

CARDIOVASCULAR AND RESPIRATORY RESPONSES OF DUCKS TO PROGRESSIVE HYPOCAPNIC HYPOXIA

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INTRODUCTION

A fall in oxygen tension of the inspired air demands that both circulatory and respiratory adjustments be made if the tissues are to receive an adequate supply of oxygen. Increase in ventilation will reduce the oxygen tension difference between the air breathed and the arterial blood, but even so when the oxygen tension of the arterial blood (P_{a,O_2}) falls a decrease in oxygen supply to the tissues will ensue unless there are adjustments in the circulatory compartment. These do not necessarily entail an increase in cardiac output; for when exposed to hypocapnic hypoxia (altitude) a pronounced Bohr shift coupled with increased oxygen extraction by the tissues, reflected in a lowering of P_{v,O_2} , could go some way towards achieving the same end.

Ducks certainly show large increases in respiratory minute volume in response to hypoxia (Jones & Purves, 1970) but there is no information concerning any changes in cardiac output or oxygen extraction by the tissues. Butler (1970) reports a significant tachycardia in ducks only when P_{a,O_2} falls below 35 mmHg, which implies an increase in cardiac output at this level of hypoxia. Since the blood of many flying birds shows a low affinity for oxygen and a pronounced Bohr shift (Lenfant, Kooyman, Elsner & Drabek, 1969), during exposure to high altitude marked changes in cardiac output may not become necessary for maintenance of oxygen supply to the tissues until P_{a,O_2} is very low.

The present paper concerns itself with measuring the respiratory and cardiac responses of resting unanaesthetized ducks to simulated high altitudes, and includes measurements of gaseous exchange at both tissues and lungs under these conditions.

METHODS

The experiments were performed on three Muscovy ducks (*Cairina moschata*) and ten White Pekin ducks (*Anas* sp.) varying in age from 10 weeks to 6 months and in weight from 2.2 to 2.6 kg. The ducks were lightly restrained in an approximately natural resting position. An endotracheal cannula made of soft vinyl tubing of the appropriate size, 4-6 mm outside diameter and 1 mm wall thickness, was inserted into the glottis and advanced some 5-6 cm into the trachea. All ducks tolerated this procedure. The tracheal tube was then attached to a Hewlett Packard 1530-1042 small-animal pneumotachograph, air flow was measured with a Hewlett-Packard 270 differential pressure transducer, and the flow signal was integrated to give tidal volume

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(\bar{V}_T). A side arm of P.E. 100 polyethylene tubing (Clay Adams, Parsippany, N.J. U.S.A.) was used to withdraw air samples from the endotracheal cannula. The oxygen tension (P_{O_2}) and carbon dioxide tension (P_{CO_2}) of these samples were measured at 40 °C with appropriate Radiometer electrodes which were frequently calibrated using oxygen/nitrogen mixtures and air/carbon dioxide mixtures.

Cardiac output (\dot{Q}_b) was measured using the dye-dilution technique. The sciatic artery was exposed under local anaesthesia and cannulated with either P.E. 60 or P.E. 100 polyethylene tubing. The cannula was passed up the artery into the arch of the aorta, a distance of some 15–20 cm, the incision was closed around the catheter and the area was periodically infiltrated with local anaesthetic. A cannula of P.E. 160 polyethylene tubing was also inserted into the brachial vein and advanced into the right atrium. In order to prevent blood clotting the cannulae were filled with saline containing 10 i.u. of heparin/ml. 0.25 mg of iodicyanine green ('Cardiogreen', Hynson, Westcott and Dunning Inc., Baltimore, Md., U.S.A.) in 100 μ l of aqueous solvent was injected into the right atrium by chasing it with a 'slug' of 2–3 ml of avian saline. Blood was withdrawn from the sciatic artery at a constant rate of between 20 and 40 ml/min, passing through the sensor of a G.M.E. Model DT dye tracer (Gilson Medical Electronics, Middleton, Wisconsin, U.S.A.) and the dye curve was displayed on the servo channel of a G.M.E. Model M5P mini-polygraph. The response time of the pen on the Gilson polygraph over the range used in the experiments was at least twice as fast as the maximum response recorded in any cardiac output determination. The blood of one of us (D. R. J.) was used to make a series of standards of iodicyanine green in order to test the linearity of the Gilson dye tracer. Over the range of pen deflexions encountered in the experiments the response was linear with a maximum error on each point of $\pm 3\%$. At the conclusion of each experiment the dye tracer was calibrated, under the experimental conditions, using two standards of iodicyanine green in the duck's blood.

In order to change the composition of the inspired gas mixture a T-piece was attached to the pneumotachograph and compressed air was passed through its cross-arm at a flow rate of 2 l/min. When respiration was stable, as judged from the record of tidal volume, a series of different oxygen/nitrogen mixtures was substituted to give a series of mixtures of progressive hypoxic conditions. After each gas change approximately 15 min was allowed for respiratory and circulatory adjustments to stabilize at a new level; tidal volume and respiratory frequency were then recorded for 1 min and samples of gas were taken for measurements of tensions of oxygen and carbon dioxide in inspired and expired air (P_{I,CO_2} , P_{I,O_2} , P_{E,CO_2} , P_{E,O_2}). Samples of arterial blood and of mixed venous blood were taken, and the oxygen tensions (P_{a,O_2} and $P_{\bar{v},O_2}$) and carbon dioxide tensions (P_{a,CO_2} and $P_{\bar{v},CO_2}$) were determined with appropriate Radiometer electrodes at 40 °C. Arterial pH and venous pH were measured with a Radiometer capillary glass electrode which was calibrated with Radiometer precision buffer solutions (Type S1510 and Type S1500). \dot{Q}_b was determined immediately before or after the blood samples had been taken. No attempt was made to control arterial pH or arterial P_{CO_2} . Blood removed for gas-tension measurements or in determination of \dot{Q}_b was returned to the animal by infusing at a constant slow rate. At least two determinations of \dot{Q}_b and other analyses were made at each level of hypoxia to ensure that a new steady state had been established following each gas change.

The dye curves were extrapolated, after plotting on semilogarithmic paper, to within 1% of the original base line. The area under the curves was determined with a planimeter or by tracing the curve onto tracing paper and cutting out the area under the curve and weighing it, the weight being compared with the weight of an area representing a known dye concentration for the time period of the extrapolated dye curve. In the calculation of central blood volume (cardiac output \times mean transit time) mean transit time was calculated by the equation of Hamilton, Moore, Kinsman & Spurling (1932) and Hamilton (1953). No correction was made for the error caused by the transit time along the arterial cannula. Oxygen consumption (\dot{V}_{O_2}) and carbon dioxide production (\dot{V}_{CO_2}) were assessed by the Fick principle from measurements of P_{I,O_2} and P_{I,CO_2} , P_{E,CO_2} , and P_{E,O_2} , and volume of inspired air (\dot{V}_I). Piiper, Drees and Scheid (1970) have shown that in the fowl P_{E,CO_2} rises rapidly during the first few millilitres of expiration and then reaches a plateau lasting for the major part of expiration. Gas samples were never taken in the early period of expiration; sampling was confined to the last two-thirds of the act. Consequently the calculated \dot{V}_{O_2} will be overestimated, at maximum by the ratio of dead space/tidal volume.

All results were analysed statistically and 5% was considered as the fiducial limit of significance. In the text and figures all measured and some calculated parameters are given \pm s.e. of the mean, but some values were derived from the means of the measured parameters, e.g. \dot{V}_{O_2} , \dot{V}_{CO_2} and R.Q. and these are given alone.

RESULTS

(a) Cardiovascular and respiratory parameters in two species of duck at rest

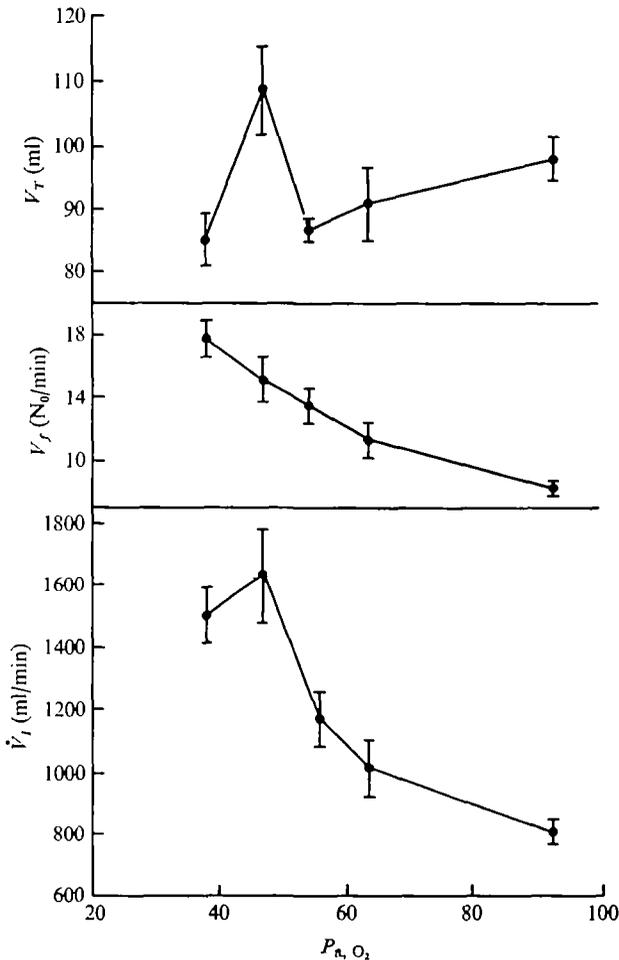
The resting heart rate of Muscovy ducks was significantly lower than that of White Pekin ducks but due to the significantly larger stroke volume of the former the total cardiac outputs were in the same range (Table 1). \dot{Q}_b of the present animals, expressed on a per kilogram basis (approx. 400 ml/min/kg), falls between the two values previously reported for ducks (Sturkie, 1966, 260 ml/min/kg, and Folkow, Nilsson & Yonce, 1967, 560 ml/min/kg). There was a marked difference in mean transit time and central blood volume in the two species.

As with the cardiac parameters significant differences existed between some of the respiratory parameters. As the breathing frequency (f) of White Pekin ducks was significantly lower than that of Muscovy ducks and V_T was significantly larger, there was no significant difference in \dot{V}_I (Table 1). No other measured parameters were significantly different except P_{v,O_2} which was lower in Muscovy than White Pekin ducks. The results gave derived values of \dot{V}_{O_2} of 50 ml/min for Muscovy ducks and 51.9 ml/min for White Pekin ducks which when expressed on a per kilogram basis are almost identical to those recorded by Butler (1970) for Mallard ducks (*Anas platyrhynchos*). The values for \dot{V}_{CO_2} are subject to a small error due to the fact that no correction was made for any difference between inspired and expired volumes. Nevertheless in both species of ducks \dot{V}_{CO_2} was well below \dot{V}_{O_2} , with R.Q. being in the range of 0.7.

Table 1. Comparison between two species of duck at rest

Species	Weight (kg)	\dot{Q}_b (ml/min)	Heart rate (beats/min)	Stroke volume (ml/min)	Mean transit time (sec)	Central blood volume (ml)	\dot{V}_I (ml/min)	f (No./min)	V_T (ml)
Muscovy duck	2.16	844	130	6.8	7.5	100	700	10.5	69
	± 0.09	± 68.5	± 13.3	± 0.3	± 0.03	± 4.89	± 26.8	± 0.45	± 3.96
White Pekin	2.4	973	178	5.5	4.3	67	807	8.2	98
	± 0.04	± 46.2	± 6.35	± 0.25	± 0.196	± 3.4	± 34	± 0.37	± 3.37

Species	P_{I,O_2} (mm-Hg)	P_{E,O_2} (mm-Hg)	P_{A,CO_2} (mm-Hg)	P_{A,O_2} (mm-Hg)	P_{V,O_2} (mm-Hg)	P_{A,CO_2} (mm-Hg)	P_{V,CO_2} (mm-Hg)	Arterial pH	Venous pH	\dot{V}_{O_2} (ml/min)	\dot{V}_{CO_2} (ml/min)	R.Q.
Muscovy duck	147	96.6	34.16	96.1	55.9	35.9	42.58	7.46	7.42	50	34	0.68
	± 1	± 2.9	± 2.2	± 2.2	± 0.66	± 1.6	± 1.85	± 0.008	± 0.01			
White Pekin	146	100.1	34.2	93.1	63.3	36.3	37.25	7.42	7.39	51.9	39.2	0.76
	± 0.92	± 1.8	± 1.04	± 1.86	± 1.71	± 1.02	± 1.5	± 0.01	± 0.016			

Fig. 1. Changes in tidal volume (V_T), respiratory frequency (f) and minute inspired volume (\dot{V}_I) during hypocapnic hypoxia. Average values from all White Pekin ducks.

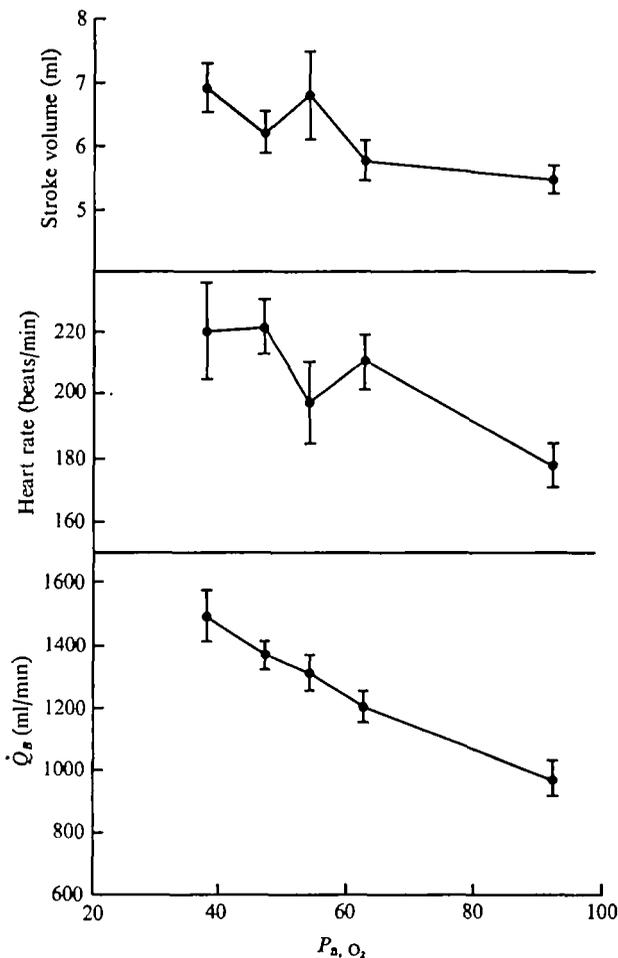


Fig. 2. Changes in stroke volume, heart rate, and cardiac output (\dot{Q}_s) during hypocapnic hypoxia. Average values from all White Pekin ducks.

(b) *Cardiovascular and respiratory responses of White Pekin ducks to progressive hypocapnic hypoxia*

It did not prove possible to subject every animal to identical levels of P_{a,O_2} so the values chosen for averaging represent a value which includes animals within ± 3 mmHg of that point. Consequently the control value (normal P_{a,O_2}) represents the contribution of ten animals to the mean, the P_{a,O_2} values of 63 mmHg, 47 mmHg and 38 mmHg include six animals in each and the 54.5 mmHg value includes only 4.

There were no significant changes in any respiratory parameters until P_{a,O_2} had reached 54.5 mmHg. At this point f was significantly higher than control and continued to increase as the hypoxia increased, more than doubling by the time P_{a,O_2} reached 38 mmHg (Fig. 1). However, \dot{V}_T was more variable and except for the result at P_{a,O_2} 47 mmHg the tendency was for it to decline, although none of the changes were significantly different from control (Fig. 1). \dot{V}_T increased due to the large increase in f , being significantly above control at a P_{a,O_2} of 54.5 mmHg (Fig. 1). From the present

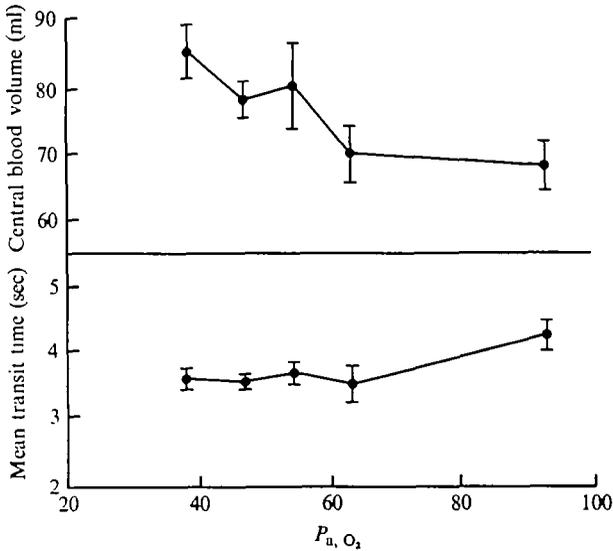


Fig. 3. Change in central blood volume and mean transit time during hypocapnic hypoxia. Average values from all White Pekin ducks.

results it appears that the increase in \dot{V}_T terminated around P_{a, O_2} 47 mmHg when it was almost double the control value (Fig. 1). However, ducks would not tolerate P_{a, O_2} below 38 mmHg without distress and, due to the rather aberrant behaviour of \dot{V}_T at P_{a, O_2} 47 mmHg, any conclusion about the maximum performance of the ventilatory muscles must be treated with suspicion.

Average heart rate increased during hypoxia and was significantly higher than control when P_{a, O_2} reached 47 mmHg (Fig. 2). Stroke volume also increased but none of the changes was significantly different from control (Fig. 2). Due to the increase in heart rate and stroke volume \dot{Q}_b increased, being significantly above control at a P_{a, O_2} of 63 mmHg. Over the range of P_{a, O_2} tested \dot{Q}_b increased almost linearly by about 50% of control values (Fig. 2). Both mean transit time and central blood volume showed significant changes during the hypoxia although they were not significantly different from control until the later stages (Fig. 3).

At all levels of hypoxia ducks were able to remove about 30% of the oxygen from the air. P_{a, O_2} was always slightly below P_{E, O_2} but in no case was the difference significant (Table 2). The oxygen tension difference between arterial and venous blood at rest was 30 mmHg but this decreased during hypoxia to 9.5 mmHg when P_{a, O_2} was 38 mmHg. P_{a, CO_2} and P_{E, CO_2} both fell during hypoxia, the difference from control being significant when P_{a, O_2} was 63 mmHg in the case of the former and 54.5 mmHg in the case of the latter. At no time was P_{a, CO_2} significantly above or below P_{E, CO_2} but at two of the levels of hypoxia the value of P_{E, CO_2} was slightly above that for P_{a, CO_2} (Table 2). A somewhat similar situation, albeit referring to the maximum P_{CO_2} of the expired air, has been recorded in the hen and has been explained on the basis of the morphological structure of the parabronchial avian lung (Piiper, Drees & Scheid, 1970). $P_{\bar{V}, CO_2}$ also fell during hypoxia from 37.3 ± 1.5 mmHg at rest to 25.9 ± 1.5 mmHg when P_{a, O_2} was 38 mmHg. Both arterial pH and venous pH rose as P_{a, CO_2} and $P_{\bar{V}, CO_2}$ fell, the increases being significantly different from control when P_{a, O_2} was 63 mmHg.

Table 2

P_{I,O_2} (mmHg)	P_{A,O_2} (mmHg)	(%) Utilization	P_{E,CO_2} (mmHg)	P_{a,O_2} (mmHg)
145 ± 0.9	100 ± 1.8	31	34.2 ± 1	93 ± 1.8
106 ± 3.7	71.6 ± 4	32.4	30.75 ± 1.4	63.25 ± 1.1
84.25 ± 6	58.6 ± 3.7	30.4	22.5 ± 1.6	54.5 ± 0.96
68 ± 4.7	47.3 ± 1.1	30.4	22.8 ± 2.3	47.4 ± 1.2
60.3 ± 2.1	42.5 ± 1.4	29.52	19.25 ± 0.8	38.25 ± 0.9
$P_{\bar{V},O_2}$ (mmHg)	P_{a,CO_2} (mmHg)	$P_{\bar{V},CO_2}$ (mmHg)	Arterial pH	Venous pH
63.3 ± 1.7	36.27 ± 1	37.3 ± 1.5	7.42 ± 0.01	7.39 ± 0.01
45.3 ± 1.7	30.1 ± 0.8	35.1 ± 0.9	7.49 ± 0.01	7.467 ± 0.008
43.5 ± 3	25.8 ± 3	30 ± 2.7	7.51 ± 0.01	7.47 ± 0.008
36.25 ± 2	22.2 ± 2.5	26.9 ± 3	7.51 ± 0.01	7.48 ± 0.01
28.75 ± 1.7	20.33 ± 0.9	25.9 ± 1.5	7.527 ± 0.02	7.48 ± 0.02

Tensions of oxygen and of carbon dioxide in inspired air, expired air and blood during progressive hypoxia.

% utilization of oxygen from the air breathed was calculated from the mean values of P_{I,O_2} and P_{A,O_2} .

Table 3. Oxygen consumption, CO_2 production and R.Q. during progressive hypoxia

P_{a,O_2} (mmHg)	\dot{V}_{O_2} (ml/min)	\dot{V}_{O_2} (ml/kg/min)	\dot{V}_{CO_2} (ml/min)	\dot{V}_{CO_2} (ml/kg/min)	R.Q.
93 ± 1.8	51.9	21.6	39.2	16.3	0.76
63 ± 1.1	49.3	19.7	45	18.75	0.91
54.5 ± 0.9	41.8	17.4	38.4	16.5	0.92
47 ± 1.27	48.9	19.6	69.8	29.1	1.43
38 ± 0.9	36.5	14.6	40.7	16.95	1.12

Mean values of P_{I,O_2} , P_{E,O_2} , and P_{I,CO_2} and P_{E,CO_2} were used to calculate \dot{V}_{O_2} and \dot{V}_{CO_2} . As Table 3 shows, apart from the rather aberrant value at P_{a,O_2} of 47 mmHg, \dot{V}_{O_2} fell below the control value at all levels of hypoxia, the decline being particularly marked when P_{a,O_2} reached 38 mmHg. Using different methods Butler (1970) obtained a similar fall in oxygen uptake in the duck during progressive hypoxia. \dot{V}_{CO_2} was maintained more or less constant throughout the hypoxia and R.Q. therefore increased from the control value of 0.76 to 1.12 when P_{a,O_2} was 38 mmHg (Table 3).

DISCUSSION

The ventilatory and circulatory responses of man to hypercapnic hypoxia (altitude exposure) have been well documented and reasonable explanations of their adaptive significance, if not their cause, have been given in most instances (Barcroft, 1934; Dejours, 1962; Houston & Riley, 1947; Korner, 1959; Smith & Crowell, 1967; Lamb, Kelly, Smith, Leblanc & Johnson, 1969). At altitudes of 15–20,000 ft hyperventilation at rest is prominent, increases of 115% being common at P_{I,O_2} of 73 mmHg. Consequently the difference in oxygen tension between inspired (or alveolar) air and arterial blood decreases (Houston & Riley, 1947). In the present ducks the difference decreased from 52 mmHg at normal P_{I,O_2} to about 20 mmHg when P_{I,O_2} was 60 mmHg. Other differences in P_{O_2} within the circulatory compartment are also reduced. In man

the difference between arterial P_{O_2} and *mean capillary* P_{O_2} falls from 48 mm at sea level to 4 mm at 20,000 ft. In ducks the difference is smaller under control conditions (20 mmHg) and decreases to around 6 mmHg at a P_{I, O_2} of 60 mmHg. The reduction in P_{O_2} difference occurs early during hypoxia, being 12.5 mmHg at P_{I, O_2} of 106 mmHg and 7.5 mmHg at P_{I, O_2} of 84 mmHg. The main reason for the reduction of this difference would appear to be the characteristics of the oxygen-dissociation curve of the blood although the increase in cardiac output is also a contributory factor (Houston & Riley, 1947).

In man at a simulated altitude of 20,000 ft cardiac activity also increases, pulse rate going up by 40% and \dot{Q}_b by 97% (Houston & Riley, 1947). The performance of the duck's cardiovascular system was somewhat inferior to this at even lower P_{I, O_2} . At P_{I, O_2} of 60 mmHg pulse rate only increased by 25% and \dot{Q}_b by 50%. Butler (1970) reports that a significant tachycardia of about 50% of control frequency is not seen in Mallard ducks until P_{a, O_2} is less than 35 mmHg, a level somewhat below that which was reached in the present experiments. Both Butler (1970, hypocapnic hypoxia) for the duck and Ray & Fedde (1969, normocapnic hypoxia) for the chicken report a fall in blood pressure at the P_{I, O_2} used in the present experiments and this would tend to offset in a small part the increased loading on the heart during hypoxia. Mean transit time in the central vascular bed decreased as \dot{Q}_b increased but central blood volume increased. Much of the increase may have been due to pooling of blood in the pulmonary circuit, due to dilatation of that vascular bed under the influence of an increase in pulmonary artery pressure which has been shown to occur in the chicken during hypoxia (Burton, Besch & Smith, 1968). There may, of course, be increases in other compartments of the central blood volume, e.g. auricles and ventricles.

A general method of assessing the ratio between \dot{V}_I and \dot{Q}_b for 95% oxygen saturation of arterial blood has been presented by Jones, Randall & Jarman (1970). The analysis relates equations for \dot{V}_{O_2} from the air being breathed and blood being perfused through the lungs, by means of a factor ΔP which describes the 'resistance' of the whole system to gas exchange in terms of mmHg. The factor ΔP includes the effects of air and blood shunts as well as the effects of any temporal or spatial diffusion barriers within the lung. In ducks a predicted \dot{V}_I/\dot{Q}_b ratio of 1 will achieve 95% saturation of arterial blood (Jones & Johansen, 1971). In the present experiments the \dot{V}_I/\dot{Q}_b ratio under control conditions, in both species investigated, was in the range of 0.8–0.85 (Table 4). During hypoxia there was a marked reduction in ΔP but the \dot{V}_I/\dot{Q}_b ratio was little affected until P_{a, O_2} was below 54.5 mmHg. Below this value \dot{V}_I increased more rapidly than \dot{Q}_b and the ratio was unity (Table 4). An increase in the ratio might certainly be predicted as the appropriate response since the oxygen-dissociation curve is shifting to the left and a given reduction in the oxygen content of the air will not result in the same reduction in oxygen content of the blood.

In ducks during hypoxia there was a marked elevation in R.Q. whereas Houston & Riley (1947) only recorded a pronounced elevation in R.Q. in one of four subjects, two of the others showing slight increases. The increase in R.Q., which occurred in the present experiments, could have been caused by the effect of hyperventilation in the unsteady state on carbon dioxide stores within the body, or may be indicative of an increase in anaerobic glycolysis. Analysis of P_{CO_2} and pH data for blood by means of a P_{CO_2} /pH diagram showed that as hypoxia progressed the points moved below but to

Table 4. *The ventilation/perfusion (\dot{V}_T/\dot{Q}_B) ratio during progressive hypoxia*

P_{a,O_2}	\dot{V}_T/\dot{Q}_B
93 ± 1.8	0.815 ± 0.06
63 ± 1.1	0.83 ± 0.07
54.5 ± 0.9	0.87 ± 0.1
47 ± 1.27	1.08 ± 0.1
38 ± 0.9	0.998 ± 0.04

the left of the buffer line obtained initially. This indicated that metabolic acids were being added to the blood during hypoxia and that the ducks were not entering a state of simple respiratory alkalosis.

The ventilatory and cardiac responses to hypocapnic hypoxia are in broad agreement with those recorded by others (Butler, 1970). There are obviously some differences in detail between the responses of normocapnic hypoxic ducks and those investigated in the present series of experiments, which may be attributed to the influence of carbon dioxide. For instance, Jones & Purves (1970) recorded increases in both \dot{V}_T and f in Mallards in response to normocapnic hypoxia, whereas in the present experiments f increased slightly more but \dot{V}_T did not change significantly so that the overall elevation in ventilation volume was not as large. Another notable difference concerned cardiac performance. In contrast to the tachycardia exhibited by the present ducks and those investigated by Butler (1970), Jones & Purves (1970) found only slight and variable effects on heart rate of a P_{a,O_2} of 37–45 mmHg. Jones & Purves (1970) confirmed that increases in P_{a,CO_2} not only increased ventilation but also caused bradycardia, which supports the suggestion that the difference in P_{a,CO_2} is responsible for the variation in results between the above experiments.

SUMMARY

1. Cardiac output, ventilatory minute volume and gaseous exchange at both tissues and lungs have been recorded in restrained unanaesthetized ducks exposed to simulated high altitudes.
2. A comparison between two species of duck showed that despite a significantly lower heart rate in resting Muscovy ducks, cardiac output, on a weight basis, was the same as in White Pekin ducks. Respiratory frequency and tidal volumes differed in the two species although their minute volumes were in the same range.
3. Ducks responded to reduction in oxygen tension of arterial blood (P_{a,O_2}) by increases in cardiac output and ventilatory minute volume, both being significantly above control (normal P_{a,O_2} at rest) when P_{a,O_2} was in the range 54.5–63 mmHg. At all levels of hypoxia ducks were able to remove about 30% of the oxygen from the ventilated air.
4. When P_{a,O_2} was 38 mmHg the P_{O_2} difference between arterial and venous blood had decreased by 20.5 mmHg from control. P_{a,CO_2} and P_{v,CO_2} fell during hypoxia and arterial and venous pH rose.
5. The rate of oxygen uptake (\dot{V}_{O_2}) fell markedly at the lowest level of hypoxia but \dot{V}_{CO_2} remained constant so that R.Q. rose from 0.76 at control at 1.12 at P_{a,O_2} of 38 mmHg.

6. It is concluded that there are many basic similarities between the cardiovascular and respiratory responses of ducks and mammals when exposed to simulated high altitude.

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