylcholine (Hoffman & Cranefield, 1960; Folkow & Yonce, 1967). Frogs, which also possess these granules in their hearts' exhibit a marked decrease in stroke volume during submergence (Shelton & Jones, 1965).

The values for cardiac output recorded directly are lower than those obtained by the dye dilution technique. Sturkie (1966) recorded values in the range of 260 ml/min per kg whereas Folkow *et al.* (1967) report values of 560 ml/min per kg. Holeton & Jones (unpublished) found values close to 400 ml/min per kg in two species of duck whereas Jones & Langille (unpublished) only obtained outputs of about half this value, in open chest preparations, using the electromagnetic flow-meter. Differences in the thermal states of the animals examined will tend to cause marked variations in cardiac output as flow to poorly insulated extremities, like the legs, is likely to increase markedly under a positive heat load. Furthermore, it may be that the cuff-type flow probe, causing some constriction of the pulmonary artery, may cause more of the cardiac output to be directed through the other unrestricted vessel. This seems a pertinent point for in one duck, after the probe was in position for 8 weeks, the pulmonary artery became completely occluded but, despite this, cardiac output, when measured by dye dilution, was in the range of 385 ml/min per kg.

The shape of the pulmonary flow pulse in ducks is unlike that recorded from the central pulmonary artery of mammals (Morgan *et al.*, 1966), in that flow is maintained during diastole, but is similar to blood flow pulses recorded in the pulmonary or pulmocutaneous arteries of reptiles and amphibians (Johansen *et al.*, 1970; Shelton, 1970; Jones & Shelton, 1971). The shape of the avian flow pulse implies a large central capacitance so that blood will continue to be fed into the lung capillaries when the ventricle is disconnected from the pulmonary circuit by closure of the valves on the pulmonary outflow tract (Jones & Langille, unpublished). During a dive the diastolic portion of the flow pulse changes shape which implies that the time constant of the pulmonary vascular bed was altered. Eliassen (1960) concluded from recordings of pulmonary arterial pressure in ducks that pulmonary vascular resistance increased during a dive and Aakhus & Johansen (1964) also found that the calibre of the pulmonary arteries was much reduced during diving. Consequently it seems probable that both changes in pulmonary resistance and capacitance promote the change in the shape of the blood flow pulse during diving.

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