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THE AVIAN LUNG: IS THERE AN AERODYNAMIC EXPIRATORY VALVE?

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Summary

The unidirectional gas-flow pattern through the avian lung is thought to result from 'aerodynamic valves'; support for this hypothesis lies mainly in the failure to find any evidence for anatomical valves. During expiration, air flows from the caudal air sacs through the major exchange area of the lung, the paleopulmonic parabronchi, instead of bypassing the lungs *via* the intrapulmonary bronchus. We tested whether the effectiveness of this expiratory flow control mechanism depends on aerodynamic factors, especially convective inertial forces that depend on gas density and flow velocity. In pump-ventilated, anaesthetized geese, a bolus of tracer gas was introduced into both the right and left caudal thoracic air sacs during an end-inspiratory pause. During the first expiration, the rise of tracer levels within the caudal trachea was measured. Valve efficacy was positively correlated with the rate of expiratory gas flow, $\dot{V}_{AO}$ (range 8–200 ml s^{-1}). At flows assumed to occur during exercise in geese ($\dot{V}_{AO}$>100 ml s^{-1}), the expiratory valve efficacy was approximately 95 %; it was less effective at lower flows. Surprisingly, the density (ρ) of the background gas (ρ of He/O_2=0.43 g l^{-1}, Ar/O_2=1.72 g l^{-1} or SF_6/O_2=5.50 g l^{-1}) had no effect on expiratory valving. We suggest two possible mechanisms that might explain this unusual combination of flow dependence without density dependence. (1) If airway geometry changes occurred between experiments with different gases, flow in the vicinity of the expiratory valve may have varied independently from flow measured at the airway opening. (2) Alternatively, valving may depend on dynamic compression of the intrapulmonary bronchus, which could depend mainly on viscous resistance and thus on flow velocity but not gas density.

Key words: birds, respiration, airflow distribution, aerodynamic valving, avian lung, convective inertia, dynamic compression.

Introduction

In sharp contrast to most air-breathing vertebrates, gas flows through the major exchange area of the avian lung, the paleopulmonic parabronchi (Fig. 1), in the same direction during both inspiration and expiration (Schmidt-Nielsen *et al.* 1969; Bouverot and Dejours, 1971; Bretz and Schmidt-Nielsen, 1971, 1972; Scheid and Piiper, 1971). The absence of any structural organization, such as valve leaflets, capable of controlling airflow direction through the bird's lung (Dotterweich, 1936; King, 1966; Duncker, 1971; Jones *et al.* 1981) has led to speculation that 'aerodynamic valving' is the controlling mechanism for the observed gas-flow patterns (Hazelhoff, 1943; Scheid *et al.* 1972; Brackenbury, 1971; Kuethe, 1988).

Butler *et al.* (1988), on theoretical grounds, suggested that the convective inertial force of the gas stream (ρu^2, strictly, the momentum flux density, where ρ is gas density and u is velocity) was the aerodynamic mechanism responsible for rectifying flow through the avian lung during inspiration and expiration. They proposed that the flow of gas through a side branch of the bronchial tree was systematically less than that through the main tube because of the axially directed momentum of the gas stream in the main tube. Tests of this hypothesis involve establishing that valve effectiveness depends on ρu^2 (Fig. 2). Wang *et al.* (1988), using models of the avian bronchial system, demonstrated that convective inertial forces could indeed explain the valving seen during inspiration in birds, i.e. no or limited flow in the ventrobronchi during inspiration (see Fig. 1). This hypothesis has been confirmed experimentally in intact birds (Banzett *et al.* 1987).

Despite the progress in our understanding of the operation of the inspiratory valve, the mechanisms controlling expiratory valving have not been previously explored in living birds. Expiratory valving is defined here as the mechanism that acts to direct the gas exiting the caudal thoracic and abdominal air sacs to flow through the dorsobronchi and then the parabronchi with no or little flow through the intrapulmonary portion of the primary bronchus (see Fig. 1).

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Physical and mathematical modelling of the inspiratory valve is relatively straightforward because the geometry of the bronchial features associated with the inspiratory valve is simple (Butler *et al.* 1988; Wang *et al.* 1992). Butler *et al.* (1988) recognized that the complex bronchial geometry associated with the expiratory valve prevented straightforward theoretical predictions but, nonetheless, suggested two mechanisms that might be operative in expiratory valving. (1) An alignment between the axis of the laterobronchus emptying the caudal thoracic air sac and the dorsobronchi would permit jet-flow of the gas stream to enter the dorsobronchi directly as well as entraining the flow exiting the abdominal air sac (Fig. 1). (2) Expiratory flow exiting the intraclavicular and cranial thoracic air sacs *via* the ventrobronchi must make a change in direction of more than 90 ° as it enters the primary bronchus. This change in direction would be accompanied by a pressure gradient directed caudally within the intrapulmonary bronchus that would act to block any cranial flow of gas through the intrapulmonary bronchus. Both of these mechanisms are consistent with the hypothesis that there is an interplay between the pressure gradient and the convective inertial forces that determines the ultimate flow partitioning, but the speculations of Butler *et al.* (1988) remained untested.

We therefore conducted a series of experiments to determine whether expiratory valve effectiveness scales with ρu^2 (the momentum flux density of the gas). We estimated valve failure by the appearance of a tracer gas in the trachea during the first expiration following injection of the tracer into the caudal thoracic air sacs. To examine the contribution of convective momentum to valve efficacy, we systematically varied both gas density and velocity in mechanically ventilated geese. The results revealed that varying gas flow ($\dot{V}_{AO}$) had a strong influence on expiratory valve efficacy but that changes in gas density had no systematic effect.

Materials and methods

Experimental rationale

During expiration, gas from the caudal sacs can either travel *via* the gas-exchange area of the parabronchi or bypass the parabronchi *via* the intrapulmonary bronchus. We define expiratory valving efficacy as the fraction of gas exiting all the caudal air sacs that passes through the parabronchi. The expiratory valve is then 100 % effective if all the gas exiting the caudal thoracic and abdominal air sacs flows through the parabronchi (Figs 1, 3A). If all gas exiting the caudal air sacs bypasses the lung, efficacy is zero. In these experiments, we estimated valve efficacy as the fraction of caudal thoracic air sac gas that flowed through the bird's parabronchial lung.

We mechanically ventilated geese at tidal volumes (V_T) chosen such that V_T was less than the combined volumes of the parabronchi, dorsobronchi, ventrobronchi and laterobronchus (lung volume 26 $cm^3\,kg^{-1}$; Maina, 1989) but much larger than the volume of the primary bronchus. During expiration, assuming 100 % valving, approximately half of the flow comes directly from the cranial group of air sacs *via* the ventrobronchi and half comes from the lung. Thus, using a tidal volume of 100 ml results in 50 ml of air coming from both lungs, less if valving is incomplete. We injected an inert tracer gas (N_2) bilaterally into the caudal thoracic air sac (Fig. 3) early in a end-inspiratory pause and then measured the rise in concentration of tracer in the caudal portion of the trachea during the following expiration (Fig. 3). We reasoned that any

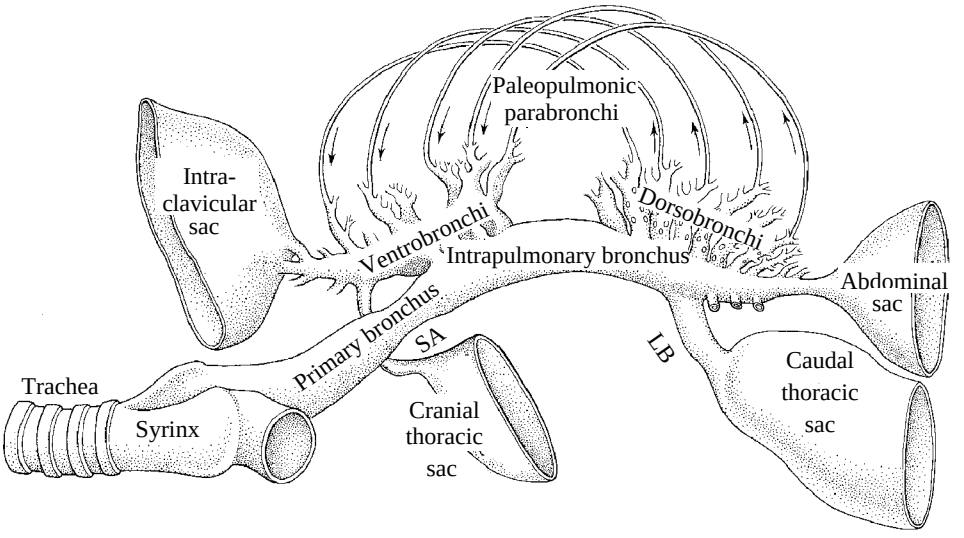

Fig. 1. General anatomy of the avian respiratory tract, right side only. The several air sacs filling the bird's coelom act as bellows to move gas through the lung. The unidirectional pattern of flow (arrows) through the major exchange area of the bird's lung, the paleopulmonic parabronchi, has been determined from flow-measuring devices implanted in the dorsobronchi (bronchi mediodorsales). During expiration, there is no or little flow in the intrapulmonary portion of the primary bronchus (bronchus primarius, pars intrapulmonalis); instead, nearly all the gas exiting the caudal thoracic and abdominal air sacs passes through the parabronchi. The intrapulmonary bronchus is commonly referred to as the mesobronchus. During inspiration, there is no or little flow in the ventrobronchi (bronchi medioventrales) connecting the intrapulmonary bronchus and parabronchi; instead, nearly all the gas passes caudally through the intrapulmonary bronchus. The laterobronchus (LB) (bronchi lateroventrales) connects the caudal thoracic air sac to the intrapulmonary bronchus. The neopulmonic parabronchi, a small portion of the lung (not shown, that varies between species from 0 to <25 % of the lung volume), consist of parabronchi connected between the laterobronchus and ventrobronchi that conduct air bidirectionally. The segmentum accelerans (SA) is a narrowing of the intrapulmonary bronchus cranial to the ventrobronchial orifices. Modified from Wang *et al.* (1992); reprinted by permission of John Wiley and Sons Inc.

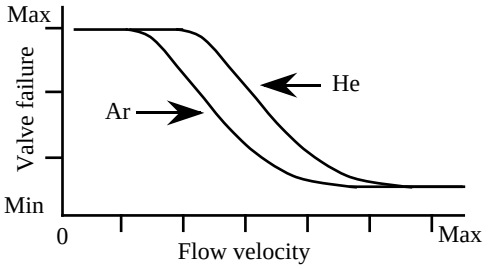

Fig. 2. Predicted theoretical relationships between valve failure, flow velocity and gas density; arbitrary units. If the mechanism responsible for expiratory valving in the bird is produced through the forces of convective inertia, i.e. ρu^2, we expect that changes in either gas density (ρ) or velocity (u) will have an effect upon valve efficacy. There may be high- (e.g. exercise) or low- (e.g. rest) flow regimes in which valve efficacy is unaffected by changes in gas density. Across a mid-range of flow velocities, valve efficacy should scale with ρu^2. Within these mid-range values of velocity, a low-density gas (He, ρ=0.18 g l^{-1}) will show a larger degree of valve failure for a given flow velocity than a high-density gas (Ar, ρ=1.79 g l^{-1}).

tracer sampled in the first expiration had exited the caudal thoracic air sac and bypassed the lung *via* the intrapulmonary bronchus (Figs 1, 3).

Animals

Nine domestic geese (*Anser anser*) of both sexes, purchased from commercial sources, were used in these experiments (mass range 3.25–3.75 kg). These experiments were approved by the Harvard Medical Area Standing Committee on Animal Use.

Animal preparation

The experimental arrangement is shown in Fig. 3. Unfasted birds were sedated with sodium pentobarbital (25 mg kg^{-1}) *via* deep muscular injection. The subcutaneous tissues over the distal brachium were infiltrated with 1% lidocaine and the ulnar vein was cannulated (PE 50) for continuous infusion of physiological saline, intermittent doses of sodium pentobarbital as needed to maintain light surgical anaesthesia, and atropine (1 mg h^{-1}). A cuffed endotracheal tube (6.5 mm) or thin-walled brass cannula of maximum allowable diameter was tightly sealed (ostomy cement, Turbot Co., USA) into the tracheal lumen in the mid-cervical region. The third most caudal rib was identified through a skin incision immediately cranial to the thigh. Flanged (2 cm) polypropylene (4 mm i.d.) tubes were inserted into both caudal thoracic air sacs through the intercostal musculature immediately cranial to the identified rib and about 3 cm dorsal to the articulation between the vertebral and costal components of the rib. The tubes were sealed to the body wall with sutures and cyanoacrylate glue (Superglue, Loctite Corp.). The skin and subcutaneous tissues surrounding the air sac catheters were sutured and sealed with cyanoacrylate glue to prevent leakage of air into or out of the air sacs.

The physiological status of the bird was continuously

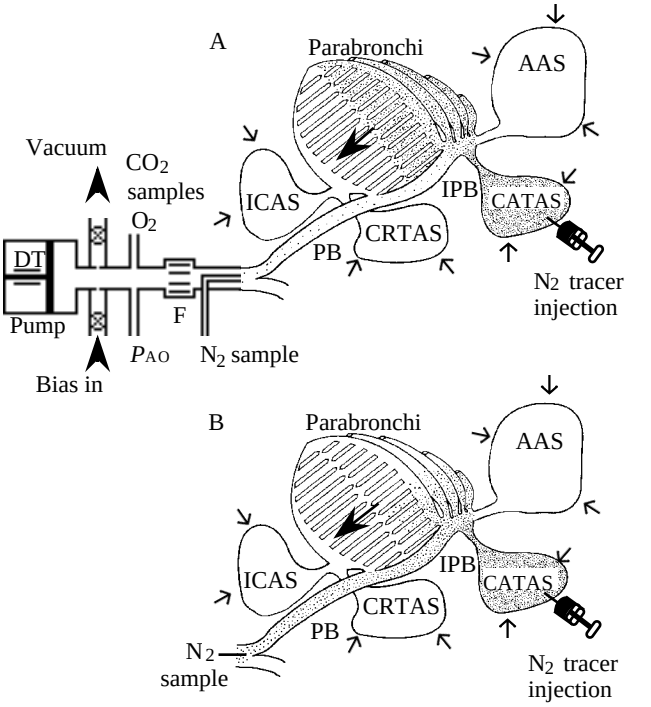

Fig. 3. Anatomy of the avian respiratory system showing the experimental arrangement and assumptions, for further details see test. Large arrow denotes unidirectional gas flow through the paleopulmonic parabronchi; small arrows denote the reduction in volume of the several air sacs during expiration. The tracer gas was injected bilaterally into the caudal thoracic air sacs (CATAS) and measured in the caudal end of the trachea. CRTAS, cranial thoracic air sac; ICAS, intraclavicular air sac; AAS, abdominal air sac; IPB, intrapulmonary bronchus; PB, primary bronchus; DT, displacement transducer of pump ventilator; F, pneumotachometer. (A) Predicted pattern for a highly effective expiratory valve. Little to no tracer (N_2) will be detected in the caudal trachea during the expiration immediately following the tracer-injection as the gas being sampled was resident in the ventrobronchi, parabronchi, primary bronchus (cranial to the ventrobronchi) and cranial air sacs (intraclavicular and cranial thoracic) during the end-inspiratory pause when tracer was added to the caudal thoracic air sac. (B) Predicted pattern for an ineffective expiratory valve. In this case, the gas exiting the caudal sacs will be equally divided between flow through the parabronchi and flow through the intrapulmonary bronchus; thus, the concentration of tracer (N_2) of the gas sampled in the caudal trachea will represent a mixture of tracer-free gas from the lung, cranial air sacs and abdominal air sac plus tracer-laden gas from the caudal thoracic air sac. Between these two extremes, a range of expiratory valving effectiveness exists. Modified from Scheid and Piiper (1987); reprinted by permission of CRC Press, Boca Raton, Florida.

monitored during the experiment *via* end-expiratory CO_2 level (LB-2, Beckman Instruments; or Datex Cardiocap, model CG-265), reflexes (pedal, corneal) and deep body temperature using a thermistor (Yellow Springs Instruments, model 45TA) inserted into the rectum through the cloaca. The position of all catheters was confirmed by necropsy following the experiment, when the bird was killed with sodium pentobarbital.

Following surgical preparation, the bird was supported

throughout the experiment in sternal recumbency in a cloth sling with holes through which its legs hung free. The sling provided even support across the ventral thorax from the cranial to caudal extremes of the sternum, a condition which mimics the weight of the bird suspended upon its pectoral muscles during flight (Zimmer, 1935).

Mechanical ventilation

A piston pump driven by a linear magnetic motor was attached to the end of the tracheal cannula and the bird was artificially ventilated throughout the experiment. Tidal volume (V_T) was controlled by the magnitude (cm) of piston travel, and the rate of piston travel ($cm\,s^{-1}$) was used to control flow rate at the airway opening ($\dot{V}_{AO}$). A high-impedance bias flow ($4.5–9\,l\,min^{-1}$) of gas mixtures of known composition (see below) was supplied at the airway opening and an equal flow was removed from the manifold at the airway opening *via* a high-impedance vacuum source (Fig. 3). Tidal volume was held constant throughout each experiment and flow rates were adjusted by changing the time of expiratory and inspiratory flow, i.e. rate of piston travel (see Fig. 5). Inspiratory and expiratory pause times could also be adjusted. Airway opening pressure (P_{AO}), referenced to atmospheric pressure (Validyne MP45), and flow (Fleisch pneumotachometer and Validyne MP45) were continuously monitored and recorded. To prevent N_2 contamination from room air of the gas volume within the bird, P_{AO} was maintained at $>0\,cmH_2O$ ($1\,cmH_2O=98.1\,Pa$) at all times (see Fig. 5). As expected, P_{AO} fluctuated during each pump-controlled breath; however, mean airway pressure during experimental breaths was held constant for each bird at $5–12\,cmH_2O$ (0.5–1.2 kPa). The amplitude of the pressure swing varied with $\dot{V}_{AO}$. For each trial, the bias flow of the gas through the manifold at the airway opening was adjusted (using gas source or vacuum, or both) to maintain a condition of zero net flow through the pneumotachometer during the pause periods.

During the trials, the birds were ventilated with commercially prepared mixtures of 20 % O_2 with a balance of He, Ar or SF_6. The He/O_2 and Ar/O_2 mixtures were commercially prepared. The gases SF_6 and O_2 were mixed just prior to entering the manifold at the airway opening (oxygen blender, model 960, Siemens Medical Systems). The density (ρ) of these gas mixtures is as follows: He/O_2 $0.43\,g\,l^{-1}$; Ar/O_2 $1.72\,g\,l^{-1}$; and SF_6/O_2 $5.50\,g\,l^{-1}$. The oxygen concentration of this mixture was continuously monitored within the manifold (Datex Cardiocap). 30 min of pump ventilation on a new gas mixture prior to any tracer injections was considered adequate for complete wash-in of that gas to occur.

Except during the tracer injections and the following expiration (see below), we ventilated the birds so as to maintain an end-tidal P_{CO_2} of approximately 40 mmHg (5.3 kPa), which rendered the

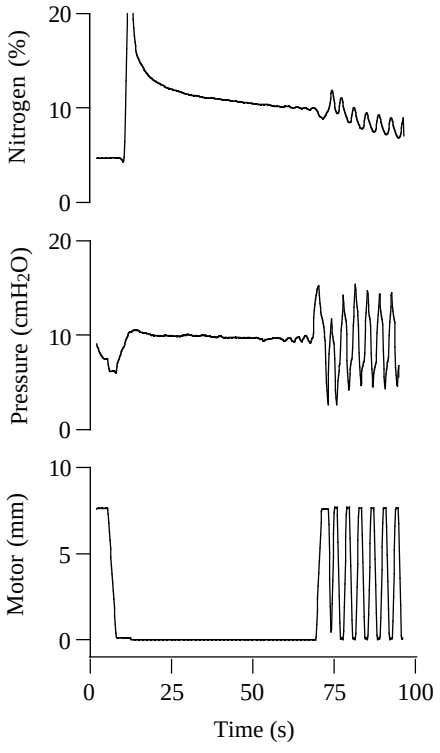

Fig. 4. Recording of dilution and washout of N_2 following injection of room air into one caudal thoracic air sac, see text for details. The pump ventilator (bottom graph) was stopped with bias flow of gas adjusted to maintain a constant P_{AO} (middle graph; $1\,cmH_2O=98.1\,Pa$) and thus no flow at the mouth. The N_2 concentration (top graph) in the caudal thoracic air sac immediately following injection (adjusted for delay plus rise time of the Nitrolyzer) into the air sac directly adjacent to the sampling catheter represents the 80 % N_2 content of the injected room air. As the nitrogen injection equilibrates with the sac gas, $\%N_2$ falls. After an approximately 2 min pause, the ventilator was restarted and the washout of N_2 per breath was determined.

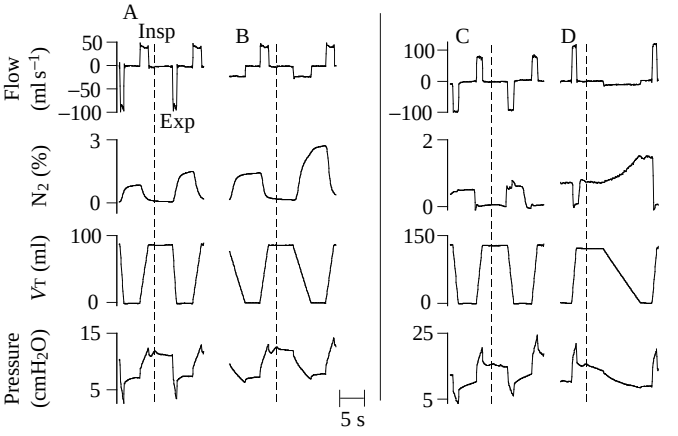

Fig. 5. Data recordings from two geese. The N_2 recording has been shifted by the measured delay plus rise time of the Nitralyzer to coordinate the timing of the nitrogen signal with that of the other data recordings. The broken lines indicate the timing of tracer (N_2) injection. Flow ($\dot{V}_{AO}$) is measured at the tracheal cannula. (A) Good expiratory valving and poor valving (B) in one goose (SK) while ventilated on 80 % He, 20 % O_2. (C) Good expiratory valving and poor valving (D) in a second bird (DL) while ventilated on 80 % Ar, 20 % O_2. V_T, tidal volume; pressure is measured at the airway opening ($1\,cmH_2O=98.1\,Pa$); N_2 (%) is measured in the caudal trachea; Insp, inspiration; Exp, expiration.

birds apnoeic for periods of greater than 10 s when mechanical ventilation was stopped. CO_2 and O_2 sampling within the manifold at the airway opening was momentarily discontinued during the tracer injection and measurement periods.

Experimental procedures

We sampled N_2 levels (Med Science, model 505 Nitralyzer) within the caudal 5 cm of the trachea immediately cranial to the syrinx (Fig. 3). To measure accurately the low concentrations of N_2 within the background gases used in these experiments, we adjusted the Nitralyzer's sample chamber pressure for stable, maximum strength signals while sampling low concentrations of N_2 in the selected background gas (see Banzett *et al.* 1987). Sample catheter (PE 60) flow rates (0.05–0.07 ml s^{-1}) were small relative to the lowest expiratory flow rates (8 ml s^{-1}) during these experiments. We calibrated the instrument and repeatedly rechecked calibrations throughout the experiment using gases of known N_2 content (0 % and 2 %) in the case of Ar/O_2 and He/O_2 mixtures and with serial dilutions (into a Douglas bag) of the SF_6/O_2 mixture using room air (80 % N_2) prepared immediately prior to experimental measurements. The Nitralyzer responded to a step change in N_2 levels at the distal end of the sampling catheter with a 1.5–11 s delay that depended upon the rate of flow through the sample chamber and sample catheter length. Sample catheter length and flow rate were kept constant throughout an experiment and the delay was repeatedly checked. The time constant of the roughly exponential response of the Nitralyzer was about 300 ms.

To introduce the N_2 tracer, we injected 20 ml of air simultaneously into both caudal thoracic air sacs (10 ml per sac) (Fig. 3). Injection time was marked by a pressure transducer connected to the tracer injection manifold. The tracer was injected at the beginning of an end-inspiratory pause of 6 s to allow for equilibration of tracer and air sac gas. We allowed a minimum of 5 min between successive injections of tracer.

Several criteria were used as a basis for rejecting a trial: (1) spontaneous respiratory efforts by the bird indicated by fluctuations in P_{AO} unrelated to pump movement; (2) non-zero net flow (imbalance of bias flows) measured by the pneumotachometer during the pause periods before or following a tracer injection; and (3) any change in the Nitralyzer calibration found during a subsequent determination.

The volume (from the dilution of the known volume of tracer) and ventilation rate (from the tracer wash-out curve) of the caudal thoracic air sacs were determined in three birds (Fig. 4). This information allowed us to estimate the percentage of the gas exiting the caudal thoracic air sac that had bypassed the lung *via* the intrapulmonary bronchus. 10 ml of air was injected into one caudal thoracic air sac while we continuously monitored N_2 concentration *via* a catheter inserted through the lumen of the injection tube. Caudal thoracic air sac volume in ml (V_{CAUD}) is:

$$V_{CAUD} = (C_{INJ} \times V_{INJ})/C_{CAUD}, \quad (1)$$

where C_{INJ} is the concentration (%) of N_2 injected, V_{INJ} is the volume (ml) of the injection and C_{CAUD} is the N_2 concentration (%) in the caudal thoracic air sac. When we sampled for 2 min, N_2 concentration continued to fall steadily, presumably because of diffusion out of the sac. We therefore estimated C_{CAUD} by extrapolating the slope between 40 and 70 s post-injection back to the time of the injection. V_T of the caudal thoracic air sac (V_{TCAUD}) (in ml) is:

$$V_{TCAUD} = \Delta\%N_2 \times V_{CAUD}, \quad (2)$$

where $\Delta\%N_2$ is the change in N_2 concentration per breath. The slope of $\%N_2$ following the reinitiation of pump ventilation (first five breaths only) was used to determine the $\Delta\%N_2$ per breath. The mean contribution of the two (left and right) caudal thoracic air sacs to V_T was 26 % (range 22–30 %), a roughly similar value to that reported by Scheid *et al.* (1974), i.e. 40 %.

Any increase in N_2 concentration that appears in the trachea on the first breath is the result of valve failure and can be used to estimate valving efficacy, as follows. First, because gas exiting the caudal sac must pass through either the parabronchi or the intrapulmonary bronchus:

$$\text{Valve efficacy (\%)} = \frac{V_{TCAUD} - V_{TCAUD,LEAK}}{V_{TCAUD}} \times 100, \quad (3)$$

where $V_{TCAUD,LEAK}$ is the volume of gas exiting the caudal thoracic air sac that bypasses the parabronchi *via* the intrapulmonary bronchus (in ml). Second, because N_2 tracer appearing in the trachea on the first expiration comes only from caudal thoracic air sac gas bypassing the lung; and is diluted by gas from all sources:

$$\Delta\%N_2 = (V_{TCAUD,LEAK} \times C_{CAUD})/V_T. \quad (4)$$

Combining the above equations:

$$\text{Valve efficacy} = 1 - \frac{\Delta\%N_2 \times V_T}{C_{CAUD} \times V_{TCAUD}}. \quad (5)$$

The highest commonly measured value of $\Delta\%N_2$, 1.0 %, represents 50 % efficacy; only when we were unable to maintain a bird's normal P_{CO_2} were higher values of $\Delta\%N_2$ measured. The lowest value commonly seen measured in the present experiments, 0.015 %, represents 95 % efficacy.

Data collection

All signals were continuously monitored and digitally stored (Superscope 2, G.W. Instruments, Somerville, Massachusetts, and Apple Macintosh IIc). Measured Nitralyzer delay times were subtracted from the $\%N_2$ data records to coordinate inputs of all instruments. Regressions were calculated and plotted using Excel.

Results

Measured end-expiratory $\%N_2$ never fell to zero even during prolonged (>2 h) ventilation with He/O_2 or Ar/O_2 during which no tracer was injected; however, it returned to its low, stable baseline value close to zero within 5 min following each tracer injection. We therefore subtracted pre-injection end-expiratory $\%N_2$ from the post-injection expiratory $\%N_2$ when estimating

expiratory valve effectiveness. We define $\Delta\%N_2$ as the peak $\%N_2$ measured in the caudal-most trachea during the expiratory pause immediately following the tracer injection minus the peak $\%N_2$ of the expiratory pause immediately prior to the injection (Fig. 5). Expired $\%N_2$ waveforms varied among birds (Fig. 5); we attribute this variation to the existence of multiple pathways of different lengths for the gas expired from the caudal thoracic air sac to reach the intrapulmonary bronchus (e.g. the laterobronchi and neopulmonic parabronchi; see Dunker, 1971).

Fig. 6 shows data from five birds. Expiratory valve failure, $\Delta\%N_2$ is strongly negatively correlated ($P<0.05$) with flow measured at the airway opening ($\dot{V}_{AO}$) in all geese. We combined all trials on all gas mixtures for each individual bird in the analysis as it was determined there was no apparent effect of gas density (mixture) on expiratory valving (see below). At flows in excess of $100\,ml\,s^{-1}$, valving efficacy reached a relatively constant value (about 95 %) that we term 'maximal effectiveness'. At flows less than $100\,ml\,s^{-1}$ valving efficacy progressively declined to about 50 %.

Surprisingly, changes in gas mixture, and density, had no systematic effect on expiratory valving effectiveness (Fig. 6). If convective momentum is an important factor in expiratory valving, data gathered during variations of flow and density should fall on the same line when $\Delta\%N_2$ (estimate of valve efficacy) is plotted against ρu^2. Because we did not measure flow velocity from the bronchi in the vicinity of the expiratory valve, we used values of $\dot{V}_{AO}$ (volume flow at the trachea) as a proxy for u (Fig. 7). When $\Delta\%N_2$ was plotted against $\rho(\dot{V}_{AO})^2$ a clear difference between gas mixtures was evident at flows $\leqslant 100\,ml\,s^{-1}$ (Fig. 7), and thus, the expected relationship did not occur.

During ventilation with 80 % SF_6, 20 % O_2 in one bird (individual JM), we encountered expiratory flow limitation at $75\,ml\,s^{-1}$. As a consequence, we were unable to maintain P_{CO_2} below 50 mmHg (6.67 kPa; see Fig. 6). We note that under this condition and at low flows, less than 20 % of the gas exiting the caudal thoracic air sac was passed through the lung. During ventilation with He/O_2 or Ar/O_2 mixtures, where the flow limitation occurred at a higher flow and a normal P_{CO_2} could be maintained, the relationship between flow rate and valving efficacy was qualitatively the same in individual JM as in the other birds (Fig. 6).

Critique of methods

These experiments produced apparently paradoxical results: a dependence of valve efficacy on gas velocity but not on gas density as predicted by a mechanism responsive to ρu^2. Can these results be explained by methodological limitations? The range of gas densities used, ρ=0.43–5.50 $g\,l^{-1}$, should have been adequate to produce measurable changes in valving. The experiments were often continued for 24 h; yet, during these rather lengthy periods, we found no consistent trend of valving performance with time. Variations in the order of use of the different gas mixtures eliminated any time-dependent factors.

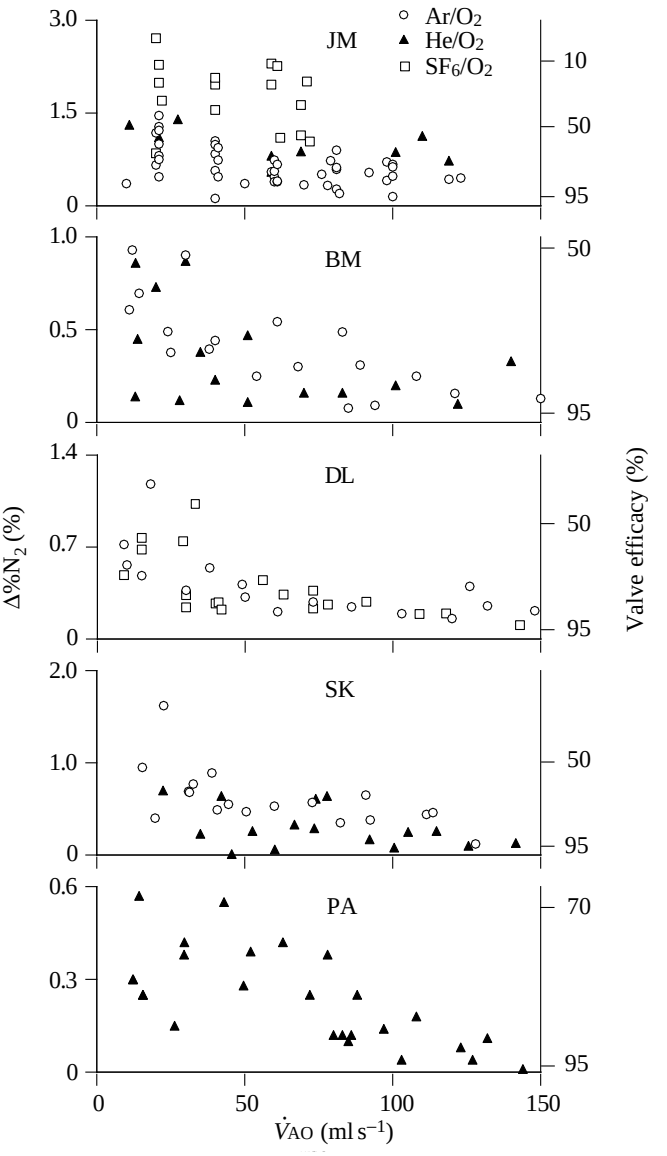

Fig. 6. Effects of changes in gas density and flow rate on $\Delta\%N_2$ measured in the caudal trachea. The magnitude of $\Delta\%N_2$ was used to estimate valve failure. The gas mixtures used were 20 % oxygen and 80 % Ar, He or SF_6. Expiratory valve efficacy (for calculation, see equations in text) was correlated with changes in gas flow rate $\dot{V}_{AO}$, i.e. reductions in flow resulted in increases in the percentage of the gas exiting the caudal thoracic air sac that bypassed the lung *via* the intrapulmonary bronchus. Although we examined flows ($\dot{V}_{AO}$) up to $200\,ml\,s^{-1}$, for clarity we have included here only those values of flow $\leqslant 150\,ml\,s^{-1}$. Above $\dot{V}_{AO}$ of $100\,ml\,s^{-1}$, the valving efficacy remained at a maximum value. Changes in gas density (ρ of He/O_2=0.43 $g\,l^{-1}$, Ar/O_2=1.72 $g\,l^{-1}$ or SF_6/O_2=5.50 $g\,l^{-1}$) had no apparent effect on expiratory valve efficacy. Each panel represents results from a different individual. Goose JM experienced a >10 mmHg (1.33 kPa) increase in end-expiratory P_{CO_2} while ventilated with 80 % SF_6, 20 % O_2, owing to a flow limitation that prevented adequate minute ventilation. The poor valving seen in this individual while on SF_6/O_2 may therefore have resulted from a physiological response to elevated levels of CO_2. These unexpected results indicate that less than 20 % of expiratory flow passed through the parabronchi in goose JM while being ventilated with SF_6/O_2.

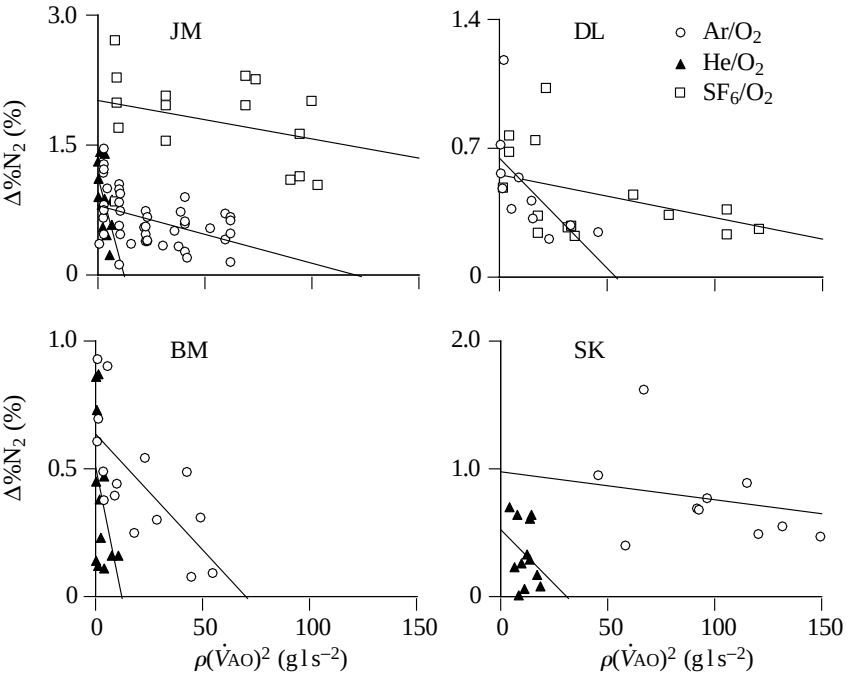

Fig. 7. Data shown in Fig. 6 replotted for four birds as $\Delta\%N_2$ *versus* $\rho(\dot{V}_{AO})^2$. If the expiratory valve was indeed controlled by convective inertial forces, replotting the valve failure estimate, $\Delta\%N_2$, against ρu^2 should eliminate any differences in the effects of gas density (ρ in $g\,l^{-1}$) on valve failure. Here we have used $\dot{V}_{AO}$ ($l\,s^{-1}$) as a proxy for u (velocity). Regression lines are given for valve failure estimates derived at flow rates of 0–100 $ml\,s^{-1}$ for each gas mixture. All birds showed maximum valving, independent of gas mixture, above flows of 100 $ml\,s^{-1}$ (see Fig. 6).

We are also unaware of any equipment limitations that would have led to our paradoxical results.

Are the estimates of efficacy accurate? During expiration, gas is expelled from both caudal thoracic and abdominal air sacs into the terminal portion of the intrapulmonary bronchus. The experimental technique of placing tracer gas in only the caudal thoracic air sac is therefore likely to underestimate expiratory valve efficacy. It is highly unlikely that gas exiting the abdominal air sac could detour the parabronchi *via* the intrapulmonary bronchus without entraining the flow of the gas exiting the caudal thoracic air sac (Fig. 1). Thus, we suggest that the percentage of the volume of gas exiting the abdominal sac that bypasses the lung will always be less than or equal to that of the gas exiting the caudal thoracic air sac.

Discussion

Efficacy of the expiratory valve

These experiments demonstrate that birds have an expiratory valving mechanism that is around 95% effective at flows greater than 100 $ml\,s^{-1}$ (Fig. 6). Such flows are less than those that the goose would encounter in flight (Berger *et al.* 1970). Powell *et al.* (1981) reported that, in ducks at rest, approximately 12% of the caudal sac gas bypassed the lung during expiration. Piiper *et al.* (1970) reported a bypass of approximately 24% in chickens and Bouverot and Dejours (1971) reported a 10% bypass. These previous studies examined spontaneously breathing, anaesthetized birds. The results reported here also suggest that expiratory valving would be less than 95% effective at the flows encountered during spontaneous breathing in anaesthetized birds, i.e. commonly less than 50 $ml\,s^{-1}$ (Fig. 6). Thus, there appears to be close agreement between previous studies and the results of this study at low flows; the present study also extends these observations, showing that valving becomes more effective at higher, more physiological flows. A common expiratory pattern in awake, resting birds, however, is two or more bursts of flow interspersed with periods of no flow (R. E. Brown, unpublished observations). Such a pattern may maintain effective expiratory valving even when expiratory duration is long.

Hypothesized mechanisms

The results reported here show a dependence of valve efficacy on gas flow rate ($\dot{V}_{AO}$) but at the same time show no effect of changes of gas mixture and density on valve efficacy (Figs 6, 7). Earlier experiments have indicated that valve efficacy depends on flow rate (Brackenbury, 1979; Hastings and Powell, 1986). Because valving was hypothesized to be governed by convective inertia, we predicted that expiratory valve efficacy would depend on both gas density and velocity and be a function of ρu^2 (Fig. 2). Our data are not consistent with this hypothesis (Fig. 6). Moreover, a fixed resistive mechanism, such as that suggested by Zeuthen (1942), would not be responsive to variations in flow rate and our data are also not consistent with this hypothesis. We propose two possible explanations for the present results, both of which invoke changes in airway calibre: (1) steady changes in bronchial diameter mediated by smooth muscle; (2) cyclic changes in bronchial diameter mediated by dynamic compression during expiration.

Steady changes in bronchial diameter mediated by smooth muscle

We do not know where the crucial point is, if normal function of the expiratory valve does indeed depend on ρu^2, to measure the relevant velocity. We have therefore used $\dot{V}_{AO}$ as an estimate of velocity, assuming that velocity is directly proportional to

flow; this will only be true if bronchial diameter is constant. It is possible that the geometry (diameter) or cross-sectional area of the bronchi in the vicinity of the expiratory valve (dorsobronchi, caudal intrapulmonary bronchi) changed between experiments with different gas mixtures and densities. It is also possible, but unlikely, that the various inert gases used (Ar, He, SF_6) could have tissue-specific effects, such as smooth muscle activation or stimulation of chemoreceptors with secondary activation of smooth muscle, resulting in a change of airway calibre. Alternatively, the airway diameter may have changed in response to local changes in P_{CO_2} or P_{O_2} consequent to altered distribution of ventilation with changes in gas density. Control of airway calibre by CO_2 has been described in the bronchi forming the inspiratory valve. A narrowed segment along the primary bronchus immediately cranial to the ventrobronchial openings, the segmentum accelerans (Fig. 1), convectively accelerates the inspired gas and thus enhances the efficacy of the inspiratory valve (Banzett *et al.* 1991; Wang *et al.* 1992). A coat of smooth muscle within the wall of the segmentum accelerans appears to modify bronchial diameter in response to changes in CO_2 levels (Wang *et al.* 1992). Thus, gas velocity in the vicinity of the inspiratory valve can be controlled independently from $\dot{V}_{AO}$.

Small changes in P_{CO_2} occurred during each trial (results not shown), the magnitude of which was related to the number of trial breaths (one to five and their lengthened pause periods) required to stabilize the bias flow for zero net flow during the pauses. Some of the variability in valving performance using a single gas mixture may have resulted from fluctuations of P_{CO_2} or P_{O_2}; however, there were no systematic changes in end-expiratory P_{CO_2} and P_{O_2} with the different gas mixtures. End-expiratory P_{CO_2} and P_{O_2} may not be an adequate measure in the bird: expired gas in birds comprises a variable mixture of gas from the parabronchi, gas exiting the cranial group of air sacs and any gas from the caudal group of air sacs that bypasses the parabronchi *via* the intrapulmonary bronchus. Further, because the cross-current gas exchange mechanism is sensitive to the flow velocity through the parabronchi, changes in arterial P_{CO_2} may not be reflected in end-expiratory P_{CO_2} (Scheid and Piiper, 1970). Changes in flow pattern caused by changes in gas density might, therefore, effect changes in airway calibre through changes in arterial blood gas levels or local pulmonary P_{CO_2} undetected in end-expiratory measurements. Between-trials ventilation was always (except for individual JM while ventilated with SF_6/O_2) performed at $\dot{V}_{AO}>125\,ml\,s^{-1}$ (above the minimum flow necessary for maximum valving). Thus, while it is reasonable to assume that overall flow patterns throughout the respiratory system remained constant except during low-velocity trial breaths, more subtle changes may have been undetected.

Cyclic changes in bronchial diameter mediated by dynamic compression

The experiments reported here show that at the lowest flow regimes there is an approximately equal distribution of flow, i.e. approximately half of the caudal air-sac gas bypasses the parabronchi (Fig. 6), consistent with the approximately equal resistive flow fractionation predicted by Zeuthen (1942). Yet no simple fixed resistive mechanism would yield a flow fractionation dependent upon on flow velocity (Figs 5, 6). This in turn implies that some variation in bronchial geometry is necessary to account for our findings. Below we consider the plausibility of the hypothesis that narrowing of the intrapulmonary bronchus during expiration caused by transmural pressures differences might effect valving. Changes in the calibre of the intrapulmonary bronchus during spontaneous breathing were observed visually a century ago by Soum (1896).

When the pressure surrounding a conducting bronchus (peribronchial pressure) is equal to the upstream driving pressure, resistive pressure losses produce negative transmural pressures that tend to narrow the tube, a phenomenon known as 'dynamic compression' (Wilson *et al.* 1986). This situation exists in the bird during expiration: the membranous medial wall of the intrapulmonary bronchus is exposed to air sac pressure over half of its diameter (Duncker, 1971). The difference in pressure between the air sacs and the bronchial lumen will tend to collapse the bronchus, and this pressure difference will increase as flow is increased. Depending on the magnitude of the pressure difference and the compliance of the bronchus, this could provide a mechanism for expiratory valving; because resistance in the intrapulmonary bronchus is thought to be dominated by viscous loss. The efficacy of such a 'dynamic compression valve' would depend on flow, but not on gas density (composition).

Morphometric measurements from Zeuthen (1942) and our own observations at low expiratory flows suggest roughly equal partitioning of flow between the intrapulmonary bronchus (IPB) and lung. This implies an equal resistance to flow *via* the two pathways. The 95 % valve efficacy we observed at high flow could result from an IPB:lung resistance distribution of 19:1. Because, during laminar flow, IPB resistance (R_{IPB}) varies as the inverse fourth power of the diameter (d), it follows that:

$$\frac{d_{IPB,high\ flow}{}^4}{d_{IPB,low\ flow}{}^4} = \frac{1}{19},$$

or

$$\frac{d_{high\ flow}}{d_{low\ flow}} \approx \frac{1}{2}.$$

The high valving efficacy observed at high flows could thus be achieved by halving the diameter of the intrapulmonary bronchus. The pressure drop from caudal thoracic air sac to cranial intrapulmonary bronchus has been determined to be $0.05\,cmH_2O$ (4.9 kPa) during artificial ventilation at low ($42\,ml\,s^{-1}$) expiratory flow velocities (Banzett *et al.* 1991). Although the *in situ* compliance of the intrapulmonary bronchus is unknown, its membranous wall suggests that dynamic compression is possible.

Dynamic compression, while a possible mechanism producing expiratory valving, is not possible during inspiration because upstream pressure is not coupled to peribronchial pressure. A highly compliant intrapulmonary bronchus during inspiration, where transmural pressures differences are

reversed, would have a relatively large diameter, thus reducing the downstream pressure loss and promoting an adequate channel for the inspiratory flow profiles.

Birds, although they have no anatomical valves in their respiratory system, have a highly effective expiratory valve that assists in maintaining the unidirectional flow of air through their lungs; the efficacy of this valve is sensitive to flow velocity but not to gas density.

In conclusion, the mechanism responsible for producing expiratory valving in the avian lung is unclear. We consider two possibilities that might explain our findings, both of which rely on changes in airway caliber. (1) Expiratory valving may be a mechanism governed by convective inertia. In these experiments, the effect of gas density on valving may have been masked by changes in airway caliber secondary to changes in gas composition and local or central variations in P_{CO_2} or P_{O_2}. If such changes occur, $\dot{V}_{AO}$ will not accurately reflect local velocity within the expiratory valve. (2) Dynamic compression of the intrapulmonary bronchus during expiration could produce an expiratory valve sensitive to flow velocity but not directly to gas density.

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