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INTRAPULMONARY CHEMORECEPTOR CONTROL OF VENTILATORY MOVEMENTS IN THE SELF-VENTILATING CHICKEN

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SUMMARY

The importance of intrapulmonary chemoreceptors (IPC), sensitive to P_{CO_2} in the lung, in the control of ventilatory movements is yet to be demonstrated in the self-ventilating bird. We distinguished between the effects of P_{CO_2} on IPC and on extrapulmonary CO_2-sensitive receptors (EPC) in anaesthetized cockerels by denervating IPC in the right lung, ligating the left pulmonary artery and changing P_{ICO_2}. Left IPC were thus exposed to a combination of P_{CO_2} from inspired gas and dead space, while EPC were exposed to greatly increased arterial P_{CO_2} resulting from the ventilation-perfusion inequality. At 0 Torr P_{ICO_2}, steady state ventilatory depth and frequency did not change ($P > 0{\cdot}5$) after the ligation, although Pa_{CO_2} rose by $12{\cdot}2 \pm 1{\cdot}7$ Torr. After the ligation, ventilatory movements were more sensitive to increasing P_{ICO_2}. Tracheostomy, which results in a greater decrease in P_{CO_2} in the innervated lung after ligation, produced greater effects. We conclude that these responses were due to the strong controlling influence of IPC on ventilatory movements in the self-ventilating cockerel.

INTRODUCTION

In the bird lung, there are CO_2-sensitive receptors that can affect ventilatory movements. These intrapulmonary chemoreceptors (IPC) are reported (Nye & Burger, 1978) to be located throughout the gas exchange region and are especially sensitive to low P_{CO_2}. IPC increase their discharge as P_{CO_2} in the airways decreases; in anaesthetized, unidirectionally ventilated birds with opened thoracoabdominal cavities, this discharge dramatically decreases tidal volume and increases, though more variably, respiratory frequency (Osborne, Mitchell & Powell, 1977; Osborne & Mitchell, 1978; Burger & Estavillo, 1978). According to Bouverot (1978), however, the concept that 'intrapulmonary CO_2-sensitive receptors may parallel systemic chemoreceptors in a physiological CO_2-chemoreflex drive of ventilation' is yet to be confirmed.

It is likely that the controlling influences of both IPC and extrapulmonary CO_2-sensitive chemoreceptors (EPC) normally change in parallel with Pa_{CO_2} if P_{ICO_2}

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increases. Ligation of a pulmonary artery to one lung produces a large ventilation-perfusion inequality since gas inhaled through the non-perfused lung does not contribute to gas exchange. Pa_{CO_2} will rise considerably unless minute ventilation is increased. Activity from arterial and central chemoreceptors should normally result in such an increase. However, the P_{CO_2} of the gas in the non-perfused lung will be decreased after ligation since CO_2 is not added to the gas from the venous pool. Therefore, the output from IPC in this lung should inhibit increases in minute ventilation caused by activity from arterial chemoreceptors. The degree of inhibition would be an index of the effectiveness of IPC in controlling ventilation.

The amount of CO_2 entering the non-perfused lung in such a preparation is determined by the dead space volume and the fractional concentration of CO_2 in the dead space. The latter increases with Pa_{CO_2}, so that P_{CO_2} in the non-perfused lung is affected indirectly by the level of Pa_{CO_2}. Reducing the dead space by tracheostomy will cause P_{CO_2} in the non-perfused lung to drop even more after pulmonary artery ligation and, if IPC do affect ventilation, should thus produce greater inhibition to ventilation than in intact birds.

We ligated the left pulmonary artery of intact and tracheostomized, anaesthetized, non-artificially ventilated chickens and cut the innervation to the IPC of the right lung. In such a preparation, the effects of P_{CO_2} on IPC could be dissociated from the effects of Pa_{CO_2}. We measured steady state ventilatory movements at three different P_{ICO_2} in order to determine if the IPC control ventilatory movements.

METHODS

Initial animal preparations

Fifteen White Leghorn cockerels (*Gallus domesticus*), 15–20 weeks of age and 1·2–1·7 kg body weight, were used. Each cockerel was restrained on its back and implanted, under local anaesthetic (2% procaine), with a cannula in the cutaneous ulnar vein. Initial doses of about 20–30 mg kg^{-1} sodium pentobarbital were given to anaesthetize the cockerels during surgery. Afterwards, supplementary doses of 2 mg kg^{-1} were given, when needed, throughout the experiment to maintain a level of 'light' anaesthesia (see Fedde, Burger & Kitchell, 1963). The right common carotid artery was cannulated to record blood pressure and heart rate (Statham transducer, P23A). Colonic temperature was measured by a thermistor probe (Yellow Springs Instrument Col, 401) inserted 10 cm into the cloaca; a second thermistor probe, similarly inserted, was attached to a proportional temperature controller (Cole-Palmer, Versa-Therm) which varied the current to a heat lamp to maintain colonic temperature at 41·5 °C. Depth and frequency of ventilatory movements were measured by a modified strain gauge transducer (Barnas, Estavillo, Mather & Burger, 1981) attached to the centre of the sternum. Blood pressure and ventilatory movements were recorded on a multichannel pen recorder (Offner, type R).

Additional animal preparations and protocol

Series 1

During the experiments of Series 2, we used high levels of $P_{I_{CO_2}}$ in order to obviate the hypoxia resulting from the ventilation-perfusion inequality after pulmonary artery ligation. In addition, we denervated IPC in the right lung by cutting the right vagus; consequently, the right carotid body was also denervated. Therefore, we first tested five cockerels to determine whether the high levels of $P_{I_{O_2}}$ and the unilateral vagotomy used in the preparations in Series 2 affected the response to CO_2. Oxygen or air with desired amounts of CO_2 from premixed tanks or blended with flow meters was delivered through a Lucite tube (10 cm diameter) into which the cockerel's head was inserted. Gas flow rate was greater than 5 l min^{-1} and was directed past the cockerel's head, out of the end of the tube. $P_{I_{CO_2}}$ was measured with either an infrared CO_2 analyser (Beckman, LB-2) or a blood-gas electrode (Instrumentation Lab, Inc., Model 123-S1).

We presented three of the cockerels with the following series of $P_{I_{CO_2}}$ twice, first in oxygen and then in air, for periods of at least 5 min at each level. 0 Torr, 21 Torr and 35 Torr. We cut the right vagus midway along the neck and repeated the two series of $P_{I_{CO_2}}$. With two other cockerels, we gave the $P_{I_{CO_2}}$ series in air before that in oxygen, both before and after vagotomy. Ventilatory movements and Pa_{CO_2} were measured at the end of each period.

Series 2

The remaining ten cockerels were additionally prepared, and additional measurements were made, in the following ways. An incision was made through the skin midway between the highest point of the sternum and the left shoulder joint exposing the pectoralis thoracicus and supracoracoideus muscles. We used haemostats to separate the bands of the pectoral muscles, exposing the thoracic cavity and the pulmonary artery. A length of suture thread was placed loosely around the left pulmonary artery, and both ends were brought to the exterior through a 15 cm silastic tube of 0·5 cm diameter. The opening in the body wall was sutured shut, and the tubing clamped with a haemostat to form an airtight seal. Rubber cannulae (Mallinckrodt, Foley 6), 5·5 cm long, were inserted through the body wall into both the left and right cranial thoracic air sacs and secured into place with a suture thread; placement of each cannula was verified following the experiment. The right vagus was cut midway along the neck, denervating both the right carotid body and lung.

Pa_{CO_2} was sampled (measured with the blood-gas electrode described above) through the cannula in the right carotid artery. The P_{CO_2} values of samples of gas (1 ml) drawn from each cranial thoracic air sac were measured with either the blood-gas electrode or in the infrared analyser. When the infrared analyser was used, the analyser was adapted to measure CO_2 statically, that is, without the usual flow of gas through it. We disconnected the usual regulated vacuum supply from the output port and connected, in its place, a 20 cm long piece of 1·5 mm nylon tubing. We calibrated the analyser with humidified gas delivered from a gas sampling

syringe to the input port. A similar procedure was followed for the cranial thoracic gas samples.

Series 2a

With five of the ten cockerels used in Series 2, we used the same system as used in Series 1 to deliver gases to the cockerel's head. We initially measured air-sac P_{CO_2}, Pa_{CO_2}, ventilatory movements and blood pressure as the cockerel breathed 0 Torr PI_{CO_2} in oxygen. We increased PI_{CO_2} to 21 and 35 Torr in oxygen and repeated the measurements at least 5 min after each change. After returning PI_{CO_2} to 0 Torr, we occluded the left pulmonary artery by tightening the suture thread through the silastic tubing, taking continuous recordings of ventilatory movements and blood pressure. Complete sets of measurements were again taken after at least 5-min periods at 0, 21 and 35 Torr PI_{CO_2} in oxygen.

Series 2b

With the five remaining cockerels of Series 2, the gases were heated and humidified and presented to the trachea, which was cannulated near the clavicle with a 'T' tube. Otherwise, the protocol was identical to that of Series 2a.

RESULTS

Series 1

Two-way ANOVA showed that there were no differences in the depth or in the frequency of ventilatory movements in response to PI_{CO_2} between normal and high PI_{O_2} ($P > 0{\cdot}5$) or before and after unilateral vagotomy ($P > 0{\cdot}5$). There was also no differences ($P > 0{\cdot}5$) in blood pressure or heart rate among the groups.

Series 2a

Table 1 lists the average steady state values of ventilatory movements, Pa_{CO_2} and P_{CO_2} in the cranial air sacs, before and after ligation of the pulmonary artery to the innervated lung. As has been often reported (see Fedde, 1976), increased PI_{CO_2} with intact pulmonary circulation causes increases in the depth of ventilatory movements and decreases, though with a more variable effect, in ventilatory frequency (see also Fig. 1; solid line, closed symbols). Pa_{CO_2} increases with increasing PI_{CO_2}. Before ligation of the pulmonary artery, the P_{CO_2} measured from the two cranial thoracic air sacs at a given PI_{CO_2} did not differ from each other ($P > 0{\cdot}4$, Student's paired t-test).

Ligation of the pulmonary artery of the innervated lung in these cockerels with intact trachea caused a moderate, short-lasting decrease in the depth and frequency of ventilatory movements (see example, Fig. 2A,B), which returned to approximately pre-ligation values within 1 min. Though blood pressure initially falls upon ligation of the artery, it begins to rise within a few seconds. Average steady state blood pressure, 147 Torr, and heart rate, 318 min^{-1}, were not affected by the ligation or PI_{CO_2}. Five minutes after ligation, P_{CO_2} of the left cranial thoracic air sac (containing gas from the non-perfused lung) decreased by 7·6 Torr (Table 1).

Table 1. *Average (± S.E.) steady state ventilatory depth (VD, in mm), respiratory frequency (f, in min^{-1}), Pa_{CO_2} (Torr) and P_{CO_2} in the left and right cranial thoracic air sacs ($Plas_{CO_2}$ and $Pras_{CO_2}$, respectively; both in Torr) of ten anaesthetized cockerels, before and after ligation of the pulmonary artery to the left, innervated lung at three levels of inspired P_{CO_2} (PI_{CO_2}, Torr).*

		Before ligation					After ligation				
	PI_{CO_2}	VD	f	Pa_{CO_2}	$Plas_{CO_2}$	$Pras_{CO_2}$	VD	f	Pa_{CO_2}	$Plas_{CO_2}$	$Pras_{CO_2}$
Intact ($N = 5$)	0	1·4 ± 0·27	104 ± 18	30·1 ± 1·6	37·1 ± 2·7	36·6 ± 3·0	1·5 ± 0·26	116 ± 14	39·4 ± 1·8*	29·5 ± 1·3*	41·4 ± 1·8
	21	2·0 ± 0·33†	85 ± 16†	33·6 ± 1·8†	39·5 ± 2·4†	39·7 ± 3·2†	2·5 ± 0·47*†	73 ± 32†	39·2 ± 1·2*	33·6 ± 1·5*†	45·6 ± 1·3*
	35	2·8 ± 0·49‡	69 ± 14	41·3 ± 0·8‡	47·9 ± 1·6‡	48·2 ± 2·6‡	3·5 ± 0·45*‡	48 ± 12*‡	49·3 ± 2·6*‡	42·4 ± 1·6*‡	51·8 ± 1·3*‡
Tracheostomized ($N = 5$)	0	1·2 ± 0·31	73 ± 16	30·6 ± 0·8	36·6 ± 2·4	37·6 ± 1·6	1·3 ± 0·27	74 ± 16	45·8 ± 2·7*	18·2 ± 1·4*	48·7 ± 2·3*
	21	2·1 ± 0·46†	62 ± 15†	34·3 ± 1·5†	39·9 ± 2·1†	39·8 ± 1·5	2·8 ± 0·79*†	51 ± 14†	39·6 ± 0·9*†	27·0 ± 1·5*†	44·5 ± 3·1
	35	3·3 ± 0·61‡	46 ± 10	38·3 ± 0·9‡	45·6 ± 1·8‡	45·9 ± 1·6‡	4·8 ± 0·78*‡	31 ± 9*‡	50·4 ± 1·8*‡	38·7 ± 1·1*‡	58·3 ± 2·6*‡

The innervation to the right lung has been cut. Five of the cockerels had intact tracheae while five were tracheostomized.
* Difference from pre-ligation values, Student's paired t-test ($P < 0·05$).
† Difference from 0 Torr PI_{CO_2}, Student's paired t-test ($P < 0·05$).
‡ Difference from 21 Torr PI_{CO_2}, Student's paired t-test ($P < 0·05$).

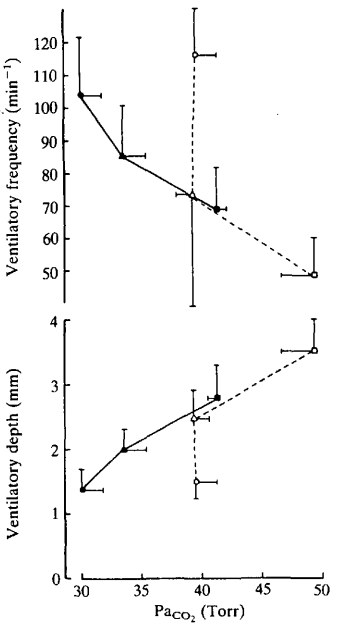

Fig. 1. Effects of Pa_{CO_2} on ventilatory frequency and depth in cockerels with intact tracheae, before (solid lines, closed symbols) and after (dotted lines, open symbols) ligation of the left pulmonary artery; the right vagus has been cut. Each point represents five animals. Standard errors represented by bars. Circles, triangles and squares = 0, 21 and 35 Torr PI_{CO_2}, respectively. Note that: (1) after ligation, Pa_{CO_2} is much higher at 0 Torr PI_{CO_2}, though ventilatory movements do not change; and (2) Pa_{CO_2} does not change as PI_{CO_2} increases to 21 Torr after ligation.

Ventilatory depth and frequency, however, did not differ from pre-ligation values ($P > 0{\cdot}5$), although Pa_{CO_2} rose by 9·6 Torr. Therefore, at 0 Torr PI_{CO_2}, Pa_{CO_2} is much higher after ligation for a given depth or frequency of ventilatory movement (Fig. 1; dotted lines, open circles).

An increase in PI_{CO_2} after ligation increased the depth of ventilatory movements and decreased ventilatory frequency more than it did before ligation. For example, before pulmonary artery ligation, inhalation of 21 and 35 Torr PI_{CO_2} increased the depth of ventilatory movements by 43 and 100 %, respectively; after ligation, depth increased to 67 and 133 % during the same respective increases in PI_{CO_2} (Fig. 1; dotted lines, open symbols). Because of these effects on ventilation, Pa_{CO_2} did not increase after the pulmonary artery had been ligated when PI_{CO_2} was changed from 0 to 21 Torr; in contrast, Pa_{CO_2} increased by 3·5 Torr when the same PI_{CO_2} increase occurred before ligation. Moreover, looking at the data from a different viewpoint, these results observed during the PI_{CO_2} increase to 21 Torr after ligation

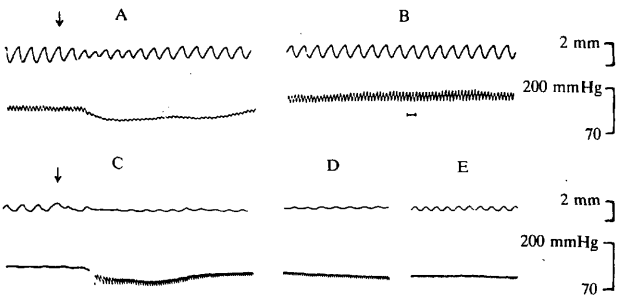

Fig. 2. Effect of ligation of the left pulmonary artery (at arrow) on ventilatory movements (upper traces) and blood pressure (lower traces) in a cockerel with an intact trachea (A and B) and in a tracheostomized cockerel (C, D and E). Traces in (B) and (D) begin 35 s after the end of ligation, and those in (E) begin 70 s after the end of ligation. Horizontal bar in (B) = 1 s. Note that: (1) ventilatory depth and frequency decrease immediately after ligation but rise again towards pre-ligation values; (2) blood pressure and heart rate decrease upon ligation but begin to increase shortly afterwards; and (3) responses are greater, and time before a steady state is reached after ligation is longer, after tracheostomy.

of the pulmonary artery clearly demonstrate that ventilatory movements are changed by the effect of P_{CO_2} on IPC, although Pa_{CO_2} remains almost constant.

Series 2b

Pulmonary artery ligation following tracheostomy results in a more pronounced depression of depth and frequency of ventilatory movements and a longer period before the return towards pre-ligation values (Fig. 2C,D,E) than in non-tracheostomized cockerels. Table 1 shows that in tracheostomized cockerels, left cranial air sac P_{CO_2} decreased further than in the intact cockerels after ligation. The increases in Pa_{CO_2} after ligation were larger in tracheostomized than intact cockerels, but steady state ventilatory movements did not differ ($P > 0{\cdot}5$) from pre-ligation values (see also Fig. 3, open circles).

Note that, in contrast to the increases in Pa_{CO_2} seen in the cockerels before ligation, increasing PI_{CO_2} to 21 Torr after ligation decreased Pa_{CO_2} by 6·2 Torr in tracheostomized cockerels (Fig. 3; dotted lines, open triangles). In other words, ventilation was enhanced so much by the increases in PI_{CO_2} that Pa_{CO_2} was driven lower. A further increase in PI_{CO_2} to 35 Torr resulted in Pa_{CO_2} and depth of ventilatory movements greater than before the ligation; ventilatory frequency was less.

The average steady state blood pressure of the tracheostomized cockerels, 118 Torr, was significantly less than in the intact cockerels but was not affected by pulmonary artery ligation or PI_{CO_2}. Heart rate (average, 337 min^{-1}) was not affected by tracheostomy or ligation but increased slightly with increasing PI_{CO_2}.

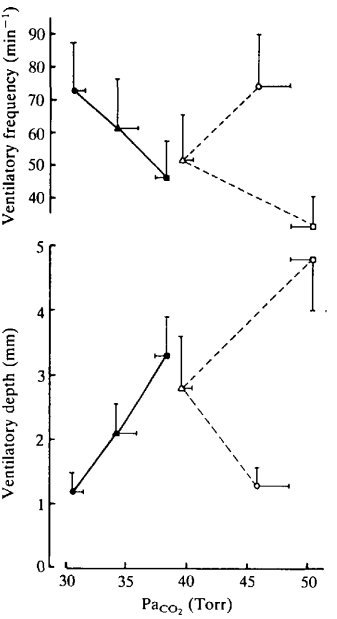

Fig. 3. Effects of Pa_{CO_2} on ventilatory frequency and depth in tracheostomized cockerels, before (solid lines, closed symbols) and after (dotted lines, open symbols) ligation of the left pulmonary artery; the right vagus has been cut. Each point represents five animals. Standard errors represented by bars. Circles, triangles and squares = 0, 21 and 35 Torr Pi_{CO_2}, respectively. Note that similar relationships are evident in cockerels with intact tracheae (Fig. 1); however, after ligation, increases in Pa_{CO_2} at 0 Torr Pi_{CO_2} and decreases in Pa_{CO_2} at 21 Torr Pi_{CO_2} are greater.

DISCUSSION

These results clearly demonstrate that the intrapulmonary chemoreceptors (IPC) contribute a physiological CO_2-chemoreflex drive of ventilation in non-artificially ventilated chickens. Immediately after ligation of the left pulmonary artery during 0 Pi_{CO_2}, P_{CO_2} in the innervated, non-perfused left lung decreases (as shown by air sac P_{CO_2}). Ventilatory movements become more shallow since the innervated IPC must begin to discharge more rapidly. Tracheostomy reduces the inhalation of dead space, which contains gas with high P_{CO_2} expired from the right lung, and innervated lung P_{CO_2} decreases further. Therefore, ventilatory movements upon ligation after tracheostomy are even more severely depressed. Subsequent to the initial depression after ligation, ventilatory movements begin to increase in depth as Pa_{CO_2} rises due to the hypoventilation. Tracheostomy prolongs, but enhances, this rise because of the aforementioned enhancement of IPC discharge. In both intact and tracheostomized cockerels, ventilatory movements reach approximately pre-

ligation values despite the fact that IPC stimulation and, therefore, increases in Pa_{CO_2} are greater in the latter. It is not obvious why the increased stimulation to ventilatory movements caused by elevated Pa_{CO_2} is exactly balanced by increased inhibition to those movements from IPC to give pre-ligation levels of ventilatory movements. One factor involved may be that the influences of IPC and EPC are of relatively equal magnitude as suggested by unidirectional ventilation studies (Osborne & Mitchell, 1978; Burger & Estavillo, 1978). In any case, the data show that IPC are capable of a strong controlling influence on ventilatory movements.

An increase in PI_{CO_2} to 21 Torr led to increased Pa_{CO_2} in all cockerels with intact pulmonary circulations. In contrast, Pa_{CO_2} decreased with this rise in PI_{CO_2} after ligation of the pulmonary artery in tracheostomized cockerels (Fig. 3, open triangles) and did not change in the intact cockerels (Fig. 1). These lack of increases in Pa_{CO_2} resulted from the greater increases in ventilatory depth after ligation compared to before ligation, as can be seen in Table 1; ventilatory frequency, on the other hand, decreases. These changes alone will increase the percentage of 'effective' minute ventilation, which contributes to gas exchange, contained within any total minute ventilation, since dead space will make up a lesser portion of each tidal breath. Since changes in the depth of ventilatory movements are roughly proportional to those in tidal volume (Kuhlmann & Fedde, 1976), we can calculate total minute ventilation in units of $mm\,min^{-1}$. Minute ventilation calculated in this way is 7·5 % higher than pre-ligation values at 21 Torr PI_{CO_2} after ligation in the intact cockerels; it is 10 % higher in the tracheostomized cockerels. Therefore, a cockerel whose pulmonary artery is ligated is more sensitive to an increase in PI_{CO_2}.

This hypersensitivity to increased PI_{CO_2} can be expected since IPC show maximal sensitivity at low P_{CO_2}, that is, the slope of their sensitivity curve to P_{CO_2} is greatest below 21 Torr (Nye & Burger, 1978). In our preparation, after pulmonary artery ligation at 0 Torr PI_{CO_2}, IPC are exposed to a minimum P_{CO_2} of near zero as P_{CO_2} ranging from inhaled to end-tidal levels reaches the lung. Changing to 21 Torr PI_{CO_2} increases this minimum to 21 Torr. In the cockerel with a perfused lung, on the other hand, venous blood buffers these P_{CO_2} changes and IPC are probably not exposed to such large changes in P_{CO_2} when PI_{CO_2} is increased. Therefore, IPC are exposed to a greater change in P_{CO_2} at their most sensitive range of sensitivity if the lung is non-perfused.

Osborne & Mitchell (1977) found that Pa_{CO_2} did not increase with increasing PI_{CO_2} in cockerels with both lungs perfused if PI_{CO_2} was less than 21 Torr. They concluded that IPC were responsible for the increases in ventilation that led to this eucapnic hypercapnea. As in their study, we found that at PI_{CO_2} higher than 21 Torr, ventilatory sensitivity *via* IPC control was less important. We found less pronounced changes in ventilation when PI_{CO_2} increased from 21 to 35 Torr; values for both ventilatory depth and frequency seemed to fall along the Pa_{CO_2} responses curves, if extended, calculated before ligation (Figs 1, 3, solid lines). Again, this can be expected since Pa_{CO_2} is more important in determining ventilation at high P_{CO_2} levels (Osborne & Mitchell, 1978). If we again estimate ventilation as the product of ventilatory depth and amplitude, we find no increases when PI_{CO_2} rose from 21 to 35 Torr. This phenomenon, where the decreases in respiratory fre-

quency in response to high levels of $P_{I_{CO_2}}$ overshadow the increase in tidal volume, has been often observed in birds (Fedde, 1976) and is yet unexplained. However, the continuous nature of both the ventilatory frequency and ventilatory depth responses to high Pa_{CO_2} in the present study suggests that the lack of an increase in minute ventilation at high $P_{I_{CO_2}}$ is merely a property of the avian respiratory control system. More exact examination of frequency-tidal volume relationships, which has not yet been done in birds, may help clarify such relationships.

Banzett, Coleridge & Coleridge (1978) similarly ligated one pulmonary artery in artificially ventilated dogs. Pa_{CO_2} rose over 6 Torr after the ligation, but phrenic nerve activity did not change significantly. They attribute their results to a 'pulmonary-CO_2 ventilatory reflex' mediated by pulmonary stretch receptors (PSR) which depress ventilatory movements when intrapulmonary CO_2 falls below normal. The correlation between bird IPC and mammalian PSR in controlling ventilatory movements may prove to be an interesting area of further experimentation.

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