Regional distribution of blood flow during diving in the duck (*Anas platyrhynchos*)

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The regional distribution of blood flow, both before and during forced diving, was studied in the duck using radioactively labelled microspheres. Cardiac output fell from 227 ± 30 to 95 ± 16 mL kg^{-1} min^{-1} after 20–72 s of submergence and to 59 ± 18 mL kg^{-1} min^{-1} after 144–250 s of submergence. Mean arterial blood pressure did not change significantly as total peripheral resistance increased by four times during prolonged diving. Before diving the highest proportion of cardiac output went to the heart (2.6 ± 0.5%, n = 9) and kidneys (2.7 ± 0.5%, n = 9), with the brain receiving less than 1%. The share of cardiac output going to the brain and heart increased spectacularly during prolonged dives to 10.5 ± 3% (n = 5) and 15.9 ± 3.8% (n = 5), respectively, while that to the kidney fell to 0.4 ± 0.26% (n = 3). Since cardiac output declined during diving, tissue blood flow (millilitres per gram per minute) to the heart was unchanged although in the case of the brain it increased 2.35 times after 20–75 s of submergence and 8.5 times after 140–250 s of submergence. Spleen blood flow, the highest of any tissue predive (5.6 ± 1.3 mL g^{-1} min^{-1}, n = 4), was insignificant during diving while adrenal flow increased markedly, in one animal reaching 7.09 mL g^{-1} min^{-1}. The present results amplify general conclusions from previous research on regional distribution of blood flow in diving homeotherms, showing that, although both heart and brain receive a significant increase in the proportionate share of cardiac output during diving only the brain receives a significant increase in tissue blood flow, which increases as submergence is prolonged.

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La distribution régionale du sang chez le canard, avant et après une immersion forcée, a été estimée au moyen de microsphères marquées par radio-activité. Le débit cardiaque passe de 227 ± 30 à 95 ± 16 mL kg^{-1} min^{-1} après une immersion de 20–72 s et baisse jusqu'à 59 ± 18 mL kg^{-1} min^{-1} après une immersion de 144–250 s. La pression artérielle moyenne ne subit pas de changement significatif puisque la résistance périphérique totale augmente d'un facteur de quatre durant une immersion prolongée. Avant la plongée, le débit cardiaque fournit surtout du sang au cœur (2.6 ± 0.5%, n = 9) et aux reins (2.7 ± 0.5%, n = 9) alors que le cerveau reçoit moins de 1% du sang. Au cours d'une immersion prolongée, les proportions de sang reçues augmentent considérablement jusqu'à 10.5 ± 3% (n = 5) dans le cas du cerveau et jusqu'à 15.9 ± 3.8% (n = 5) dans le cas du cœur, alors que le rein ne reçoit plus que 0.4 ± 0.26% (n = 3) du débit cardiaque. Comme le débit cardiaque diminue au cours de la plongée, l'irrigation sanguine des tissus (millilitres par gramme par minute) du cœur ne change pas, mais celle des tissus du cerveau augmente d'un facteur de 2.35 après une plongée de 20–75 s, et d'un facteur de 8.5 après une immersion de 140–250 s. L'irrigation de la rate, la plus élevée avant la plongée (5.6 ± 1.3 mL g^{-1} min^{-1}, n = 4) devient à peu près nulle durant la plongée, alors que l'irrigation des surrénales augmente considérablement, ayant atteint 7.09 mL g^{-1} min^{-1} chez un individu. Ces résultats viennent corroborer les conclusions générales déduites d'études antérieures sur la distribution régionale du sang chez les homéothermes qui plongent: bien que le cœur et le cerveau reçoivent une plus grande proportion du débit cardiaque durant la plongée, seuls les tissus du cerveau reçoivent une plus grande irrigation et cette augmentation devient plus importante à mesure qu'augmente la durée de l'immersion.

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Introduction

Since cardiac output falls dramatically in diving birds and mammals during forced submergence it is obvious that peripheral circulation must be curtailed in order to maintain arterial blood pressure (see Elsner (1969) and Jones and Johansen (1972) for reviews). The nature of blood redistribution during diving has been investigated using both direct and indirect techniques and these studies have resulted in the generalization that systemic perfu-

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sion is largely restricted to the heart and brain during submergence (Irving 1934; Johansen 1964; Andersen 1966; Elsner *et al.* 1966; Blix *et al.* 1976). Unfortunately, due to the nature of the techniques used, only studies by Johansen (1964) and Blix *et al.* (1976) have been able to provide a picture of the integrated cardiovascular response to forced submergence and their data indicate marked differences between the nature of blood flow redistribution in ducks and seals. Using radioactively labelled ^{86}Rb, Johansen (1964) concluded that, in ducks, there was a three- to five-fold increase in blood flow to tissues like the ventricular myocardium and central nervous tissue. On the other hand, Blix *et al.* (1976), using radioactively labelled microspheres, found that in seals although the proportionate share of cardiac output going to the cerebrum increased four times during diving the absolute tissue blood flow to both heart and brain fell dramatically. In fact, ventricular tissue blood flow was reduced in proportion to the reduction in heart rate (Blix *et al.* 1976). The difference in the cardiovascular response of ducks and seals to forced submergence is intriguing but, even aside from a genuine species difference, differences in technology and experimental protocol may have contributed to the apparent divergence between the species. For instance, only a rough estimate of cerebral perfusion can be obtained using ^{86}Rb since it does not cross the blood–brain barrier (Johansen 1964). Also, in the study of Blix *et al.* (1976), useful results were only obtained from one animal and the spheres were introduced into the circulation after 5 min of submergence, about one-quarter of the animal's known tolerance to submergence, so that part of the species difference could be merely a reflection of different tolerances to prolonged apnoea.

In the present series of experiments we have used the microsphere method to determine blood flow to a wide range of tissues in the duck (*Anas platyrhynchos*) before and during forced submergence. We studied blood redistribution during short and long dives to establish not only the blood perfusion pattern in diving but also whether there are any marked changes in the nature of regional flow redistribution as diving is prolonged. The results show that although both heart and brain receive an increase in the proportionate share of cardiac output during diving, only the brain receives a significant increase in tissue blood flow which increases as submergence is prolonged.

Methods

Data have been obtained from 14 mallard and White Pekin ducks (*Anas platyrhynchos var*) of both sexes varying in mass from 1 to 3.2 kg (mean mass = 1.5 kg). The animals were held in the Vivarium of the University of British Columbia and all experiments were performed at the temperature at which ducks were held (20–22°C). All data obtained were analysed statistically and in the text and tables mean values are presented ± SEM. Mean values of a particular variable were compared by a *t* test and 5% was considered the fiducial limit of significance. The adjectives 'initial' and 'control' refer to observations made before a dive (submergence of the head).

All operative techniques were of a superficial nature and were done under local anaesthesia. A sciatic artery was exposed and cannulated with a polyethylene cannula. The cannula was connected to a T-piece, one arm leading to a Bio-Tec BT70 pressure transducer and the other to a Harvard infusion–withdrawal syringe pump. In some experiments a cuvette, containing an oxygen electrode surrounded by a water jacket at 41°C (the duck's body temperature), was connected between the T-piece and the syringe pump. In these experiments blood was withdrawn from the sciatic artery continuously before and during the dive. The oxygen electrode was calibrated, in the cuvette, with humidified air and N_2 gas before a dive and, in the course of a dive, blood samples were taken and analysed on an IL micro 13 pH – blood gas analyser with an IL 326 temperature controller set to 41°C, to check that the predive calibration of the oxygen electrode was unchanged. These blood samples were also analysed for Pa_{CO_2} and pH*a*. Microspheres were injected into the left ventricle or pulmonary vein. For injecting spheres into the left ventricle a polyethylene cannula filled with heparinized avian saline (40 IU mL^{-1}) and connected to a pressure transducer was inserted into the right brachial artery and advanced until its tip lay in the left ventricle. A pressure trace was used as positional guide and a large increase in the pulse pressure signified the desired tip location. For injecting spheres into the pulmonary vein the left pulmonary vein was exposed by a small incision between two anterior ribs. A catheter was inserted, after first piercing the vein with a needle, and its tip advanced to the region of the left atrium. The catheter was held in the vein by tissue cement and was further secured by ligatures to the body wall. The volume of the cannula or catheter was less than 0.1 mL. The electrocardiogram (EKG) was monitored bipolarly using thin insulated wires, with their bare tips located under the skin on the sternum and thigh. All signals were amplified conventionally and displayed on a Watanabe pen recorder, writing on rectilinear coordinates.

Regional distribution of blood flow and cardiac output were assessed using ^{85}Sr and ^{141}Ce radioactively labelled microspheres of 15 ± 5 μm diameter (3M Company, Nuclear Products Div., St. Paul, Minnesota). The spheres were supplied in vials with 10 mL of isotonic saline with Tween 80 and each vial contained 1 mCi (1 Ci = 37 GBq) of isotope. A number of tests were conducted on the microspheres to establish radionuclide purity, size uniformity, and surface characteristics (Heymann *et al.* 1977). Radionuclide purity was determined by spectrography of a subsample of both types of spheres. The subsamples were taken directly from the manufacturer's vials and diluted in distilled water to give a count rate within the counting equipment's range of maximum efficiency. Spectral analysis revealed the ^{85}Sr photopeak at 490 keV, and the ^{141}Ce photopeak at 140 keV. No other extraneous peaks were present that would have indicated contamination by other isotopes. After allowing the spheres to settle out of suspension, a sample of the isotonic saline suspending agent taken directly from the manufacturer's vials showed no appreciable amounts of leached activity.

Sphere concentration and size range were checked by taking subsamples from the manufacturer's vials. A drop of this subsample was placed on the counting grid of a hemocytometer. Results from a number of observations indicated no clumps, aggregations, or irregularly shaped spheres and that the dimen-

sions were within the manufacturer's specifications. Scanning electron micrographs indicated the sphere's surface to be very smooth and a very uniform size distribution was seen in a random sample.

Subsamples were taken from the manufacturer's vials and diluted with 6% (w/v) Dextran 70 to give $10^5 \pm 10^4$ ^{85}Sr-labelled spheres per millilitre and $5 \times 10^4 \pm 5 \times 10^3$ ^{141}Ce-labelled spheres per millilitre for injection. The required sample volume was drawn into a 1.0-mL syringe and transferred to a 30-mL sterile vial containing a magnetic stirring bar and the appropriate quantity of diluent. Before injection into an animal these solutions were agitated on a vortex mixer and then ultrasonified for 1–2 min to disperse the spheres. The injection solution vials were placed on a magnetic stirrer and stirred continuously. A 1-mL sample was withdrawn into a disposable syringe which was then attached to the side arm of a plastic stopcock fixed to a mechanical vibrator. The other two arms of the stopcock were connected to a 10-mL syringe containing warm saline and to the cardiovascular cannula. The entire injection assembly was vibrated before and during sphere injection to prevent settling. By preparing a series of duplicate samples we estimated an accuracy of ±5% in the volume withdrawn for injection. After each experiment, a 0.1-mL sample was prepared, in duplicate, in an identical manner to the above except that the sample was taken into a calibrated micropipette. The sample and micropipette were placed into a counting tube containing distilled water so that it approximated the tissue sample counting geometry (see later). The activity registered from these samples, after multiplying by 10, served as a measure of the maximum possible injected activity. In order to determine the total number of counts injected the entire injection assembly (i.e. 1.0-mL syringe, stopcock, and injection cannula) was cut up and placed in counting tubes containing distilled water. The counts remaining in the injection assembly were totalled and subtracted from the measure of maximum possible injected activity to give the total number of counts injected.

The ducks were restrained, ventral side down, on an operating table with the head held pointing downwards into a large funnel. Submergence was effected by filling the funnel from a beaker of room temperature water after first clamping a piece of tubing attached to the spout. Removal of the clamp drained the funnel for emersion. In the majority of experiments 1 mL of ^{85}Sr-labelled microspheres was injected before submergence and 1 mL of ^{141}Ce-labelled microspheres injected during the dive. However, this order was reversed on two occasions when 2 mL of ^{141}Ce-labelled spheres was injected predive. In three experiments both groups of spheres were injected during the dive, ^{85}Sr-labelled spheres early and ^{141}Ce-labelled spheres late. The injection period was from 20 to 30 s and was followed by a wash of 2 mL of warm saline over a period of 40 s. Continuous withdrawal of blood from the sciatic artery, at a rate of 2 mL min^{-1}, started 10 s before sphere injection and continued for at least 1 min after injection predive and 1.5 min after during submergence. The period of withdrawal was accurately noted and the volume withdrawn calculated. The reproducibility and accuracy of the volume withdrawn with respect to calculated volumes, for a given time, was tested frequently by weighing the syringe before and after withdrawal periods. In those experiments in which Pa_{O_2} was monitored continuously, blood withdrawal started 1 min before the dive and continued throughout the dive. Arterial blood gas tensions were also monitored in blood samples taken from the sciatic artery at the end of each period of continuous blood withdrawal. Some of these blood samples were saved and monitored for radioactivity to check that the withdrawal periods were long enough for all spheres to be lodged in the tissues. No radioactivity was found in any of these samples. Blood samples were also withdrawn from the ventricle or pulmonary vein to check that no spheres were remaining uncirculated, near the injection sites. No radioactivity was detected in these samples either. At the end of the dive the funnel was drained and the animal's head was raised. Some 10–20 min after emergence the animal was killed by an overdose of Nembutal (sodium pentobarbital), injected in the pulmonary vein or left ventricle.

One day after death the carcass was autopsied and tissue samples were prepared. Each tissue sample was placed in a numbered, preweighed counting tube, and weighed again. Pellets of KOH or 10 *M* KOH solution were added to each tube until the fluid level reached a standard height. The tubes were then placed in a water bath overnight to enhance digestion of the solid samples. Immediately before counting the sample tubes were centrifuged at 1000 rpm for 20 min to concentrate the spheres and suspended solids at the bottom of the tube. Consequently, sample counting geometry was standardized over a wide range of sample weights and volumes.

All samples were counted in a Nuclear Chicago automatic gamma counter with a 7.5-cm (approx.) true well, lithium activated, sodium iodide crystal. ^{141}Ce and ^{85}Sr microsphere radiation was counted simultaneously using a dual pulse height analyser with windows spanning 85% of the total photopeaks for each isotope. The spillover from the high energy window (^{85}Sr) raised the count rate in the low energy window (^{141}Ce). One-third of the ^{85}Sr counts appeared in the ^{141}Ce window so, when counting mixed samples, the actual counts in the ^{141}Ce window were corrected for this artifact. All samples were counted for at least 20 min or until 1.0×10^6 counts had been accumulated. Background counts were automatically subtracted from each count.

The following equations were used in calculating cardiac output and tissue blood flow (Wagner *et al.* 1969; Archie *et al.* 1973; Bartrum *et al.* 1974).

$$[1] \quad \text{Fraction of injected microspheres in sample} = \frac{\text{radioactivity of sample}}{\text{total radioactivity injected}}.$$

$$[2] \quad \text{Cardiac output (mL min}^{-1}) = \frac{\text{rate of withdrawal of reference blood sample (mL min}^{-1})}{\text{fraction of injected microspheres in reference blood samples}}.$$

$$[3] \quad \text{Tissue blood flow (mL min}^{-1}) = \text{cardiac output} \times \text{fraction of injected microspheres in the tissue.}$$

The cardiac outputs were normalized to body weight and tissue blood flow to gram weight of that tissue. Blood flow to the right and left kidneys was compared with test for evenness of microsphere mixing and samples containing less than 400 microspheres were not included in any analysis (Buckberg *et al.* 1971). The number of counts per microsphere was determined as described by Heymann *et al.* (1977) and a half-life plot was made for each batch of spheres, so that the number of spheres per tissue was known from the number of counts per tissue in every experiment.

Results

The lungs were removed from three animals and their radioactivity measured to indicate whether or not spheres were trapped in the peripheral circula-

TABLE 1. Cardiovascular variables measured before and during diving. All values are given as means ± SEM and the numbers in parentheses refer to number of animals on which observations were made

		Dive	
	Predive	20–72 s	144–250 s
Heart rate, beats min^{-1}	205 ± 11(10)	46 ± 7(9)	44 ± 12(5)
Cardiac output, mL kg^{-1} min^{-1}	227 ± 30(10)	95 ± 16(6)	59 ± 18(5)
Stroke volume, mL kg^{-1}	1.00 ± 0.14(10)	2.54 ± 0.58(6)	1.65 ± 0.56(5)
Mean arterial blood pressure, mmHg	156 ± 9(10)	156 ± 8.5(8)	142 ± 27(5)
Left ventricular power, W	$7.35 \times 10^{-2} \pm 1.4 \times 10^{-2}$(7)	$3.1 \times 10^{-2} \pm 0.4 \times 10^{-2}$(5)	$2.05 \times 10^{-2} \pm 0.7 \times 10^{-2}$(5)
Total peripheral resistance, dynes s cm^{-5}	$0.67 \times 10^5 \pm 0.09 \times 10^5$(7)	$1.51 \times 10^5 \pm 0.34 \times 10^5$(5)	$2.65 \times 10^5 \pm 0.75 \times 10^5$(5)
Pa_{O_2}, mmHg	90 ± 3(12)	45 ± 2(3)	32 ± 4.5(4)
Pa_{CO_2}, mmHg	30 ± 0.7(12)	44 ± 2(3)	53 ± 5(4)
pH*a*	7.48 ± 0.01(10)	7.40 ± 0.03(3)	7.26 ± 0.03(3)

tion. Predive the lungs received 0.6% of the cardiac output while during the dive this rose to 0.7%. We concluded that this amount of blood could be supplied by the bronchial circulation and that all spheres were removed in one circulation. It has previously been shown that avian lungs extract this size of microspheres from the circulation (Boelkins *et al.* 1973). Evenness of mixing and distribution of microspheres was confirmed by a plot of the blood flow to the right and left kidneys, before and during diving. The regression equation for this relation was $y = 1.01x + 0.04$, with a correlation coefficient(r) of 0.94.

During diving the spheres were injected either during the period from 20 to 72 or from 144 to 250 s. The longest dive in this series of experiments was 420 s. No significant changes were observed in blood pressure or heart rate during microsphere injection or cardiac output determination although in about one-third of the experiments heart rate increased slightly during the saline wash in a dive (Fig. 1). Injection of three times as many spheres as usual had no long-term effect on blood pressure or heart rate. Some 10–20 min after the dive, both heart rate and blood pressure returned to control values showing that there were no adverse effects of sphere injection in the dive.

The cardiovascular responses to submergence are shown in Table 1. Heart rate fell from 205 ± 11 to 46 ± 7 beats min^{-1} within the period of submergence from 20 to 72 s and this rate was maintained throughout the remainder of the dive. Total peripheral resistance rose 2.25 times, at 20–72 s of the dive, and continued to increase to 4 times control towards the end of the dives (144–250 s). Pa_{O_2} fell rapidly in the early period of dive but later the rate of fall declined markedly (Fig. 1) so that even at the end of the longest dives Pa_{O_2} was usually above 30 mmHg. Stroke volume increased 2.5 times over the control stroke volume in the early part of the dive but was not significantly different from the control value in the diving period from 144 to 250 s. Left ventricular power output fell progressively during the period of submergence, being 44% of the initial value during the dive period from 20 to 72 s and 28% of the initial value at 144–250 s. Mean arterial blood pressure did not change significantly during diving (Fig. 1).

In the majority of cases tissue samples were taken from the larger organs and only the brain, eyes, spleen, kidney, and heart were removed, weighed, and counted in their entirety. Of these tissues, the highest proportion of predive cardiac output went to the heart and kidneys, being 2.6 ± 0.5% (n = 9) and 2.7 ± 0.5% (n = 9), respectively. The brain and the eyes both received less than 1% of the cardiac output, being 0.83 ± 0.3 (n = 7) and 0.51 ± 0.1 (n = 11), respectively. During a dive the share of cardiac output directed to the central nervous structures and heart increased spectacularly. In the dive period from 20 to 72 s the brain's share of cardiac output rose 5 times, the eye's 2–3 times, and the heart's 2.5 times. All of these organs increased their share of cardiac output as the dive was prolonged. During the dive period from 144 to 250 s the brain received 10.5 ± 3% (n = 5) of cardiac output, the eyes 2.1 ± 0.5% (n = 5), and the heart 15.9 ± 3.8% (n = 5). The proportion of cardiac output going to the kidneys and spleen fell markedly. During the dive period from 20 to 72 s the kidney received 0.72 ± 0.25% (n = 4) and during the dive period from 144 to 250 s it received only 0.41 ± 0.26% (n = 3) of the cardiac output.

Since cardiac output fell dramatically during diving (Table 1) an increase in the proportionate share of cardiac output does not necessarily mean that that tissue has received any increase in actual blood flow. For instance, although the propor-

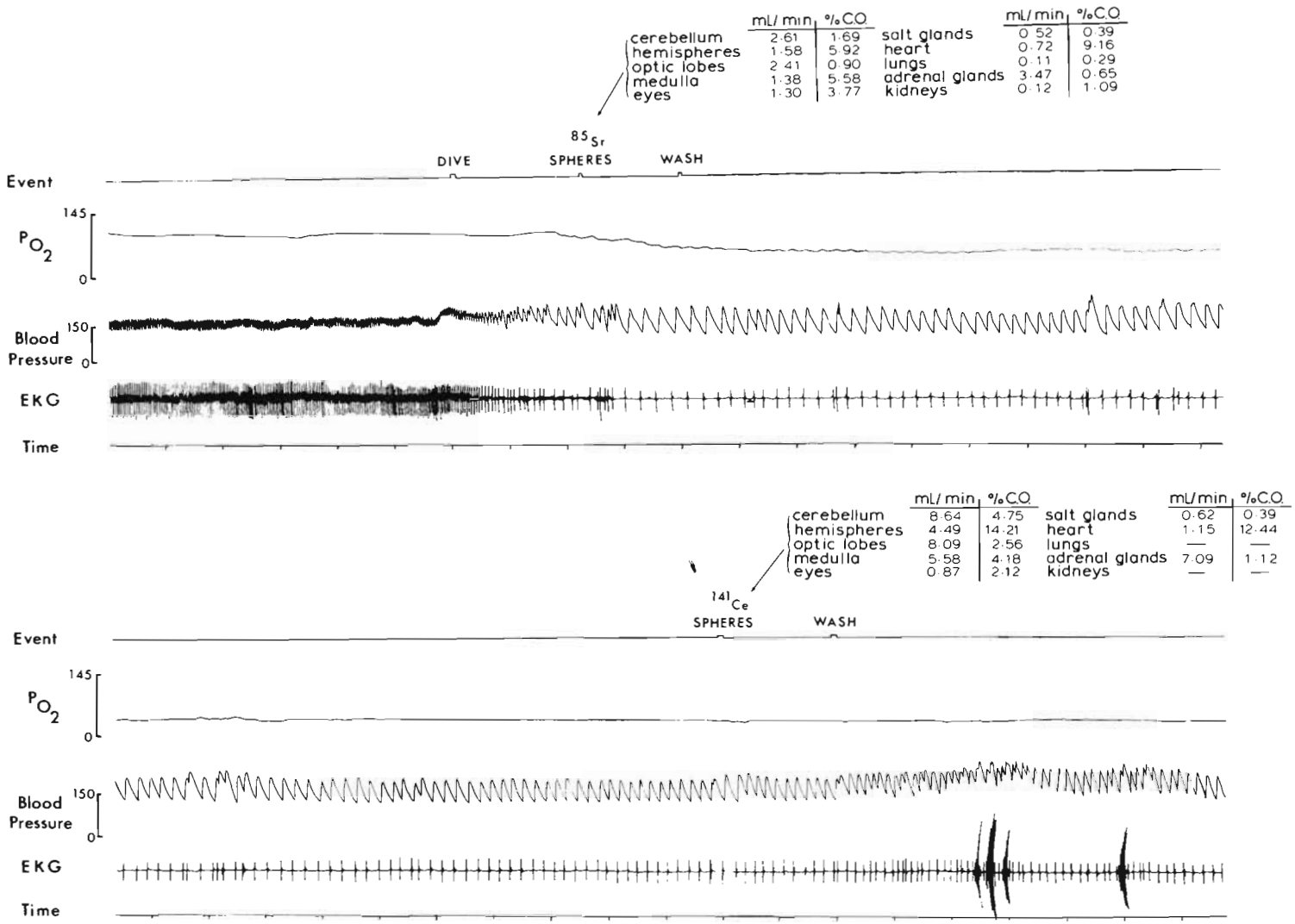

FIG. 1. Tracing of a continuous recording during a prolonged dive in a duck. In each group of four traces the upper trace is an event marker indicating diving and microsphere injection; next is Pa_{o_2}(mmHg) recorded continuously; next is arterial blood pressure (mmHg); next is EKG; the bottom trace is a time marker indicating 10-s intervals. In this experiment microspheres were injected after 25 (^{85}Sr) and 240 (^{141}Ce) s of submergence and the dive was terminated after 372 s. The percentage distribution of cardiac output and blood flow per gram wet tissue after 25 and 240 s of submergence are given in the accompanying tables.

TABLE 2. Blood flow per gram of wet tissue in ducks before and during submergence. All values are given as means ± SEM and the numbers in parentheses refer to the number of ducks from which tissue samples were taken

Organ	Absolute blood flows, mL g^{-1} min^{-1}		
	Predive	20–72 s dive	144–250 s dive
Brain	0.43±0.10(8)	1.01±0.24(7)	3.68±1.86(4)
Eyes	0.59±0.06(7)	0.66±0.23(4)	0.63±0.14(5)
Liver	0.58±0.15(6)	—[a]	—
Lung	0.19±0.08(3)	0.06±0.03(4)	—
Spleen	5.56±1.29(4)	—	—
Small intestine	0.48±0.16(5)	—	—
Kidneys	1.08±0.33(8)	0.07±0.04(6)	—
Heart	0.73±0.23(8)	0.63±0.19(5)	0.88±0.28(5)
Muscle	0.32±0.11(5)	—	—

[a]—, no accurately measurable flow.

tionate share of cardiac output to the heart rose six times, by the dive period from 144 to 250 s, tissue blood flow only increased insignificantly from 0.73 ± 0.23 mL g^{-1} min^{-1} (n = 8) initially to 0.88 ± 0.28 mL g^{-1} min^{-1} (n = 5) late in the dive (Table 2). However, in the case of the brain, tissue blood flow increased throughout the dive: 2.35 times after 20–75 s and 8.5 times after 144–250 s of submergence. The proportionate share of cardiac output to the kidney fell to ¼ early in the dive while, at the same time, tissue blood flow fell to $\frac{1}{15}$th. Blood flow to the spleen, which was the highest of any tissue before the dive, was insignificant during diving, while flow to the adrenal glands increased during diving and, in the animal shown in Fig. 1, rose from 3.47 mL g^{-1} min^{-1} in the early part of the dive to 7.09 mL g^{-1} min^{-1} after 240 s of submergence. Tissue blood flow to the eyes and salt glands was unaffected by diving while flow to the muscles in the head, neck, breast, and hindlimb, the pancreas, the liver, and small and large intestine was reduced but we were unable to gauge the degree of reduction since, during dives, less than 400 spheres were deposited in any tissue sample.

Discussion

The general conclusions from previous research on regional distribution of blood flow in diving homeotherms have been amplified by the present research since it allows assessment of the integrated cardiovascular response to forcible submergence. During diving blood flow to virtually all vascular regions was reduced except that to the adrenals, brain, heart, and eyes. The brain is about 0.35% and the heart 0.9% of the body mass of the ducks used in these experiments (measurements on five ducks with mean mass of 1.53 kg). Consequently, as was shown or may be calculated from data given in Tables 1 and 2, in prolonged dives the heart and brain, which constitute only 1.25% of body mass, receive 25–35% of the cardiac output. This marked redistribution of blood flow meant that it was impossible to quantitate, with any precision, flow to those tissues showing a large reduction in a dive. We sampled a large number of extracranial tissues, however, and, based on our observations, would suggest that blood flow regulation is achieved at the tissue and not at the segmental level as was suggested by Johansen (1964). In 14 of 18 samples of extracranial tissues such as neck and facial muscle, tongue, oesophagus, and skin we found marked reductions in flow during diving, while the eyes received the same flow and the brain received a marked increase in flow.

Cerebral blood flow increased steadily during diving and after 144+ s of submergence was 8.5 times predive values. An increase of this order of magnitude has never been reported in other divers although differences in dive times and in measurement techniques may have obscured this result in previous work. Cerebral blood flow in dives has previously been estimated by the microsphere technique, by reading cerebral angiograms, or by monitoring flow in one of the major arteries supplying the brain. These estimates of cerebral flow have ranged from a slight increase (1.5 to 2.5 times) (Dormer *et al.* 1977), through little or no change in flow (Bron *et al.* 1966; Butler and Jones 1971; Kerem *et al.* 1975; Lin and Baker 1975), to marked reductions in flow (Blix *et al.* 1976).

In ducks cerebral blood flow is greatly affected by increases in Pa_{CO_2} or decreases in Pa_{O_2} (Grubb *et al.* 1977; Grubb *et al.* 1978). At the level of Pa_{CO_2} encountered in prolonged dives brain blood flow

would be little more than doubled from normocapnic flow levels while the decline in Pa_{O_2} alone would increase cerebral perfusion some six times (Grubb *et al.* 1977; Grubb *et al.* 1978). Obviously the effects of Pa_{O_2} and Pa_{CO_2} in promoting increased cerebral perfusion must sum to produce the observed increase in cerebral blood flow. The fall in pH during a dive may be a further factor increasing brain blood flow. Since flow to white matter increases much more than flow to grey matter (Grubb *et al.* 1977; Grubb *et al.* 1978), our estimate of cerebral flow, which includes both white and grey matter, may underestimate any role of synergism and other factors in promoting cerebral perfusion.

The majority of cardiac blood flow occurs during diastole so an increase in cardiac flow might be expected in diving since diastole is prolonged while arterial blood pressure is maintained. Further, cardiac flow is indirectly proportional to Pa_{O_2} and, since Pa_{O_2} falls markedly during diving, cardiac blood flow should increase. On the other hand, cardiac blood flow is directly proportional to myocardial work which falls during diving (Kirk *et al.* 1975; Gibbs 1978) and, in the present dives, the increase in stroke volume which accompanied bradycardia will tend to increase the efficiency of cardiac contraction, further reducing myocardial energy demand and cardiac flow (Sarnoff, Braunwald *et al.* 1958; Sarnoff, Case *et al.* 1958). The fall in myocardial energy demand will tend to offset any tendency for cardiac flow to increase due to hypoxic or haemodynamic factors. In our experiments cardiac flow was little changed from resting levels during diving although, due to the reduction in cardiac output, the proportionate share of cardiac output going to the heart increased markedly. This, in fact, must also have been the case with Johansen's (1964) ducks although the differentiation between tissue blood flow and proportionate share was not clearly made. This is very different from the situation which exists in the seal. Although flow to the left ventricular myocardium in the seal, after 5 min of diving, is $\frac{1}{10}$th of the predive flow, the proportion of cardiac output it receives is similar to what it was before diving (Blix *et al.* 1976). However, after a 5-min dive in a seal the Pa_{O_2} is 47 mmHg which means that the blood could be 80% saturated with oxygen (Lenfant 1969) so the main determinant of the decreased cardiac flow, at this time, would be myocardial work. Ducks do not exhibit this prodigious decrease in cardiac work. Based on the data of Blix *et al.* (1976) it seems unlikely, even in a prolonged dive, that cardiac flow will ever exceed predive levels in the seal.

The renal vascular bed was shut down in the duck during diving confirming other results obtained by both direct and indirect methods (Murdaugh *et al.* 1961; Johansen 1964; Sykes 1966; Elsner 1969; Anschuetz *et al.* 1971; Lin and Baker 1975; Blix *et al.* 1976). On the other hand adrenal blood flow increased progressively throughout the dive in our ducks as it apparently did in those studied by Johansen (1964). Besides generalized psychic or physical stress (Usdin *et al.* 1976) many factors which affect ducks during diving such as nasal stimulation (Allison and Powis 1971), hypoxia (Cannon and Hoskins 1911; Ludemann *et al.* 1955; Lammerant and Herdt 1965), hypercapnia (Tenny 1956), acidemia (Nahas *et al.* 1960), and imminent circulatory collapse (Watts and Bragg 1958; Nakano *et al.* 1961) are known to raise plasma catecholamine levels in mammals. In view of the large increase in adrenal blood flow during prolonged diving we would suggest that, in these conditions, circulating catecholamines make an important contribution to the cardiovascular adjustments.

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