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Open-loop respiratory chemosensitivity in chickens and ducks

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JONES, DAVID R., AND OWEN S. BAMFORD. *Open-loop respiratory chemosensitivity in chickens and ducks. Am. J. Physiol.* 230(4): 861–867. 1976.—In order to study the behavior of the avian respiratory center under open-loop conditions, recordings of respiratory motor discharges were made from an intercostal nerve in a unidirectionally ventilated paralyzed preparation during normoxic hypercapnia, hypoxic normocapnia and asphyxia, and under control conditions. Respiratory motor burst rate (RMBR) in ducks showed a maximum at 40 mmHg Pa_{CO_2} with depression at higher and lower values, both in the steady state and during rapid transients. RMBR was apparently independent of Pa_{CO_2} in chickens, and of Pa_{O_2} in both species. During asphyxia, RMBR in ducks fell steadily while the number of spikes in each burst did not change. RMBR during asphyxia bore the same relationship to Pa_{CO_2} as during unidirectional ventilation. Chickens did not respond consistently to asphyxia, but RMBR tended to increase and, in some cases, the number of spikes per burst also increased. Possible mechanisms and functions of the asphyxic respiratory depression observed in ducks are discussed.

respiratory motor nerves; hypoxia; hypercapnia; asphyxia; diving

THE NORMAL PATTERN of respiratory chemosensitivity in birds is well established and is generally similar to that of mammals, both hypoxia and hypercapnia inducing increases in minute volume and tidal volume. This is true of diving birds (e.g., ducks) as well as of nondivers (e.g., chickens and pigeons), and there is no evidence for reduced CO_2 sensitivity in divers. However, there is some evidence from mammals (8) that the normal pattern is altered in the open-loop condition, i.e., in the absence of feedback from the periphery to the respiratory center. Under these conditions the respiratory center appears to become less sensitive to CO_2, and the respiratory stimulation of hypercapnia may become respiratory depression. The importance of rhythmic pulmonary afferent traffic in the regulation of avian respiration has also been demonstrated by Kunz and Miller (15) and it is, therefore, possible that a bird in a state of apnea, whether voluntary or involuntary, may show a sensitivity to CO_2 which is somewhat different from that normally seen in a breathing bird. This, in turn, raises the possibility that such a mechanism might play a part in the maintenance of diving apnea which may be prolonged despite the progressive hypoxic hypercapnia that, under normal circumstances, constitutes a powerful stimulus to breathing.

To study respiratory central activity in mammals under open-loop conditions, Cohen (8) recorded phrenic discharge in vagotomized cats with spinal section and pneumothorax. To achieve the same effect in birds, it is only necessary to record respiratory motor nerve activity in the paralyzed bird on unidirectional ventilation. Unidirectional ventilation has been widely used in avian studies since its introduction by Burger and Lorenz (5), for the tubular structure of the avian gas exchanger allows the ventilating gas mixture to be passed over it at many times the normal rate. Blood gas tensions may be altered rapidly and precisely by changing the composition of the gas stream, and the outflowing gas is usually close to equilibrium with arterial blood. The paralyzed unidirectionally ventilated bird is in an open-loop condition in regard to its respiratory control system, and this preparation therefore allows study of the respiratory sensitivity to CO_2 in the absence of respiratory feedback.

Paralyzed unidirectionally ventilated ducks were exposed to a range of steady Pa_{O_2} and Pa_{CO_2} levels and to asphyxia while we recorded respiratory motor activity from a cut intercostal nerve. The respiratory motor burst rate (RMBR) was found at each blood gas tension. In some ducks the experiments were repeated after vagotomy to abolish all vagal afferent traffic. Similarly prepared domestic fowl were also tested and the results compared with those from the ducks.

METHODS

Results were obtained from 16 khaki Campbell ducks, weighing between 0.9 and 1.8 kg, and from 5 domestic fowl (Rhode Island red and light Sussex) weighing between 1.1 and 4.0 kg. All preparative operations were of a superficial nature and were performed under local anesthesia (Xylocaine, Astra-Hewlett Ltd.). An ulnar or sciatic vein and a sciatic artery were respectively catheterized with PE-90 and PE-190 tubing filled with heparinized saline. The arterial catheter was connected to a Hewlett-Packard 267 BC physiological pressure transducer via a three-way stopcock that allowed withdrawal of arterial blood samples for analysis through the side arm; the venous catheter was used for drug injection. The trachea was cannulated via a midventral incision in the neck with a well-fitting soft polyvinyl chloride or silicone rubber tube which was connected to a pneumotachograph and Hewlett-Packard model 270

differential gas-pressure transducer for measurement of tracheal air flow. A soft latex tube was inserted into a convenient air sac through a small incision; the interclavicular was used in the duck, and an abdominal sac in the chicken. The tube was then clamped distally and the skin and muscle layers sutured tightly around it.

The bird was secured on the operating table with wings positioned to expose the dorsal part of the thorax, and a light plane of anesthesia was induced by inhalation of halothane (Ayerst Laboratories), the level of anesthesia being reduced if arterial pressure fell. The skin over ribs 4, 5, and 6 on one side was cut and reflected to expose superficial musculature and adipose tissue. The ribs and intercostal muscles were then exposed, and an incision made along the caudal border of a rib. The intercostal nerve was usually visible, adhering loosely to the exposed air sac surface, though sometimes it was attached to the central face of the rib. The nerve, together with any accessible branches, was cut as far distally as possible, separated from underlying membranes, and lifted onto a grounded metal plate, desheathed, and divided. The activity of small nerve slips was recorded using a pair of hook electrodes, and a slip having purely inspiratory or expiratory activity was chosen for study. The nerve on its electrodes was covered in petroleum jelly to prevent drying out.

The bird was then paralyzed by intravenous injection of curare (Metubine iodide, Eli Lilly and Co, Inc., or Tubarine, Burroughs Wellcome Ltd.). When respiratory movements failed, the air-sac tube was unclamped and a stream of humidified air +4–5% CO_2 passed through the tracheal cannula at 2 liters/min. A gas mixing and metering device allowed independent manipulation of N_2, CO_2, and air percentages in the inflowing gas. All experiments were performed at room temperature and the animal's body temperature was monitored with a rectal thermometer and maintained at the normal value of 40°C by an infrared lamp placed over the bird.

In steady-state experiments, a test gas mixture was passed through the bird for 5 min to ensure that a stable state with respect to blood gas tensions and pH had been reached. A recording was made of the respiratory motor activity and arterial blood pressure, and an arterial blood sample taken and analyzed on a Radiometer BMS3 and PHM71 system for Pa_{O_2}, Pa_{CO_2}, and pH_a. In some experiments the percentages of O_2 and CO_2 in outflowing gas were monitored using Beckman F3 and L15A systems, respectively. In the majority of experiments in both ducks and chickens, the partial pressures of O_2 and CO_2 calculated from these percentage composition measurements, after correction for water vapor pressure, were within 5 mmHg of equilibrium with the blood gases. Following analysis of the blood sample, the composition of the ventilating gas stream was altered and another 5 min allowed for equilibration.

The effect of sudden hypocarbia on central respiratory periodicity was studied in ducks by raising inflowing CO_2 to over 6% and, after establishment of steady-state conditions, abruptly shutting off the CO_2. It was not possible to use blood samples to monitor the time course of the change in Pa_{CO_2} as it was essentially complete in less than a minute, but FE_{CO_2} was recorded in the outflowing gas using the Beckman apparatus. In four ducks the central respiratory periodicity in response to both steady-state normoxic hypercapnia and transient hypocapnia was studied before and after bilateral cervical vagotomy. No vagotomies were performed on the chickens.

In asphyxia experiments the composition of the ventilating gas was adjusted to give blood gas tensions within the preparalysis control range, after which airflow was shut off and a continuous recording made of respiratory motor activity during asphyxia. The air sacs were left open to the atmosphere so the pulmonary system was deflated. In ducks, arterial blood was sampled at 150 s and gas flow restored after 180 s; in chickens, sampling and restoration of gas flow occurred after 90 and 120 s of asphyxia, since these times gave comparable values for blood gases.

Data were recorded on a seven-channel FM tape recorder (Hewlett-Packard 3907C) and integrated neural activity, arterial blood pressure, and tracheal air flow were displayed on a chart recorder. Periodicity of the respiratory central motor bursts was measured from the chart record, while the tape was analyzed to determine the respiratory burst amplitude, in terms of number of action potentials per burst, using a Digital LAB 8-e computer. This analysis was only used with data obtained from asphyxia and CO_2 transient experiments, as in steady-state experiments it was found that reliable comparison of signals, recorded at intervals over long time periods (1 h), was precluded by variations in the signal-to noise ratio due to accumulating physiological fluids around the electrodes.

Where appropriate, values are given in text and figures as means ± 1 standard error of the mean (SE). "Significant" indicates $P < 0.05$. "Asphyxia" denotes the absence of lung ventilation.

RESULTS

Changes in respiratory motor burst rate in response to changes in Pa_{CO_2}. In both ducks and chickens rhythmic activity in the intercostal nerves continued after paralysis, provided Pa_{CO_2} was over 20–30 mmHg. Burst profiles were very similar before and after paralysis, provided blood gas tensions and pH_a were kept constant (Fig. 1). In paralyzed birds at Pa_{O_2} levels of 90–120 mmHg, 113 recordings were made from ducks and 33 from chickens of respiratory motor activity at steady Pa_{CO_2} values between 8 and 80 mmHg in ducks, and 20 and 100 mmHg in chickens. For each recording the mean interburst interval was determined and converted to rate (min^{-1}) to give the respiratory motor burst rate (RMBR). In ducks, data were grouped in six ranges of Pa_{CO_2}, points within each range being pooled to yield a mean and standard error for the rate. Since fewer points were obtained from chickens, the data in this case were divided into a low and high range of Pa_{CO_2}.

Results are shown in Fig. 2*A*. In ducks, RMBR showed a maximum of 9.75 min^{-1} at 30–40 mmHg Pa_{CO_2} and below this the rate declined sharply. In some individuals there was no detectable activity in the intercostal nerve at Pa_{CO_2} below 20 mmHg. Above 40 mmHg the

rate declined more slowly, and in some cases activity ceased at a Pa_{CO_2} of 60 mmHg. In no case was there any acceleration of RMBR above 40 mmHg Pa_{CO_2}. In chickens there was no significant change in RMBR between the low and high ranges of Pa_{CO_2}, the mean value for RMBR remaining at about 8 min^{-1} over a range of a Pa_{CO_2} of 30 mmHg (Fig. 2). Below 28 mmHg Pa_{CO_2} respiratory activity became irregular and rate measurements unreliable.

Cardiovascular responses to blood gas changes were different in ducks and chickens. In normoxic hypercapnia, heart rate and mean arterial pressure of the duck declined from control values by 18 and 9%, respectively, while in the chicken they rose by 13 and 9%, respectively.

Some observations were made of the response to a step change in FI_{CO_2} from 0.06 to 0. FE_{CO_2} values were measured by the Beckman analyzer, a correction being applied for transit time in the sampling tube so that the true FE_{CO_2} time course could be established and compared with the RMBR response. In most such experiments, RMBR rose in the first few seconds of the change, then declined toward zero as FE_{CO_2} and Pa_{CO_2} continued to fall. A record from a typical experiment is shown in Fig. 3*A*, in which Pa_{CO_2} has been derived from the measured value of FE_{CO_2} by using the previously determined relationship between them in this duck. The value of RMBR for a given Pa_{CO_2} in such experiments was generally in good agreement with that obtained in steady-state experiments, though in some cases where the FE_{CO_2} fall was rapid, the phase of increasing RMBR was absent and only the decline was seen. Mean number of spikes per burst declined by 26% by 40–60 s after the withdrawal of CO_2 from the ventilating gas stream.

In order to distinguish between central and peripheral mediation of the central respiratory rate response to CO_2, this response was measured in four ducks before and after bilateral cervical vagotomy using steady-state

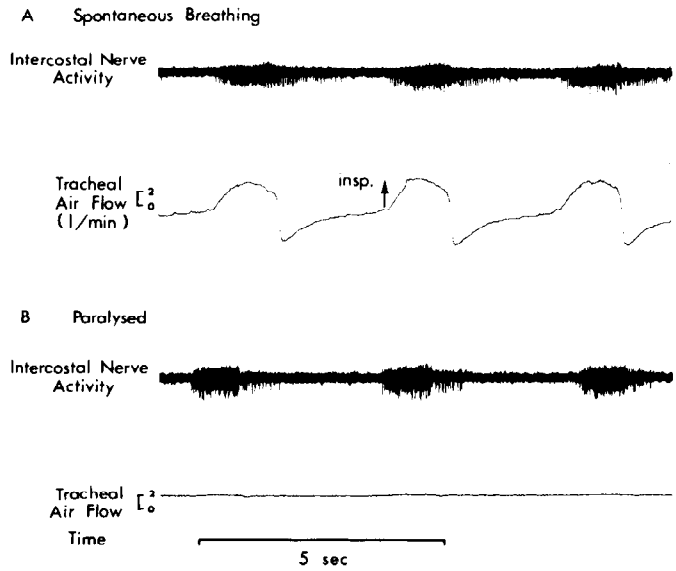

FIG. 1. Neurograms from cut central end of an intercostal nerve of a duck, during *A:* spontaneous breathing; and *B:* unidirectional ventilation after paralysis.

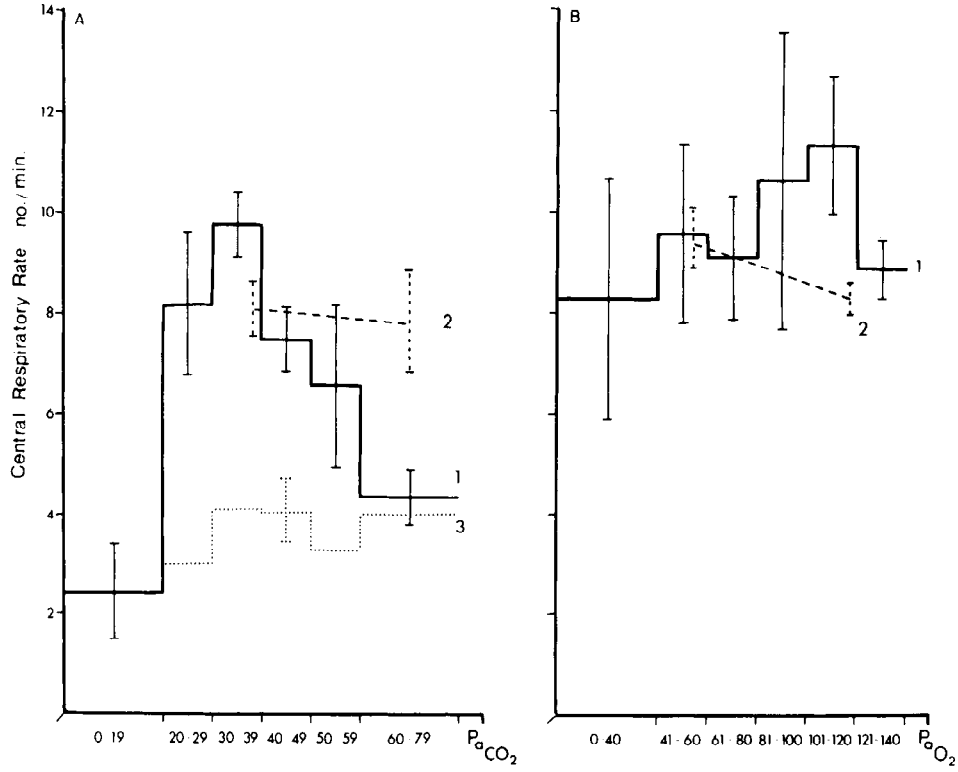

FIG. 2. Relationship between central respiratory rate and steady-state Pa_{CO_2} (mmHg) and Pa_{O_2} (mmHg) in ducks and chickens. *A:* central respiratory rate in relation to Pa_{CO_2}, with Pa_{O_2} in control range, in intact ducks, 1; intact chickens, 2; and vagotomized ducks, 3. *B:* central respiratory rate in relation to Pa_{O_2}, with Pa_{CO_2} in the control range, in intact ducks, 1; and chickens, 2. All values given are means ±SE. Data are grouped as described in text.

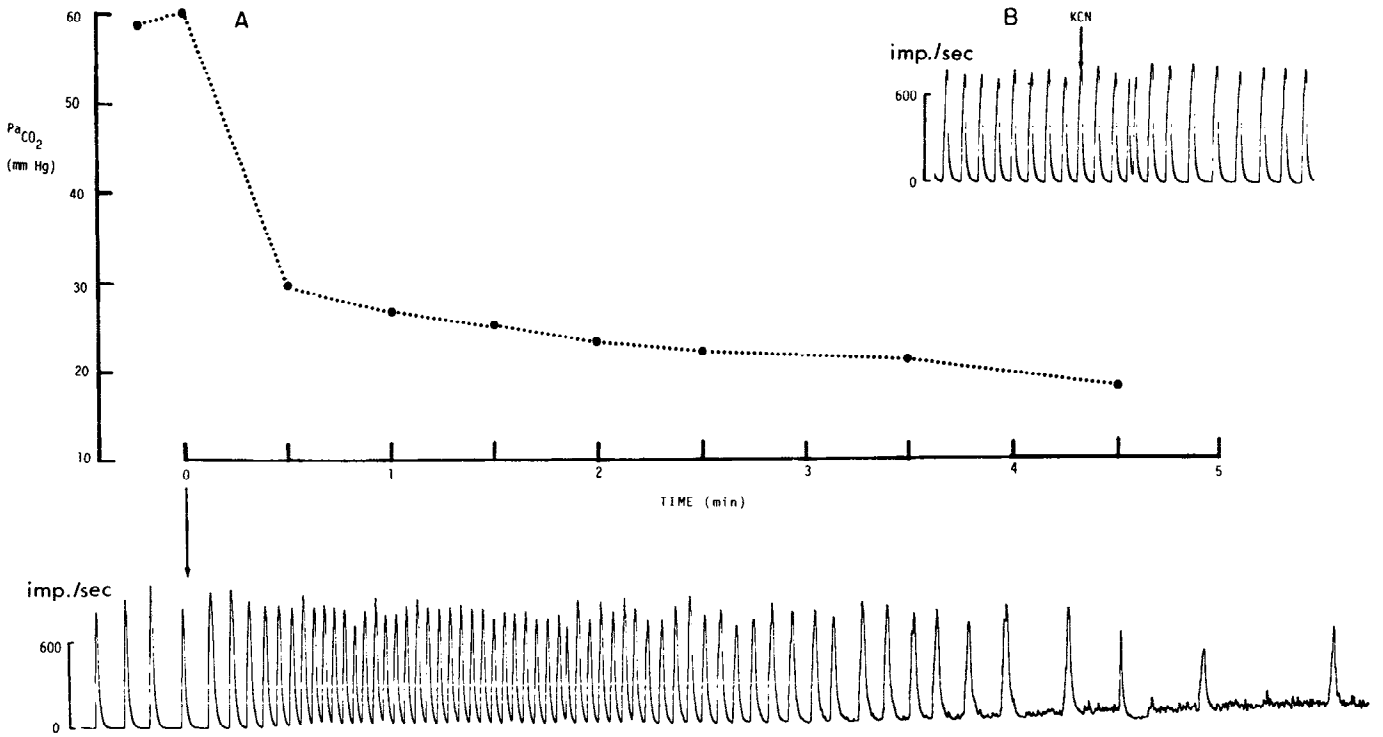

FIG. 3. *A:* Effect of sudden withdrawal of CO_2 from ventilating gas stream on central respiratory activity. Records, reading from above down: Pa_{CO_2} (mmHg), derived from $F_{E_{CO_2}}$; time; intercostal nerve firing rate (impulses/s). CO_2 was shut off at *time 0. B:* effect of intravenous injection of 80 μg KCN in saline on central respiratory activity. Trace shows intercostal nerve firing rate (impulses/s). Arrow shows time of injection. Time scale is same as that of *A*.

and transient values for $F_{I_{CO_2}}$. Bilateral vagotomy was always followed by a marked reduction in RMBR, as well as tachycardia. After vagotomy, RMBR was in the range of 3-4 min^{-1} regardless of the rate before vagotomy, and the responses described above were virtually abolished, both in steady-state (Fig. 2*A*) and in step-change experiments.

Changes in respiratory motor burst rate in response to steady-state normocapnic hypoxia. In ducks, 54 recordings of central respiratory motor activity were made at values of Pa_{O_2} from 15–130 mmHg and in chickens, 26 recordings at values of Pa_{O_2} from 30 to 120 mmHg. In both species Pa_{CO_2} was held in the range which had been recorded from the animals before paralysis (30–34 mmHg).For ducks the values of RMBR against Pa_{O_2} were grouped as described for exposure to normoxic hypercapnia (first section in RESULTS) and are illustrated in Fig. 2*B*. It can be seen that Pa_{O_2} did not significantly affect RMBR over the range used when Pa_{CO_2} was maintained at control levels. To test the effect of even more intense stimulation of oxygen-sensitive receptors, intravenous 1-ml injections of 80 μg KCN in 0.9% NaCl were made, and there was usually a transient increase in which the burst rate doubled momentarily, though the burst amplitude remained constant (Fig. 3A). Control injections of saline alone were without effect. These results suggest that very intense stimulation of oxygen receptors is necessary before the central respiratory rate is affected under the conditions of our preparation. Chicken data were divided into only two groups—a high Pa_{O_2} group (average value = 113.0 ± 2.5 mmHg) and a low Pa_{O_2} group (average value = 49 ± 2.4 mmHg). The central respiratory rates at these levels of Pa_{O_2} are illustrated in Fig. 2*B*. As with the ducks, chickens under the conditions of our experiments failed to show any significant change in central rate with hypoxia.

The cardiovascular responses to normocapnic hypoxia in both chickens and ducks were more or less the same in that mean arterial blood pressure fell by 14–21% in both and heart rate rose by 7–56%. It was interesting that the greatest decline in arterial blood pressure occurred in chickens (mean 21%), while the heart rate in these animals rose by over 50%.

Changes in respiratory motor burst rate in response to asphyxia (transient hypercapnic hypoxia). Continuous recordings were made of intercostal nerve activity during periods of asphyxia (no ventilation) lasting for 3 min with ducks and 2 min with chickens. Before the start of the asphyxia there was no significant difference in Pa_{O_2} and Pa_{CO_2} between the chickens and ducks and their RMBRs were very similar (Fig. 4). There was no significant difference between Pa_{O_2} and Pa_{CO_2} after 90 s asphyxia in chickens and 150 s asphyxia in ducks, but at these time periods the RMBR of chickens had increased significantly, whereas in ducks it had decreased significantly from their respective preasphyxic values (Fig. 4). In all ducks examined the RMBR fell during the asphyxic period (Figs. 4, 5*A*) and frequently the bursts stopped altogether. Furthermore, the rate at the time of blood sampling during asphyxia (Fig. 4) was not significantly different from the rate at the same level of hypercapnia with Pa_{O_2} held at control levels (Fig. 2*A*), which tends to confirm the ineffectiveness of hypoxia as a

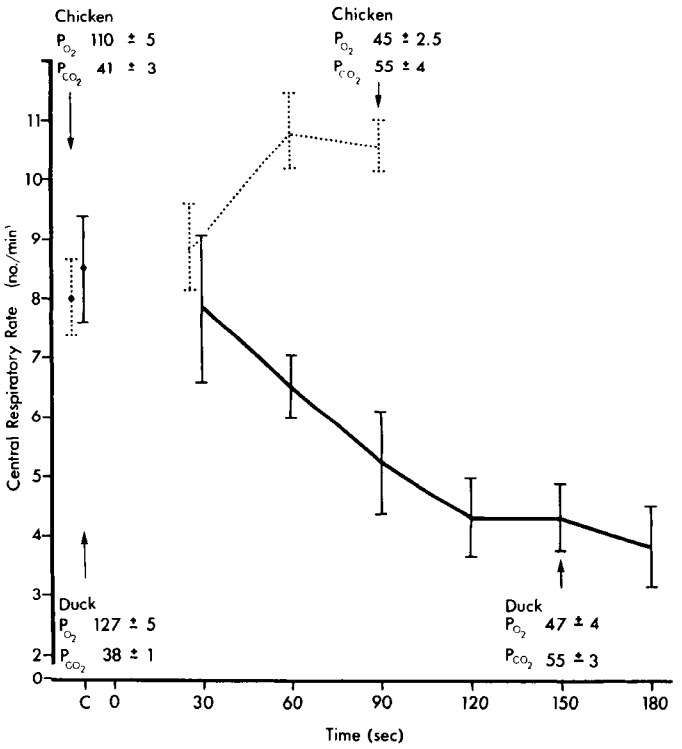

FIG. 4. Respiratory motor burst rate during asphyxia in paralyzed ducks and chickens. Values for central respiratory rate are derived from interval between respiratory bursts during successive periods of 30 s. Values of Pa_{O_2} (mmHg) and Pa_{CO_2} (mmHg) recorded in control situation and at arrow during period of asphyxia are also shown. C represents values in control situation and period of asphyxia commenced at *time 0*. All figures shown are mean ±SE.

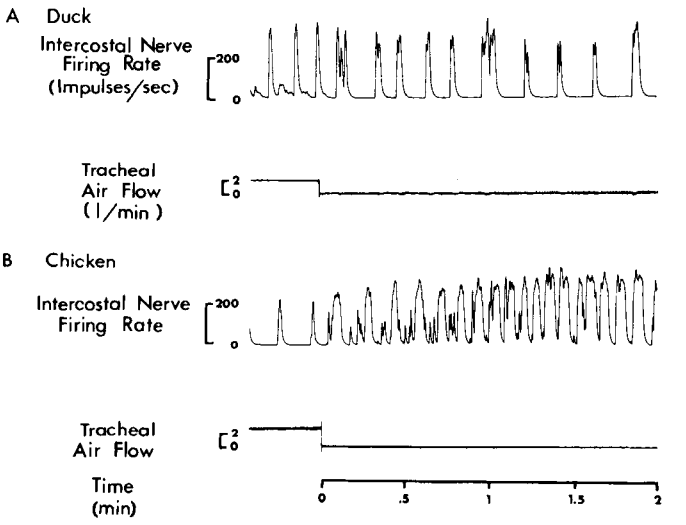

FIG. 5. Respiratory motor activity in duck *(A)* and chicken *(B)* during first 2 min of asphyxia. In each case, upper trace shows impulse rate recorded from central end of a cut intercostal nerve and lower trace shows tracheal airflow (l/min).

stimulant of respiratory rate in this preparation. The RMBR of chickens was more variable than that of ducks, although on average it increased significantly from the preasphyxic level by 90 s after cessation of ventilation (Figs. 4, 5*B*) and in no case did the rate fall during asphyxia. In ducks the number of spikes in each burst (burst amplitude) was virtually unchanged throughout the asphyxic period (Table 1), as was the case with transient hypocapnia, and it seems reasonable to conclude that this also applied to the steady-state recordings. In other words, the lowered RMBR in hypoxic, and probably in normoxic, hypercapnia was not accompanied by any compensatory increase in burst amplitude. However, in chickens the number of spikes per burst increased to a maximum of 312 ± 77% of the burst amplitude in the preasphyxic period during the period of asphyxia from 61 to 90 s (Table 1).

During asphyxia heart rate fell in both chickens and ducks. The fall in chickens after 2 min was to 71% of the value obtained before asphyxia, whereas 3 min asphyxia in ducks provoked a fall in heart rate to 21% of the value during normoxic normocapnia with unidirectional ventilation. Butler and Taylor (6, 7) report considerably greater bradycardia in their paralyzed chickens and ducks after only 60 s asphyxia, although the changes the animals suffered in Pa_{O_2} and Pa_{CO_2} appear to have been as severe as those undergone by our animals after the longer asphyxic periods.

DISCUSSION

The response of the respiratory center to hypercapnia is well documented in the breathing bird (1, 2, 6, 7, 13, 20), but this appears to be the first open-loop study and the results obtained are strikingly different from those previously reported. Hypercapnia normally causes an increase in respiratory minute volume, but our ducks with paralyzed respiratory systems showed marked respiratory center inhibition at high-CO_2 levels. Hypoxia, which is also normally a respiratory stimulant, had no apparent effect on the respiratory center of paralyzed ducks or chickens. Thus the normal responses to inspired gases were lost entirely under these conditions and, in one case, that of the normoxic duck exposed to a range of CO_2 levels, a new response pattern was seen.

In ducks vagotomy abolished all responses to CO_2, suggesting that some vagal CO_2-sensitive receptor was involved in these responses. Such receptors are present in the respiratory system (4, 10–12, 16, 18, 19), the heart (9), and carotid bodies (3, 14), pulmonary receptors being inhibited by CO_2 and the other two classes stimulated. There is no conclusive evidence to indicate which of these groups of receptors is involved, but Fedde et al. (10) showed that selective lung denervation altered

TABLE 1. *Change in number of spikes in central respiratory bursts during asphyxic periods in ducks and chickens*

	Time From Start of Asphyxia, S	Change in Burst Size, %	No. of Bursts Analyzed
Duck	0–60	−7.5 ± 6	19
N = 10	61–120	−12 ± 1	15
	121–180	−19 ± 7	11
Chicken	0–30	+170 ± 67	17
N = 5	31–60	+240 ± 43	27
	61–90	+312 ± 77	26

Values are means ± SE. The mean change in number of spikes per burst during the indicated time period is expressed as a percentage of the average number of spikes in at least five bursts before the start of asphyxia. N, number of animals used.

CO_2 sensitivity in a unidirectionally ventilated but breathing bird, a finding which may implicate pulmonary receptors. In the paralyzed ducks of this study it was found that high levels of pulmonary CO_2, known to inhibit the activity of pulmonary receptors, resulted in respiratory central activity at the same slow rate as was observed following vagotomy. Thus lack of pulmonary afferent activity due to high CO_2 had the same effect as lack of any vagal afferent activity due to vagotomy, even though in the first case the high CO_2 undoubtedly caused intense stimulation of carotid body and ventricular CO_2-sensitive receptors. Such evidence as we have, therefore, indicates that pulmonary CO_2-sensitive receptors are involved in the respiratory central response of the paralyzed duck to CO_2. The lack of any apparent CO_2 sensitivity after vagotomy argues against the presence of a medullary CO_2-sensitive area such as has been demonstrated in mammals (17, 21). However, Peterson and Fedde (19) showed that the apnea produced in four species of bird by removal of CO_2 from the ventilating gas stream was still seen after vagotomy. Clearly a nonvagal CO_2-sensitive area exists and is used in respiratory regulation and, for some reason, was inactive in our preparation.

The abolition of normal CO_2 and O_2 responses in our preparations was probably due to lack of respiratory movements. It has been shown by Cohen (8) that in the cat hypercapnia, which normally stimulates respiration, has the reverse effect if feedback from the lungs to the respiratory center is abolished by vagotomy and spinal cord section. Under these conditions, although the cat was being ventilated passively, no rhythmic information reached the respiratory center from the lungs or thoracic cage, and this was apparently effective in modifying the normal responses. It is possible that a similar effect was present in our preparations, and it is interesting to speculate what role this has in the adaptations to voluntary apnea such as diving. If a lack of rhythmic pulmonary afferent information is itself sufficient to modify the sensitivity of the respiratory center to CO_2, then once apnea is induced the new pattern of sensitivity appears. Although the respiratory center is, in any case, inhibited at this time, the additional inhibition by high CO_2 would assist in the maintenance of prolonged apnea. Certainly this appears to happen in asphyxia where the activity of the duck respiratory center declined from the onset of asphyxia onward, and it may be that this mechanism is important in the prolongation of diving apnea. The effect would be to remove any pressure to breathe, despite an increasingly severe hypercapnia and hypoxia.

The fact that both ducks and chickens had their respiratory responses to hypercapnia profoundly altered after removal of respiratory feedback suggests that this is itself not an adaptation to diving. Kuntz and Miller (15) have shown the importance of rhythmic pulmonary afferent traffic in eupnea in the chicken, and it is reasonable that withdrawal of this traffic should disrupt normal respiratory responses. However, the extent of the disruption is surprising. If birds depend on pulmonary CO_2 oscillations for maintaining eupnea, their respiratory control systems must be constructed on different principles from those of mammals. The fact that withdrawal of rhythmic vagal afferent traffic eliminates the respiratory response of chicken to CO_2 cannot be of much functional significance in terms of endurance of apnea, since the chicken does not become apneic spontaneously and tolerates forced apnea poorly. However, the duck, which habitually becomes apneic, sometimes for long periods of time, appears to have improved on the respiratory insensitivity to CO_2 of the apneic chicken by developing a CO_2-mediated respiratory depression. The potential value of such a depression to a diving animal is obvious, and it would be of interest to examine diving vertebrates of other classes for similar mechanisms.

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REFERENCES

1. ANDERSEN, H. T., AND A. LØVØ. The effect of carbon dioxide on the respiration of avian divers (ducks). *Comp. Biochem. Physiol.* 12: 451-456, 1964.
2. BOUVEROT, P., N. HILL, AND Y. JAMMES. Ventilatory responses to CO_2 in intact and chronically chemodenervated Peking ducks. *Respirat. Physiol.* 22: 137-156, 1974.
3. BOUVEROT, P., AND L.-M. LEITNER. Arterial chemoreceptors in the domestic fowl. *Respirat. Physiol.* 15: 310-320, 1972.
4. BURGER, R. E. Pulmonary chemosensitivity in the domestic fowl. *Federation Proc.* 27: 328, 1968.
5. BURGER, R. E., AND F. W. LORENZ. Artificial respiration in birds by unidirectional air flow. *Poultry Sci.* 39: 236-237, 1960.
6. BUTLER, P. J., AND E. W. TAYLOR. The effect of hypercapnic hypoxia, accompanied by different levels of lung ventilation on heart rate in the duck. *Respirat. Physiol.* 19: 176-187, 1973.
7. BUTLER, P. J., AND E. W. TAYLOR. Responses of the respiratory and cardiovascular systems of chickens and pigeons to changes in Pa_{O_2} and Pa_{CO_2}. *Respirat. Physiol.* 21: 351-363, 1974.
8. COHEN, M. I. Respiratory periodicity in the paralyzed, vagotomized cat: hypocapnic polypnea. *Am. J. Physiol.* 206: 845-854, 1964.
9. ESTAVILLO, J., AND R. E. BURGER. Avian cardiac receptors: activity changes by blood pressure, carbon dioxide, and pH. *Am. J. Physiol.* 225: 1067-1071, 1973.
10. FEDDE, M. R., R. E. BURGER, AND R. L. KITCHELL. Localization of vagal afferents involved in the maintenance of normal avian respiration. *Poultry Sci.* 42: 1224-1236, 1963.
11. FEDDE, M. R., R. N., GATZ, H. SLAMA, AND P. SCHEID. Intrapulmonary CO_2 receptors in the duck. I. Stimulus specificity. *Respirat. Physiol.* 22: 99-114, 1974.
12. FEDDE, M. R., AND D. F. PETERSON. Intrapulmonary receptor response to changes in airway-gas composition in *Gallus domesticus*. *J. Physiol., London* 209: 609-625, 1970.
13. HIESTAND, W. A., AND W. C. RANDALL. Species differentiation in the respiration of birds following carbon dioxide administration and the location of inhibitory receptors in the upper respiratory tract. *J. Cellular Comp. Physiol.* 17: 333-340, 1941.
14. JONES, D. R., AND M. J. PURVES. The effect of carotid body denervation upon the respiratory response to hypoxia and hypercapnia in the duck. *J. Physiol., London* 211: 295-309, 1970.
15. KUNZ, A. L., AND D. A. MILLER. Pacing of avian respiration with CO_2 oscillation. *Respirat. Physiol.* 22: 167-177, 1974.

16. LEITNER, L.-M. Pulmonary mechanoreceptor fibres in the vagus of the domestic fowl. *Respirat. Physiol.* 16: 232–244, 1972.
17. MITCHELL, R. A. Respiratory chemosensitivity in the medulla oblongata. *J. Physiol., London* 202: 3P, 1969.
18. OSBORNE, J. L., AND R. E. BURGER. Intrapulmonary chemoreceptors in *Gallus domesticus*. *Respirat. Physiol.* 22: 77–85, 1974.
19. PETERSON, D. F., AND M. R. FEDDE. Avian intrapulmonary CO_2-sensitive receptors: a comparative study. *Comp. Biochem. Physiol.* 40: 425–430, 1971.
20. RAY, P. J., AND M. R. FEDDE. Responses to alterations in respiratory P_{O_2} and P_{CO_2} in the chicken. *Respirat. Physiol.* 6: 135–143, 1969.
21. TROUTH, C. O., H. H. LOESCHCKE, AND J. BERNDT. A superficial substrate on the ventral surface of the medulla oblongata influencing respiration. *Pflügers Arch.* 339: 135–152, 1973.