J. Physiol. (1971), **214**, *pp.* 457–479
With 6 text-figures
Printed in Great Britain

THE EFFECT OF VARIATIONS IN HEART RATE AND REGIONAL DISTRIBUTION OF BLOOD FLOW ON THE NORMAL PRESSOR RESPONSE TO DIVING IN DUCKS

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(*Received 14 October 1970*)

SUMMARY

1. During a 2 min period of submersion of normal ducks, sciatic artery blood flow fell to $10 \pm 1{\cdot}5\,\%$ control and carotid artery blood flow was reduced to $71 \pm 7\,\%$ control. Mean arterial blood pressure (M.A.P.), however, was maintained at $83 \pm 3{\cdot}5\,\%$ of control. The whole animal showed a constrictor response during submersion, with the sciatic vascular bed showing average constriction. Both resistance to flow and yield pressure increased in the sciatic bed but changed little in the carotid bed. After 1 min submersion P_{a,O_2} was 52 ± 1 mm Hg.

2. Upon emersion, as soon as ventilation commenced, the whole animal showed a dilator response. The carotid bed exhibited marked vasodilatation whereas the sciatic bed returned to its control level.

3. After α-receptor blockade, ducks were submerged for 1 min. During this time M.A.P. fell to $64 \pm 5{\cdot}6\,\%$ of control and heart rate was reduced to $49 \pm 8{\cdot}3\,\%$ of control. Blood flow through the sciatic and carotid arteries also fell to values of $41 \pm 6{\cdot}9\,\%$ of control and $91 \pm 13\,\%$ of control respectively. There was little change in either resistance to flow or yield pressure in the sciatic bed compared to normal ducks, and the carotid bed showed reductions in resistance to flow and yield pressure during submersion. P_{a,O_2} after 1 min under water was $41 \pm 1{\cdot}1$ mm Hg.

4. β-receptor blockade had no effect on any of the measured variables during submersion. Upon surfacing, however, although the whole animal response was one of dilatation, the carotid bed was less dilated than in normal ducks at this time and the sciatic bed was more constricted.

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5. Injection of atropine not only abolished the bradycardia during submersion but also caused a rise in M.A.P. and sciatic blood flow during the period under water. After 1 min submersion P_{a,O_2} was $30 \pm 1{\cdot}2$ mm Hg.

6. It is concluded that stimulation of adrenergic α-receptors is responsible for the increase in resistance to flow through the sciatic artery and the maintenance of blood pressure during submersion in the normal animals. This selective constrictor activity and the resulting ischaemia is important in the maintenance of P_{a,O_2} during submersion. Adrenergic β-receptors (cardiac and/or peripheral) are involved, to a small extent, in the blood pressure and blood flow changes that occur when ventilation commences upon emersion.

INTRODUCTION

Mean systemic blood pressure of diving mammals and birds is maintained, during a dive, in spite of the pronounced diving bradycardia (Irving, Scholander & Grinnell, 1942; Hollenberg & Uvnäs, 1963; Folkow, Nilsson & Yonce, 1967). Consequently either stroke output and/or peripheral resistance must increase in order to compensate for the bradycardia. However, cardiac output decreases during a dive (Elsner, Franklin & van Citters, 1964; Folkow *et al.* 1967), so it follows that peripheral resistance must increase. By use of the thermostromuhr technique and from measurements of blood lactate, a reduction in muscle blood flow during submersion of seals and ducks was postulated by Scholander, Irving & Grinnell (1942) and Grinnell, Irving & Scholander (1942). Johansen (1964) using a radioisotopic technique concluded that in ducks large masses of skeletal muscle become ischaemic during submersion whereas the oesophagus, brain and heart become hyperaemic. But these results are at variance with the only direct measurements of muscle blood flow in ducks. Hollenberg & Uvnäs (1963) found no reduction in blood flow in the sciatic artery, which supplies a largely muscular area, and suggested that during short dives arteriovenous shunts may be in operation thereby short circuiting the muscle capillaries but not reducing total flow.

The present investigation was concerned with direct measurement of the blood flow in the carotid and sciatic arteries of the duck. The results are presented and analysed in terms of the pressor response to submergence. Finally, the significance of the cardiovascular adjustments to diving, in regard to survival, was investigated by recording P_{a,O_2} during dives in the normal animal, after α-adrenergic receptor blockade, and after injection of atropine.

METHODS

The experiments were performed on domestic ducks (*Anas platyrhynchos*) of either sex, weighing between 1·8 and 3·9 kg. During experiments the birds were restrained horizontally, ventral side down. Heart rate and respiratory frequency were determined as described previously (Butler & Jones, 1968).

All of the operations were of a minor nature and were performed after a local injection of 2 % (w/v) lignocaine hydrochloride with adrenalin 1:80,000 (Xylocaine, Astra-Hewlett Ltd.). This produced sustained local anaesthesia and the animal showed no signs of distress either during or after the operation. Periods of 2–20 hr were allowed to elapse after any operation before the start of the experiment. No quantitative differences in the responses of the ducks could be related to the length of the recovery period. All animals that had undergone operations were killed at the end of an experiment with an overdose of pentobarbitone sodium (Nembutal; Abbott).

Blood pressure was measured either from the sciatic or brachial artery. In either case a short length of polyethylene cannula (15 cm long, 0·1 cm bore) was inserted into a side branch of the major vessel. The cannula was connected to a Sanborn 267B pressure transducer. With this arrangement the catheter-transducer-recorder had a natural frequency of 35 Hz and damping was 20 % of critical. Just before an experiment the bird was injected with 500 i.u. heparin/kg (Evans Medical Ltd.) and the manometer filled with heparinized White's avian saline (Lockwood, 1961).

Blood flow was measured in the left sciatic and left common carotid arteries by a Biotronex BL610 pulsed-logic electromagnetic flowmeter (Biotronex Laboratory Inc.) 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 % over the full range). The sciatic artery was exposed at the proximal end of the femur and the common carotid artery was displayed as low down the neck as possible without puncturing the cervical air sac. This position was invariably just after the vessel had penetrated the mid line of the ventral musculature of the neck. A peri-arterial 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-probe constricted the cross-sectional area of the vessel by no more than 15 %, and this amount of restriction is thought to have a negligible effect on flow (Spencer & Denison, 1956). Zero flow was obtained by occluding the artery distally with a pneumatic cuff; 'pounding' during occlusion was not noticeable. Zero flow was established before, during and after submersion of an animal, and the presence of the deflated cuff had no effect on blood flow in the artery. The flow transducers were calibrated *in vivo*. At the end of a series of experiments the duck was injected with 500 i.u. heparin/kg and anaesthetized with pentobarbitone sodium (30 mg/kg). The artery was cannulated distal to the flow meter probe and the cannula was connected to a vertical 10 ml. syringe. Blood flow through the transducer was regulated by a screw clamp applied to the cannula. At different flow rates, the time taken for a given volume of blood to be delivered into the syringe was measured and a calibration curve plotted. The blood collected in the syringe was injected back into the animal after each delivery. Consequently each flow probe was calibrated while still in the position on the artery that it occupied during an experiment.

$P_{a,\,O_2}$ was measured continuously by cannulating one carotid artery and circulating blood through a thermostatically controlled glass cuvette containing a Radiometer oxygen electrode and back into the artery. The blood and electrode were

maintained at 41·5 ± 0·2° C and the electrode was calibrated at this temperature with air-equilibrated saline and nitrogen gas. The same reading was obtained for air-equilibrated duck blood and air-equilibrated saline at 41·5° C. The 90 % response time was 3–5 sec for the oxygen electrode and circulation time through the cannula-cuvette-cannula system was of the order of 1–2 sec in the resting animal. Output of the oxygen electrode was unaffected by positive pressure and was independent of flows from 0 to 50 ml./min.

Drugs were administered through a cannula inserted into the left brachial vein. Adrenergic α-receptor blockade was achieved by injecting 10 mg/kg of phenoxybenzamine (Dibenyline; Smith, Kline & French Ltd.). Before α-blockade injection of adrenaline bitartrate, 5 μg/kg (Sigma Chemicals Ltd.), caused a significant rise in blood pressure and a significant reduction in sciatic artery blood flow ($P < 0{\cdot}05$). Dibenylene depressed blood pressure and completely abolished the response to adrenaline injection. Stimulation of the β-receptors by injection of isoprenaline sulphate 5 μg/kg (Sigma Chemicals Ltd.), had no significant ($P < 0{\cdot}05$) effect on blood flow or blood pressure but it did cause a significant tachycardia. Injection of propanolol 0·5 mg/kg (Inderal; I.C.I. Ltd.), a β-blocker, had no effect on any cardiovascular parameter but it abolished the tachycardia caused by injection of isoprenaline. Acetylcholine chloride (Sigma Chemicals Ltd.) was given at a dose of 5–10 μg/kg and the hypotension caused by this was blocked by injection of atropine sulphate, 2·5 mg/kg (Sigma Chemicals Ltd.). The effectiveness of each blocking agent was determined before and after every dive. All cardiovascular recordings were displayed on either a Sanborn 966 six-channel recorder or a Devices M4, four-channel instrument; both instruments write on rectilinear co-ordinates.

In the present account, the words 'dive', 'submersion', 'under water' and 'submergence' signify immersion of only the duck's head into water while 'surface', 'emersion' and 'emergence' indicate removal of the head from water (Butler & Jones, 1968). Several dives were usually performed on each duck. Dives were of a maximum of 2 min duration and were interspersed with rest periods of 30–45 min. It was found that brief introductory dives whose duration was gradually increased enabled the birds to endure the experimental dives with little sign of discomfort or stress.

In the present account 'left' and 'right' refer respectively to the left and right sides of the animal. The word 'normal' when applied to the ducks means before injection of any drugs and 'control' describes the response of the animal before submergence under any conditions. Mean arterial blood pressure (M.A.P.) was calculated from the formula M.A.P. = diastolic pressure + ($f \times$ pulse pressure). The factor, f, was not a constant but varied with heart rate and the particular treatment to which the animal had been exposed. For each set of conditions f was calculated and the value of mean pressure, obtained from the formula, was compared with measurements of mean pressure obtained by the 'area under the curve' method. Ultimately it was possible to construct a curve of the variation in f with heart rate for a given set of conditions. The agreement between calculated and measured values was checked periodically and was found to be ± 2 %. All numerical values are given ± S.E. of mean. The results were analysed statistically by means of Fisher's t test and 5 % ($P < 0{\cdot}05$) was considered the fiducial limit of significance. All experiments were carried out at room temperature (18–20° C).

RESULTS

The normal pressor response during diving

A total of thirty-six dives were performed on thirteen normal ducks and the mean results are given in Fig. 1*a*. During a 2 min dive, heart rate decreased to $17 \pm 1{\cdot}3\,\%$ of control but the onset of bradycardia was slow, full expression of the response taking 30–60 sec. No significant changes in heart rate occurred during the second minute of the dive, but there were significant changes in both systolic and diastolic blood pressure during this period. Systolic blood pressure fell to $95 \pm 3{\cdot}4\,\%$ of control and diastolic to $71 \pm 4{\cdot}2\,\%$ of control. Due to the marked change in diastolic pressure, pulse pressure increased and M.A.P. decreased.

Sciatic minute blood flow fell during 2 min submergence to $10 \pm 1{\cdot}5\,\%$ of the control value whereas carotid minute blood flow was only reduced to $71 \pm 7\,\%$ of control (Fig. 1). The changes in minute flow in both the sciatic and carotid arteries were not related in a simple manner to the bradycardia. Stroke flow in the sciatic artery fell from $0{\cdot}204 \pm 0{\cdot}0085$ (control value) to $0{\cdot}121 \pm 0{\cdot}0095$ ml. (2 min submergence) but at the same time stroke flow in the carotid artery increased from $0{\cdot}0943 \pm 0{\cdot}0077$ (control) to $0{\cdot}357 \pm 0{\cdot}037$ ml. (2 min submergence). The values at 2 min submergence were not significantly different from those recorded after 1 min of submergence.

When the animal was at the surface, the flow pulse in the sciatic artery was maintained well above zero, whereas in the carotid artery during diastole, the flow approached zero. In no case was reversal of flow recorded in surfaced animals (Fig. 1*b*). During a dive the sciatic artery flow pulse invariably fell to zero during diastole as did the carotid flow pulse on many occasions.

Both in the surfaced and submerged animal the flow pulses had similar characteristics. At the start of systole, flow increased rapidly and after peak flow was reached it declined rapidly until this fall was interrupted by the start of a more gradual decline (Fig. 1*b*). On a number of occasions sciatic pressure and flow were traced on the recording paper within 1 cm of each other. It was established from these recordings that peak flow was reached before peak pressure and that flow declined rapidly until the incisura on the pressure pulse. After the incisura both pressure and flow fell more gradually (Fig. 1*b*). In all pressure records it was noticed that the incisura was more pronounced during submergence and tended to occur closer to the peak systolic pressure.

During recovery from a dive, heart rate increased rapidly and within 5 sec was $171 \pm 9\,\%$ of control. Systolic and diastolic pressures rose and after 5 sec mean blood pressure was $115 \pm 3{\cdot}7\,\%$ of control. All these values

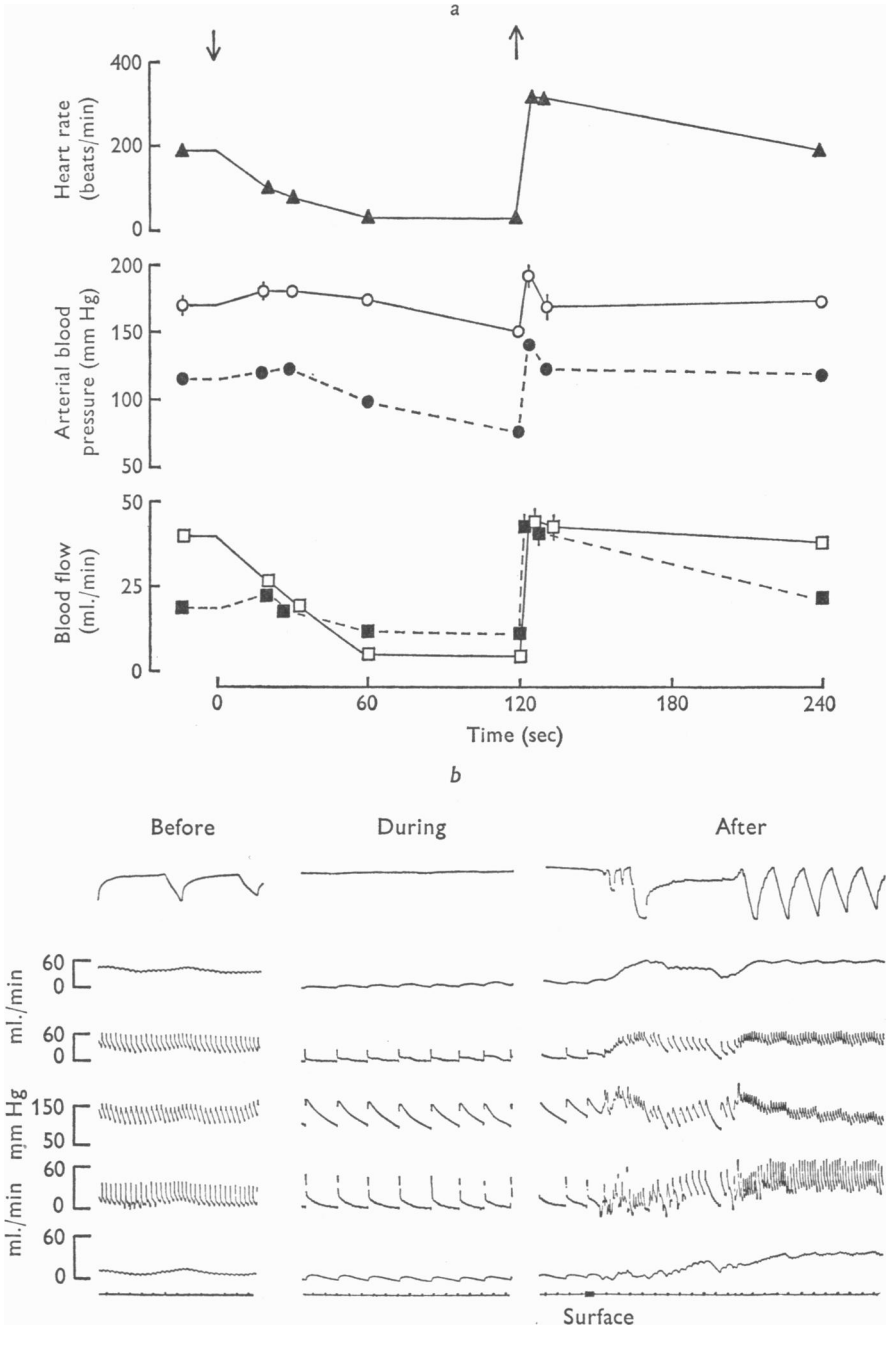

Fig. 1. For legend see opposite page.

were significantly different from control. These changes only occurred when rhythmic pulmonary ventilation commenced. Fig. 1*b* illustrates this. It shows one of several occasions when the duck's head was taken out of water without immediate tachypnoea taking place. Following emersion there was one initial respiratory movement and this was accompanied by an increase in heart rate and blood flow, but it was not until normal rhythmic tachypnoea occurred that sustained cardiovascular adjustments were manifest. Sciatic minute blood flow returned almost immediately to control levels although, due to the large increase in heart rate, stroke flow was little different from the value recorded after two minutes submergence. However, carotid flow increased to $240 \pm 16{\cdot}5\%$ of control and stroke flow was now $0{\cdot}135 \pm 0{\cdot}0014$ ml., significant increases over control values. Within 2 min of the restoration of normal respiratory movements all the recorded cardiovascular variables had returned to control levels. Respiratory frequency showed changes similar to those reported by Butler & Jones (1968).

In the restrained, unanaesthetized duck the amount of blood flowing through one sciatic artery was about twice that flowing through one common carotid artery (Fig. 1). Therefore the resistance of the vascular bed supplied by the common carotid was about twice that of the bed supplied by the sciatic artery. This relationship changed markedly when the duck's head was placed underwater. Calculation of vascular resistance by the formula

$$\frac{\text{Mean arterial blood pressure (mm Hg)}}{\text{Blood flow along the particular artery (ml./min)}}$$

assuming that venous pressure remained constant, showed that vascular resistance in the sciatic artery increased by some 7·97 times whereas

Legend to Fig. 1.

Fig. 1. Effect of submersion on normal ducks. *a*. Mean results from thirty-six dives on thirteen ducks (for carotid artery blood flow, mean values are from twenty-four dives on eight ducks). Downward pointing arrow indicates submersion, upward pointing arrow indicates emersion. From above, downwards: ▲, heart rate; ○, systolic pressure; ●, diastolic pressure; □, sciatic artery blood flow; ■, common carotid artery blood flow. In each case, vertical lines indicate ± S.E. of mean. Where vertical lines are absent, the S.E. of mean is within the limits of the symbol. *b*. *A platyrhynchos*. Traces showing arterial blood pressure, and blood flow in the carotid and sciatic arteries, before, during and after a normal dive of 2 min duration. In each series the traces from top to bottom are: pneumogram (down on trace, inspiration); mean blood flow through sciatic artery; pulsatile blood flow through sciatic artery; arterial blood pressure; pulsatile blood flow through common carotid artery; mean blood flow through common carotid artery; time marker in sec.

TABLE 1. Changes in peripheral resistance of the sciatic and carotid vascular beds during diving and recovery. All values are in P.R.U.S (1 P.R.U. = 79,600 dynes sec. cm. -5), and are given $\pm$ S.E. of mean. Each value is the mean of n observations. The figure in parentheses after n is the number of animals upon which the observations were made

		Normal		β-block		α-block		Atropine
		Sciatic $n = 36$ (13)	Carotid $n = 24$ (8)	Sciatic $n = 16$ (8)	Carotid $n = 8$ (5)	Sciatic $n = 18$ (7)	Carotid $n = 12$ (5)	Sciatic $n = 8$ (3)
Control		$3{\cdot}6 \pm 0{\cdot}103$	$7{\cdot}8 \pm 0{\cdot}222$	$3{\cdot}5 \pm 0{\cdot}187$	$7{\cdot}2 \pm 0{\cdot}384$	$2{\cdot}7 \pm 0{\cdot}159$	$6{\cdot}3 \pm 0{\cdot}418$	$4{\cdot}5 \pm 0{\cdot}317$
Dive	30 sec	$8{\cdot}1 \pm 0{\cdot}323$	$8{\cdot}2 \pm 0{\cdot}327$	$5{\cdot}9 \pm 0{\cdot}289$	$5{\cdot}6 \pm 0{\cdot}300$	$3{\cdot}5 \pm 0{\cdot}195$	$4{\cdot}3 \pm 0{\cdot}244$	$5{\cdot}3 \pm 0{\cdot}313$
	60 sec	$27{\cdot}4 \pm 0{\cdot}958$	$10{\cdot}6 \pm 0{\cdot}371$	$23{\cdot}6 \pm 0{\cdot}784$	$8{\cdot}1 \pm 0{\cdot}268$	$4{\cdot}6 \pm 0{\cdot}341$	$4{\cdot}3 \pm 0{\cdot}469$	$5{\cdot}3 \pm 0{\cdot}303$
	120 sec	$28{\cdot}7 \pm 1{\cdot}25$	$9{\cdot}7 \pm 0{\cdot}424$	$35{\cdot}2 \pm 1{\cdot}597$	$7{\cdot}4 \pm 0{\cdot}334$	—	—	—
Emergence	5 sec	$3{\cdot}7 \pm 0{\cdot}138$	$3{\cdot}8 \pm 0{\cdot}142$	$4{\cdot}7 \pm 0{\cdot}300$	$4{\cdot}1 \pm 0{\cdot}263$	$2{\cdot}6 \pm 0{\cdot}244$	$3{\cdot}0 \pm 0{\cdot}431$	$4{\cdot}8 \pm 0{\cdot}514$
	10 sec	$3{\cdot}4 \pm 0{\cdot}169$	$3{\cdot}6 \pm 0{\cdot}176$	$3{\cdot}9 \pm 0{\cdot}252$	$3{\cdot}9 \pm 0{\cdot}254$	—	—	$5{\cdot}7 \pm 0{\cdot}602$
	120 sec	$3{\cdot}9 \pm 0{\cdot}142$	$6{\cdot}9 \pm 0{\cdot}251$	$3{\cdot}5 \pm 0{\cdot}182$	$6{\cdot}2 \pm 0{\cdot}323$	$3{\cdot}3 \pm 0{\cdot}245$	$6{\cdot}6 \pm 0{\cdot}641$	$5{\cdot}5 \pm 0{\cdot}405$

resistance to flow in the bed supplied by the carotid artery only increased by 1·24 times (Table 1). These values must represent the extremes, since it is well known that venous pressure rises substantially during a dive of 2 min duration (Johansen & Aakhus, 1963; Folkow *et al.* 1967).

The relationship between pressure and flow in the sciatic and carotid vascular beds is illustrated in Fig. 2. Pressure and flow pulses were analysed from the instant of peak flow to the start of the next cardiac contraction. The pressures and flows are expressed as percentages of maximum pressure and flow recorded in the pulses which were analysed. Approximately one hundred points from 10 to 20 pressure and flow pulses were utilized to construct each 'run-off' curve. Pressure recordings were made from the sciatic artery and since peak flow is established before peak pressure the 'run-off' curves for the sciatic vascular bed are initially concave to the flow axis. The same pressure recordings were used to construct run-off curves for the carotid vascular bed and consequently the phase relationships between the pressure and flow pulses must necessarily be inaccurate.

The falling slope of the 'run-off' curve gives some indication of the resistance of the vascular bed. For the sciatic bed the slope of the line increased about 4 times during 1 min submergence. During submergence zero flow occurred at 56% of the peak pressure and this was approximately twice as high as the yield pressure obtained by extrapolation of the slope for the surfaced animal back to the ordinate. The slope of the line for the carotid bed changed little during 1 min submergence, increasing by 1·15 times, and the yield pressure only rose from 46 to 56% of the peak pressure.

During recovery there were intense changes in resistance in the two vascular beds (Table 1). Resistance in the sciatic vascular bed returned to control levels within 5 sec whereas resistance in the carotid bed decreased to approximately half of control, taking approximately 120 sec before the value was not significantly different from control. It was not possible to construct accurate pressure–flow curves for this part of the experiment.

Factors affecting the normal pressor response to diving

α-adrenergic receptor blockade. Dibenyline was injected into a total of seven ducks. Five of these animals, upon which twelve submersions were performed, showed reductions in mean blood pressure to $49 \pm 2{\cdot}5\%$ of control and in heart rate to $27 \pm 1{\cdot}9\%$ of control after 1 min submergence. Six dives were performed on the remaining two ducks, and although during the first 20 or 30 sec of submersion both mean blood pressure and heart rate fell slightly, after 1 min under water both of these variables had returned to control levels. Submersion usually lasted for 1 min with ducks after α-blockade as they often became agitated if maintained under water

for a longer period. However, when any of the five ducks which showed hypotension and bradycardia after 60 sec submersion were kept under water for longer than 60 sec, then heart rate increased and mean blood pressure returned to control level after about 90 sec submersion.

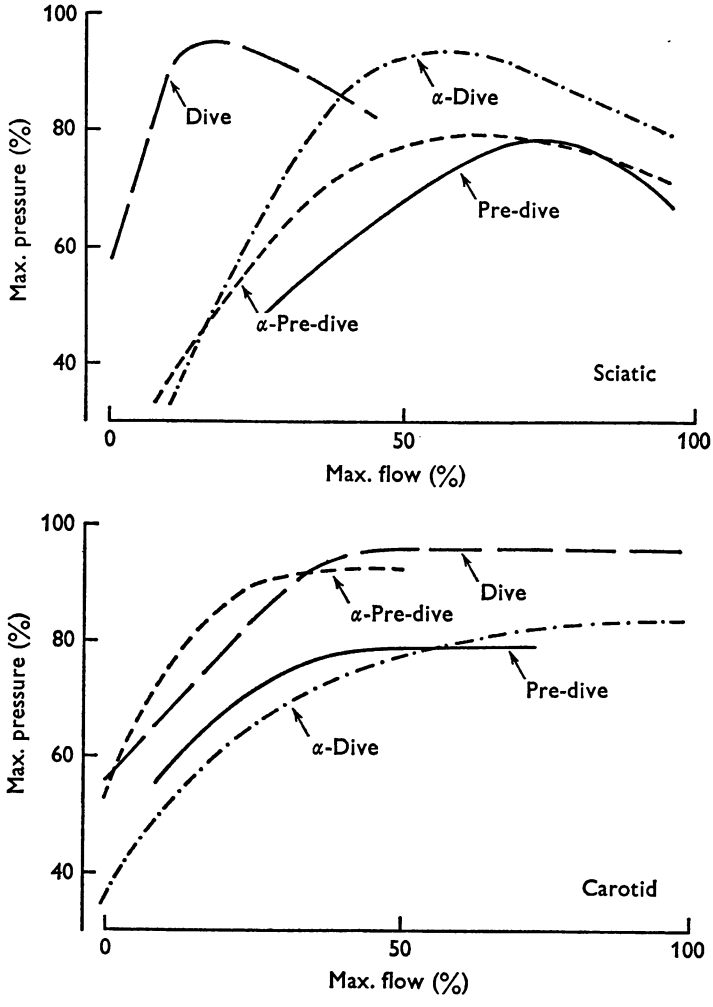

Fig. 2. Relationship between pressure and flow in the sciatic and carotid vascular beds in normal and α-blocked ducks. Average values from analysis of ten to twenty cardiac cycles from the instant of peak flow to the start of the next cardiac contraction. Each pair of pulses was broken down into fifteen to twenty points and the values were measured at these points. The pressures and flows were expressed as % of the maximum pressure and flow attained in the cardiac cycles analysed, and averaged graphically. Pressure and flow pulses were analysed before and after 1 min submersion in both normal and α-blocked ducks. For further details see text.

Fig. 3 shows the mean results of all the dives performed on ducks after α-blockade. Injection of Dibenyline caused a significant increase in heart rate compared with normal ducks, but mean blood pressure, sciatic artery and carotid artery minute flows were not significantly changed from normal. Mean blood pressure showed a progressive decline during submersion, and after 60 sec under water had fallen to $64 \pm 5{\cdot}6\,\%$ of control, mainly due to a reduction in diastolic pressure. This value was significantly

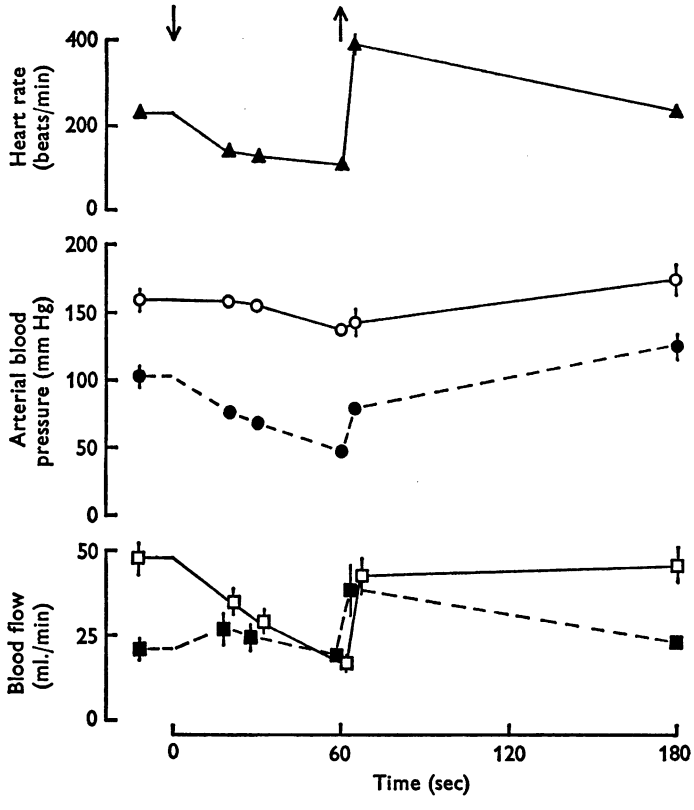

Fig. 3. Effect of submersion on ducks after α-receptor blockade. Mean results from eighteen dives on seven animals (for carotid artery blood flow, mean values are from twelve dives on five ducks). Notation as in Fig. 1.

lower than that recorded in normal ducks after 60 sec submersion. During the first 20 sec of submergence the percentage change in heart rate was similar to that in normal ducks, but after 60 sec under water it was significantly higher than in normal animals ($49 \pm 8{\cdot}3\,\%$ of control). A similar pattern was evident with blood flow through the carotid and sciatic arteries. During the first 20 sec of submersion, both variables followed the same pattern as in normal ducks. After 60 sec under water blood flow

through the sciatic artery was 41 ± 6·9 % of control and flow through the carotid artery was 91 ± 13 % of control. Both of these values are higher than those recorded in normal ducks, but only the former is significantly greater.

During control conditions resistance in both the sciatic and carotid vascular beds was significantly lower than in normal ducks (Table 1). During 60 sec submersion resistance to flow through the sciatic artery increased by almost 75 % (Table 1) and this is significantly lower than the change recorded in normal ducks. Resistance to flow through the carotid artery fell by almost 30 % during 60 sec submersion. This is the opposite to the change recorded in normal animals after 60 sec under water (Table 1). Upon emersion heart rate increased within 5 sec to 168 ± 14 % of control, sciatic flow returned to its control value and flow through the carotid artery was 180 ± 22 % of control.

Pressure–flow 'run-off' plots for both the sciatic and carotid vascular beds showed that resistance changed little in the sciatic vascular bed during 1 min submergence; yield pressure in both control and diving animals was low and of the order of 12–22 % of peak pressure (Fig. 2). Resistance and yield pressure in the carotid vascular bed decreased during a 1 min dive. These changes in resistance were obviously very different from those shown by normal ducks. During recovery the whole animal response was dilatation.

β-adrenergic receptor blockade. Injection of Inderal caused a slight reduction in heart rate compared with normal ducks and the situation is clearly different from that seen in the chicken (Butler, 1967). This may be because there is in fact a higher resting level of sympathetic activity in the chicken's heart, or that the fowl is more sensitive to handling and restraint thereby giving abnormally high sympathetic tone.

Sixteen dives were performed on eight ducks after β-receptor blockade, and apart from a few details the cardiovascular changes in these ducks during 2 min submergence were the same as in normal animals (Fig. 4*a*). There were no significant changes in systolic or diastolic levels of blood pressure during submergence. However, following emersion the normal pattern of recovery was not observed (Fig. 4*b* and *c*). Neither carotid nor sciatic artery blood flow showed the same initial degree of increase as in normal ducks and mean blood pressure did not increase (Fig. 4*a*). However, the tachycardia on emersion was unaffected by β-blockade. Two minutes after emersion all measured variables had virtually returned to control levels.

The resistance of the sciatic vascular bed increased by 10 times during a 2 min dive whereas carotid resistance showed little change (Table 1). Resistance in both the sciatic and carotid vascular beds fell 5 sec after

emergence. Sciatic resistance was 133% of control and carotid resistance was 57% of control, both these values were higher than those recorded in normal ducks.

Injection of atropine. It is well known that atropine abolishes diving bradycardia in ducks but its effect on other cardiovascular variables in these animals is not known. Following atropine injection, animals were only submerged for 1 min (see Butler & Jones, 1968). Carotid artery blood flow was not measured in these experiments.

Fig. 5 shows the mean results of eight dives on three ducks after injection of atropine. As previously reported (Butler & Jones, 1968) atropine itself caused a significant increase in heart rate in ducks, although systolic and diastolic blood pressures were not significantly different from those of normal animals. During 1 min submergence there was no bradycardia, heart rate in fact increased to $110 \pm 7\%$ of control. Both systolic and diastolic blood pressure increased throughout the period of submergence finally reaching levels some 60% above control (Fig. 5) and sciatic stroke and minute blood flows also increased significantly. During recovery heart rate increased and after 5 sec at the surface was $126 \pm 7{\cdot}8\%$ of control whereas systolic and diastolic blood pressure had fallen to $113 \pm 15{\cdot}4\%$ and $122 \pm 9{\cdot}5\%$ of control. Sciatic blood flow decreased on emersion from the level recorded after 1 min under water. Within 2 min all recorded cardiovascular variables had returned to control levels (Fig. 5). Vascular resistance in the sciatic bed did not change significantly during 1 min submergence (Table 1). There was a slight increase in resistance which may have been indicative of active vasoconstriction. As during the dive, during recovery there were only slight and non-significant changes in vascular resistance.

P_{a,O_2} *during diving in normal, α-blocked and atropinized ducks*

Actual traces of P_{a,O_2} during submersion are shown in Fig. 6*a* and *b*. Although arterial P_{O_2} was measured continuously, due to time lags in the registration of the actual P_{a,O_2} at any instant in time, no claims can be made that these traces accurately reflect temporal relationships during a dive, although the results are qualitatively and in many respects quantitatively similar to those recorded by Jones & Purves (1970), using a faster responding oxygen electrode in an arteriovenous loop (Purves 1966).

In all, seven ducks were used to test the effect of α-blockade and the injection of atropine on P_{a,O_2} during submersion. Two normal dives were performed on each of these animals. Four of the ducks were then injected with Dibenyline and two more dives of 1 min duration were performed on each of these. Although P_{a,O_2} followed the same course during the first 20 sec of submergence in both normal and α-blocked ducks, after 60 sec under

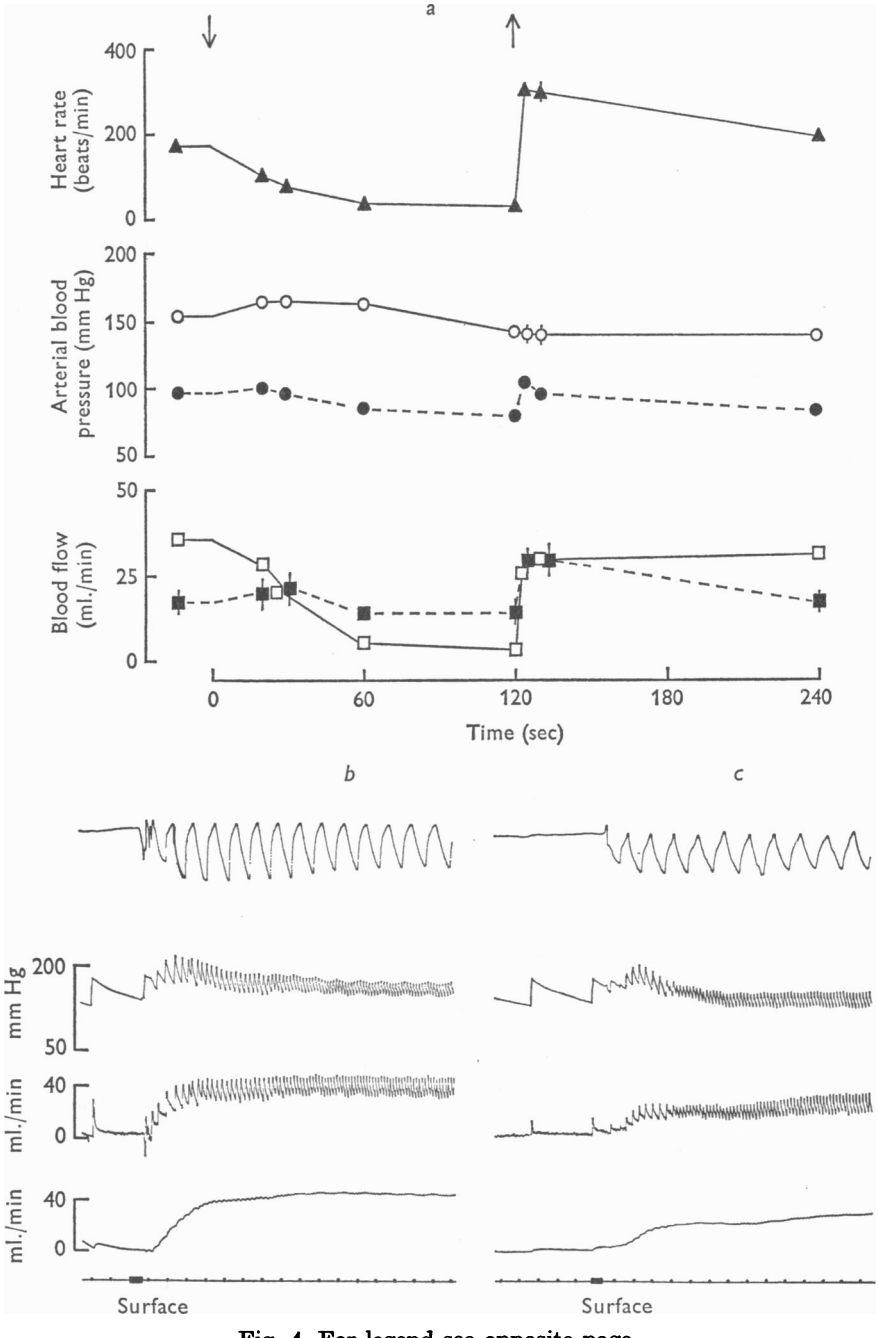

Fig. 4. For legend see opposite page.

water P_{a,O_2} was significantly lower after α-blockade (Fig. 6*a* and Table 2). Mean heart rate of all four α-blocked animals after 60 sec submergence was $60 \pm 7\%$ of control. Heart rate of the normal animals was similar to that cited earlier (see Fig. 1). Recovery of P_{a,O_2} after emersion was somewhat faster in α-blocked than in normal ducks, although in both cases control P_{a,O_2} was reached within 20 sec, and then rose above control where it remained for 50–60 sec before it began to fall back to the control level.

Three ducks, after their normal submersions, were injected with atropine before each being subjected to two dives of 1 min duration. P_{a,O_2} followed the same course in the ducks injected with atropine during the first 20 sec

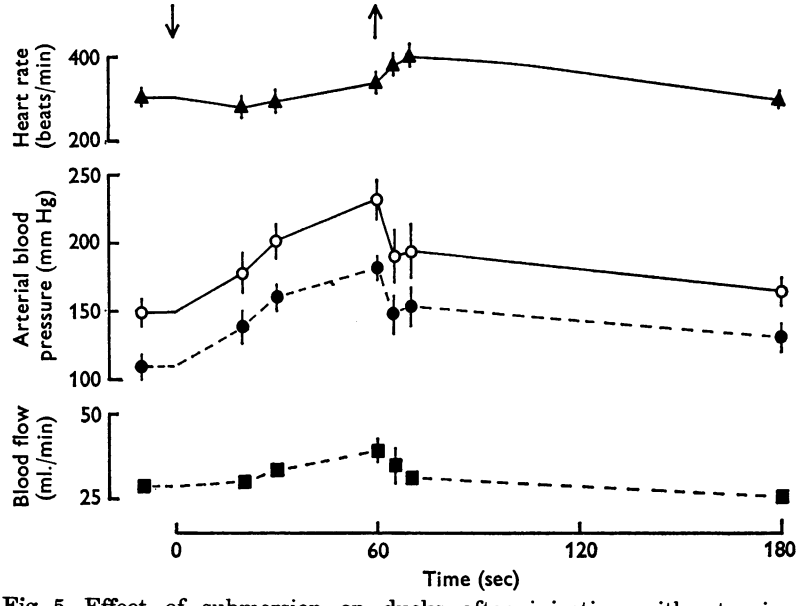

Fig. 5. Effect of submersion on ducks after injection with atropine. Mean results from eight dives on three ducks. Notation as in Fig. 1, except that measurements on carotid artery flow were omitted in the present experiments.

Legend to Fig. 4.

Fig. 4. Effect of submersion on ducks after β-receptor blockade. *a*. Mean results from sixteen dives on eight ducks (for carotid artery blood flow, mean values are from eight dives on five ducks). Notation as in Fig. 1. *b*. Traces showing effect of emersion on sciatic artery blood flow and arterial blood pressure in normal duck, *A. platyrhynchos*. *c*. Traces showing effect of emersion on sciatic artery blood and arterial blood pressure in the same duck after β-receptor blockade. In both *b* and *c*, traces from top to bottom are: pneumogram (down on trace, inspiration); arterial blood pressure; pulsatile flow through the sciatic artery; mean flow through the sciatic artery; time marker in sec.

of submersion as in normal ducks. After 1 min under water, however, P_{a,O_2} was significantly lower than in both normal and α-blocked ducks (Fig. 6*b* and Table 2). Heart rate changes during submersion after injection of atropine were similar to those in Fig. 5. Recovery of P_{a,O_2} after surfacing was initially slower after injection of atropine although the control value was reached within 20 sec and subsequently exceeded. This elevated level was maintained for 40–50 sec before it returned to the control value.

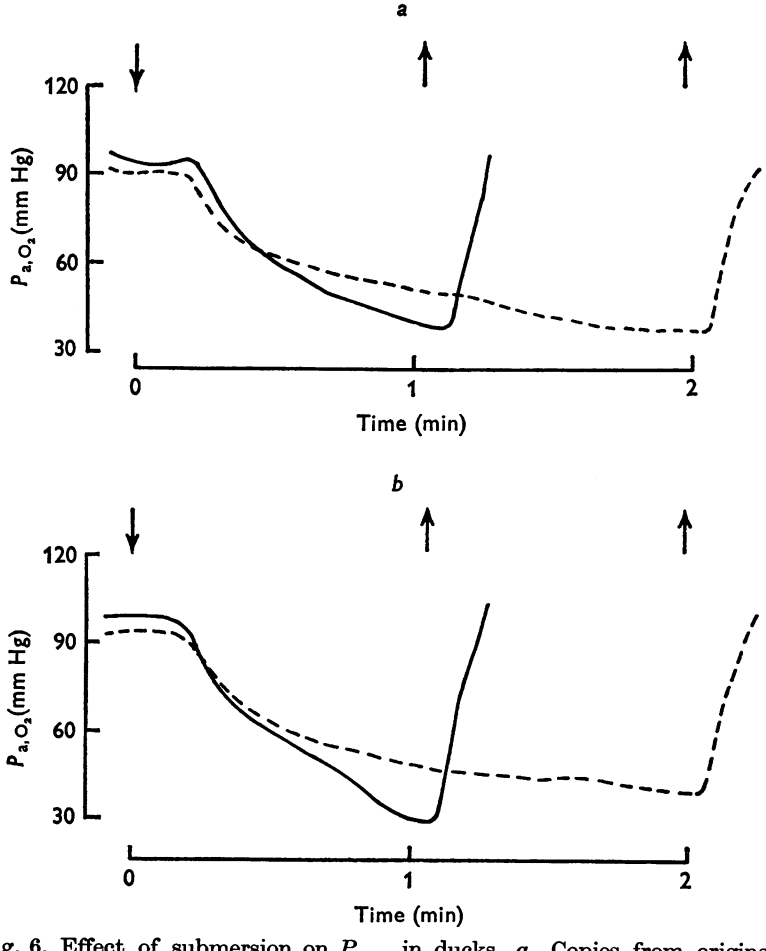

Fig. 6. Effect of submersion on P_{a,O_2} in ducks. *a*. Copies from original traces of P_{a,O_2} during submersion in a normal duck, *A. platyrhynchos* 1·75 kg (----) and in the same animal after α-receptor blockade (—). *b*. Copies from original traces of P_{a,O_2} during submersion in a normal duck, *A. platyrhynchos* 2·65 kg (---) and in the same duck after injection of atropine (——). In each case arrows indicate submersion and emersion as in Fig. 1.

TABLE 2. Effect of α-receptor blockade and injection of atropine on P_{a,O_2} during submersion in ducks. Mean values ± S.E. of mean from n observations. Figure in parentheses after n gives the number of animals upon which the observations were made

		P_{a,O_2} (mm Hg)		
		Normal $n = 14(7)$	α-block $n = 8(4)$	After atropine $n = 6(3)$
Control		93 ± 1·5	95 ± 1·5	96 ± 1·6
Dive	10 sec	87 ± 1·3	87 ± 1·2	91 ± 1·7
	20 sec	71 ± 1·5	70 ± 1·3	69 ± 2·2
	60 sec	52 ± 1·0	41 ± 1·1	30 ± 1·2
	120 sec	40 ± 1·5	—	—
Emergence	10 sec	66 ± 3·8	83 ± 4	50 ± 7
	20 sec	90 ± 3·7	100 ± 2	90 ± 6·4
	60 sec	104 ± 1·7	103 ± 1·5	105 ± 1·6

DISCUSSION

There can be no doubt that the duck undergoes profound cardiovascular adjustments during even a short dive. Assuming no changes in central elasticity, since there is such a marked change in input to the systemic circulation then blood pressure will only be maintained by a reduction in output from the central arterial bed. The reduction in output is achieved by marked vasoconstriction in many vascular beds including that supplied by the sciatic artery. This not only has the effect of reducing total flow but also raises the yield pressure which has to be overcome before measurable flow can commence.

Measurement of the input impedance of the sciatic and carotid vessels would provide a much more adequate description of the cardiovascular adjustments to diving. Unfortunately this has not been possible and we are left with a somewhat rudimentary analysis of the response in d.c. terms. The resistance of the sciatic vascular bed increases about 8 times during a dive whereas the carotid vascular bed only changes by 1·24 times. After 2 min under water the resistance of the carotid vascular bed is only one-third of the sciatic, whereas carotid resistance was twice that of the sciatic vascular bed at the surface.

Iriuchijima, Koike & Matsuda (1969) have suggested a more sophisticated approach to the d.c. analysis of resistance changes by the use of indices of participation and contribution to any cardiovascular reflex shown by a particular vascular bed. The index of participation in any cardiovascular reflex is established by comparing the response shown by that vascular bed with that of the whole body. The index of contribution assesses the role of that particular vascular bed in the whole body response. Of course the latter is very much a function of the cardiac output.

During a dive of 2 min duration the whole animal showed a constrictor response. The indices of participation for the sciatic and carotid vascular beds, after 2 min under water, were 1·36 and 0·11 respectively (Table 3). Obviously 1·0 represents an average constriction so the sciatic bed constricted somewhat more than average whereas constriction of the carotid vascular bed was slight.

The index of contribution of the regional vascular bed to the total reflex response is inversely proportional to the stroke volume of the heart. Sturkie (1966) reported cardiac outputs of the order of 286·8 ml./min.kg

TABLE 3. Indices of participation and contribution for the sciatic and carotid vascular beds in the cardiovascular response to diving. Indices obtained from mean values used in Fig. 1. Control stroke volume assumed to be 5 ml. (Folkow *et al.* 1967). Venous pressure and stroke volume assumed to change in like manner to ducks investigated by Folkow *et al.* (1967)

		Index of participation = $\frac{1(i\Delta P - P\Delta i)}{i(I\Delta P - P\Delta I)}$*		Index of contribution = $\frac{i\Delta P - P\Delta i}{I\Delta P - P\Delta I} \times 100$*	
		Normal		Normal	
		Sciatic	Carotid	Sciatic (%)	Carotid (%)
Dive	20 sec	0·74	−0·53	3	−1
+ = constriction	30 sec	0·96	0·00	3	0
− = dilatation	60 sec	0·24	0·25	5	0
	120 sec	1·36	0·11	5	0
Emergence	5 sec	0·01	1·85	0	3
+ = dilatation	10 sec	0·08	1·61	0	3
− = constriction					

* I = control cardiac output (ml./min).
i = control blood flow along carotid or sciatic artery (ml./min).
P = control mean blood pressure (mm Hg).
ΔI = change in cardiac output (ml./min).
Δi = change in flow along carotid or sciatic artery (ml./min).
ΔP = change in mean blood pressure (mm Hg).

whereas Folkow *et al.* (1967) recorded cardiac outputs about twice this value. Approximately twenty attempts have been made by the authors to measure stroke output of the heart using various types of cardiometer (Rushmer, 1961). The results were variable and allowed no quantitative estimate to be made, however; it appeared that stroke volume changed little during 2 min submergence confirming the results of Folkow *et al.* (1967). At a control stroke volume of 5 ml. as used in the present analysis the contribution of the sciatic bed to the total reflex response is only 5 % (Table 3).

The whole animal, compared with control, showed a dilator response on emersion and the indices of participation for the sciatic and carotid vascular beds, at 5 sec after emersion, were 0·01 and 1·85 respectively. Consequently the sciatic bed had returned to control levels of vasoactivity whereas the carotid bed exhibited marked vasodilatation (Table 3). Based on a control stroke volume of 5 ml., the index of contribution for the sciatic bed was negligible whereas that for the carotid vascular bed was 3%. In view of these low indices of contribution during diving and recovery our results can only be used to indicate that two vascular beds within the same animal can behave very differently and not for attempting a complete analysis of the cardiovascular changes during diving.

An attempt at a more dynamic analysis of resistance changes, by plotting pressure against flow, confirmed that there was an increase in resistance to flow in the sciatic vascular bed and that the change in the carotid vascular bed was negligible. The yield pressure increased considerably during a dive in both carotid and sciatic vascular beds. Several factors could have contributed to the raising of the yield pressure in the diving ducks. The increased venous pressure (Folkow *et al.* 1967; Johansen & Aakhus, 1963) and the fall in systemic mean pressure would tend to decrease the pressure difference for flow across the capillary bed. Another factor may be that under intense vasomotor tone the arterioles become unstable at fairly high pressures and exhibit critical closure (Burton, 1951). Changes in inertia of the system may also have some effect but this seems unlikely since although the inertial term must increase during diving, since blood flow stops between cardiac contractions, the change is not marked enough even to affect the phase relationships between the pressure and flow pulse. As at the surface, flow leads pressure during the early period of ventricular systole so that peak flow is reached before peak pressure. However, the technique of utilizing 'run-off' curves is limited by the fact that it neglects the early period of ventricular systole.

The fact that α-blockade abolished the intense vasoconstriction in the sciatic vascular bed showed that this response is mediated by stimulation of adrenergic α-receptors. Furthermore, after α-blockade yield pressure hardly changed during a dive. Therefore these results add confirmation to a suggestion that it is the active vasomotor tone which contributes to the elevation of the yield pressure.

Folkow, Fuxe & Sonnenschein (1966) have described a dense aggregation of adrenergic nerve terminals along the length of the femoral artery in ducks and found that about 20–30% of total vascular resistance was due to constriction of the 'large artery' during a dive. However, this seems unlikely to be involved in our present experiments since much of this corresponding nerve plexus around the sciatic artery would have been

destroyed by application of the flow probe. Furthermore, since the sciatic artery is in series with the resistance at the periphery then the total resistance of the vascular bed will be given by the value of the greatest resistance under consideration and this is unlikely to be the sciatic artery (Vonruden, Blaisdell, Hall & Thomas, 1964; Weale, 1964). It may be that the function of the adrenergic supply to the major arteries is to increase the stiffness of these vessels during a dive so that the oscillatory work load of the left ventricle is decoupled from the greatly increased terminal impedance (Taylor, 1964).

At present we can say nothing about the relative distribution to the various tissues supplied by the sciatic or carotid arteries. Certainly a fair proportion of the blood distributed from the common carotid artery of the duck will supply extracranial tissues. It may be that the increase in resistance exhibited by this vascular bed is due to the contribution of extracranial vessels so that blood supply to the brain will be little affected. In fact Johansen (1964) actually recorded increased flow to the brain and certain muscles supplied by the carotid artery and a similar conclusion for our ducks can be drawn in the light of dives on α-blocked animals, where resistance to flow in the common carotid artery decreased during a dive, indicating dilation in some part of the vascular bed.

For the maintenance of arterial pressure during a dive, cardiac output must be matched to the change in vascular resistance or vice versa. Obviously chemo- and baroreceptors may be involved in the response. The importance of the former has been established by denervation of the carotid body in the duck (Hollenberg & Uvnäs, 1963; Jones & Purves, 1970). The role of the latter can be assessed from the present experiments. For instance, after α-blockade the cardiac chronotropic response varied; in two animals heart rate was high after 60 sec submergence and systemic blood pressure was maintained. In five ducks heart rate fell, as did blood pressure, throughout the dive. However, if these latter animals were kept submerged for periods of 90 sec or longer then the bradycardia broke and blood pressure returned to control levels. Consequently, the difference between these two groups of animals was one of degree rather than response. The response to diving after injection of atropine provides more evidence for the involvement of baroreceptors in the response of the normal animal. Atropine not only affects cardiac chronotropic activity but also abolishes a negative inotropic response mediated via the vagus (Folkow & Yonce, 1967; Ferrante & Opdyke, 1969). Hence in the face of an eightfold increase in vascular resistance, as exhibited by the normal duck during a dive, pressure should increase proportionately. However, mean blood pressure only rises by 50%. Of course, vasomotor tone was probably higher during submersion in atropinized ducks than is indicated

by calculating resistance to flow from the present set of data. Any potential change in resistance may have been modified by a rise in stroke output of the heart due to an increase in left ventricular contractibility during submergence in the absence of parasympathetic innervation to the heart (Ferrante & Opdyke, 1969). This is likely in view of the fact that sciatic stroke flow increased during submersion. It is unlikely, however, that this would account for the whole of the difference in resistance to flow through the sciatic artery between normal and atropinized ducks during submersion. Consequently, there must be an overriding of vasoconstrictor activity which is probably mediated through the agency of arterial baroreceptors.

Upon surfacing, the adjustments made during the period under water were completely reversed in the normal bird on the commencement of breathing movements, but not before. Angell James & Daly (1968) found that vasoconstriction and bradycardia achieved during simulated dives in dogs could be reduced or abolished by inflating the lungs even without any change in P_{a,O_2} or P_{a,CO_2}, and Butler & Jones (1968) showed that post-dive tachycardia was only seen with the onset of ventilation. The same is also true for the hyperaemia and reduced vasomotor tone in the two vascular beds studied. Blood pressure increased and sciatic flow returned to control values within 5 sec of the start of breathing while at the same time carotid artery flow was 2·5 times normal. Since blood pressure rose, the resistance of the sciatic vascular bed was slightly above control whereas carotid vascular resistance was about half control values.

Although β-blockade with Inderal did not have any significant effect on the cardiovascular responses to submersion it did reduce the immediate hyperaemia and abolished the rise in blood pressure which occurred in normal ducks on surfacing. This could indicate that sympathetic β-receptors may be involved in increasing stroke output of the heart upon recovery, as reported by Folkow *et al.* (1967) and/or that β-receptor stimulation at the periphery is involved in the vasodilatation seen upon emergence in the normal animal.

The pronounced cardiovascular adjustments shown by diving ducks certainly conserve oxygen in the sense that P_{a,O_2} falls less rapidly in normal ducks after 20 sec under water than it does in α-blocked and atropinized animals. Although the dissociation curve of duck's blood is moved to the right during submergence due to a rise in acidity (Andersen & Løvø, 1967), which in itself tends to elevate P_{a,O_2}, vasoconstriction may also contribute to the slow reduction of P_{a,O_2} in the normal animal. This is indicated by the fact that after α-blockade, although sciatic blood flow is reduced it is not as low as in normal ducks after 60 sec submersion and

P_{a,O_2} in α-blocked ducks is 10 mm Hg lower than in normal animals at this time. Furthermore, after injection of atropine, sciatic flow is actually above its control value after 60 sec submersion, and P_{a,O_2} is 20 mm Hg lower than in normal animals at this time.

Upon surfacing, P_{a,O_2} returns comparatively slowly to its control value compared with the change in heart rate, which occurs immediately. Even allowing for lags in the P_{a,O_2} recording system, this indicates that it is unlikely that changes in blood gas tensions are responsible for the trachycardia at emersion and lends support to the suggestion that post-dive hyperventilation, through the agency of pulmonary reflexes, causes a reduction in efferent cardiac vagal tone to a sub-normal level (Butler & Jones, 1968).

The authors wish to thank the Science Research Council for financial support.

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