Respiratory Physiology & Neurobiology 161 (2008) 298–305

Contents lists available at ScienceDirect

Respiratory Physiology & Neurobiology

journal homepage: www.elsevier.com/locate/resphysiol

Tracheal compression delays alveolar collapse during deep diving in marine mammals

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ARTICLE INFO

Article history:
Accepted 7 March 2008

Keywords:
Breath-hold diving
Boyle's law
Diving physiology
Gas exchange
Lung collapse
Mathematical modeling
Pulmonary shunt
Seals

ABSTRACT

Marine mammals have very compliant alveoli and stiff upper airways; an adaptation that allows air to move from the alveoli into the upper airways, during breath-hold diving. Alveolar collapse is thought occur between 30 and 100 m and studies that have attempted to estimate gas exchange at depth have used the simplifying assumption that gas exchange ceases abruptly at the alveolar collapse depth. Here we develop a mathematical model that uses compliance values for the alveoli and upper airspaces, estimated from the literature, to predict volumes of the respiratory system at depth. Any compressibility of the upper airways decreases the volume to contain alveolar air yielding lung collapse pressures 2× that calculated assuming an incompressible upper airway. A simple relationship with alveolar volume was used to predict relative pulmonary shunt at depth. The results from our model agree with empirical data on gas absorption at depth as well as the degree of tracheal compression in forced and free diving mammals.
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1. Introduction

Marine mammals have stiffened upper airways that resist compression and very compliant rib cages (Leith, 1979). This anatomical adaptation is thought to result in a compression of the chest during diving which causes air from the compliant alveoli to move into the rigid upper airways (Scholander, 1940). As the alveoli collapse the rate of gas exchange decreases. Based on this adaptation, simple attempts at modelling gas exchange at depth have made two assumptions: (1) the lungs collapse at shallow predictable depths based on an idealized view of the respiratory system and (2) gas exchange ceases when all alveoli are collapsed (Fahlman et al., 2006; Falke et al., 1985; Ridgway and Howard, 1979; Stephenson, 2005). Lung collapse depth has been modelled using an idealized non-compressible trachea and infinitely compliant lung, similar to how a thin rubber balloon connected to a rigid pipe behaves under pressure (Denison and Kooyman, 1973; Stephenson, 2005). Consequently, alveolar collapse can be estimated from Boyle's law as $P_{coll} = (V_{A,o} + V_{D,o})P_{surface}V_{D,o}^{-1}$, where P_{coll} is the ambient pressure at which collapse occurs, $P_{surface}$ is the ambient pressure before the animal dives, $V_{A,o}$ is diving alveolar volume and $V_{D,o}$ the volume of the incompressible dead space. Using this equation, Scholander (1940) predicted that an animal could alter it's alveolar collapse depth by varying its initial diving lung volume, and thereby $V_{A,o}$. However, estimating collapse depth does not determine the effect lung compression has on gas exchange due to a pulmonary shunt. Even though experimental data in the harbour seal and California sea lion showed that the pulmonary shunt increased with depth (Kooyman and Sinnett, 1982), several studies have assumed that gas exchange is not influenced by the continually decreasing alveolar volume. Instead it has been assumed that termination of gas exchange occurs instantaneously at P_{coll} (Fahlman et al., 2006; Falke et al., 1985; Ridgway and Howard, 1979; Stephenson, 2005).

Ridgway and Howard (1979) measured intramuscular N_2 washout in trained bottlenose dolphins after a 1 h series of 23–25 repeated dives to 100 m (1100 kPa; 101.3 kPa = 1 ATA = 0 m depth). With the assumption of symmetrical gas exchange rates at the surface and at depth, the authors argued that the intramuscular N_2 levels measured at the end of the dive bout indicated that the lungs must have been fully collapsed at 810 kPa. Falke et al. (1985) found that the arterial N_2 tension (P_{N_2}) started to decrease at 405 kPa in Weddell seals diving freely to depth and it was suggested that this was the depth at which the seal's lungs collapsed. These collapse depths are at the shallow end of the range predicted by Scholander (1940).

As a marine mammal dives to depth the trachea is thought to act as an incompressible reservoir that receives air forced out of the alveoli. However, compression of the trachea has been measured by radiography during forced dives in both the Weddell and elephant seals (Kooyman et al., 1970). At 650 kPa, the diameter of the trachea decreased ~20% in both species. Qualitatively, tracheal compression would reduce the volume available for the alveolar air

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doi:10.1016/j.resp.2008.03.003

Nomenclature

C	permeability of the alveolar wall to gas
C_{Dif}	parameter that is equal to CSL^{-1}
$C_{Dif,TAC}$	C_{Dif} when V_A = TAC
D	diffusion rate
ΔP	partial pressure difference of gas between blood and alveoli
L	alveolar wall thickness
MAV	V_A when $P_{int} = P_{amb}$
P_{amb}	ambient pressure
$P_{A,S}$	structural pressure of alveoli
$P_{D,S}$	structural pressure of dead space
P_{coll}	P_{atm} at alveolar collapse
P_{int}	pressure inside the respiratory system
P_{N_2}	partial pressure of N_2
$P_{surface}$	pressure at the surface of the water
S	total alveolar surface area
TAC	total alveolar capacity
V_A	alveolar volume
$V_{A,o}$	diving alveolar volume
$V_{A,N}$	normalized alveolar volume (V_A TAC^{-1})
V_D	dead space volume
$V_{D,o}$	diving dead space volume
$V_{D,N}$	normalized dead space volume ($V_D V_{D,o}^{-1}$)

to move into. Therefore, tracheal compression would increase P_{coll} as the alveolar volume (V_A) would be greater at any a given ambient pressure, P_{amb}. Although the compressibility of the trachea is expected to alter lung collapse depth, there has been no in depth attempt to predict the magnitude of this effect.

Kooyman and Sinnett (1982) measured pulmonary shunts in harbour seals and California sea lions in a pressure chamber, in an attempt to define the degree of alveolar collapse with pressure. Pulmonary shunt is a measure of incomplete gas exchange with a pulmonary shunt of 100% equivalent to complete alveolar collapse. Kooyman and Sinnett (1982) found that pulmonary shunt increased with compression and predicted that alveolar collapse would occur at approximately 1700 kPa in both species, much deeper than suggested in freely diving Weddell seals (Falke et al., 1985) and bottlenose dolphins (Ridgway and Howard, 1979). Furthermore, the data presented by Kooyman and Sinnett (1982) suggested that the pulmonary shunt occurs gradually as theorized by Scholander (1940).

To improve our understanding of how a closed respiratory system compresses under pressure, we derived a mathematical model to predict alveolar volume at depth based on estimated compliances of the alveoli and trachea of marine mammals. Furthermore, we made a simple approximation to predict changes in relative pulmonary shunt as a function of depth. We suggest that the behavior of a marine mammal's respiratory system at depth is predictable with adequate knowledge of the physical properties of the upper and lower airways as well as the volume of air in the alveoli when an animal dives. Our results predict that alveoli remain open at a greater pressure than previously estimated, although, due to a decrease in V_A, the pulmonary shunt increases with depth.

2. Materials and methods

2.1. Model

The respiratory system of a diving mammal was modelled as a two-component system (Denison and Kooyman, 1973). One component is the upper airways, or dead space (trachea and bronchi) where no gas exchange occurs of volume V_D. The other component is the alveolar compartment of volume V_A. The respiratory system during diving is considered to be a closed system in which air is free to pass between V_A and V_D. Consequently, the pressure inside the respiratory system (P_{int}) is equal in both V_A and V_D. P_{int} is calculated after a change in the total volume of the respiratory system ($V_A + V_D$) using Boyle's law.

Upon compression, two forces in the alveoli and dead space balance P_{amb}, (1) the pressure exerted by the air inside the closed respiratory system being compressed (P_{int}) and (2) the pressure due to the structural properties of the alveoli and dead space ($P_{A,S}$ and $P_{D,S}$, respectively). The alveoli and dead space compress until a steady state is achieved where $P_{A,S}$ or $P_{D,S}$ and P_{int} balance P_{amb}. Therefore, in the alveoli

$$P_{Int} + P_{A,S} = P_{amb} \quad (1)$$

and in the dead space:

$$P_{Int} + P_{D,S} = P_{amb} \quad (2)$$

Since P_{int} is the same in the alveoli and dead space

$$P_{D,S} = P_{A,S} \quad (3)$$

showing that alveoli and dead space must compress and have equal structural contributions to pressure (for convenience referred to in the text as structural pressure). The structural pressure in either compartment can be re-arranged to give $P_{amb} - P_{int}$. Therefore, a positive structural pressure refers to a pressure that opposes P_{amb}, i.e., resists compression during diving. Given an estimate of $P_{D,S}$ and $P_{A,S}$, it is possible to predict V_A, V_D, and P_{int} in terms of P_{amb} based on measurable properties of a diving mammal's respiratory system.

2.2. Alveoli

The relationship between $P_{A,S}$ and V_A (P–V curve) was estimated for an 'average' marine mammal using experimental data for the harbour porpoise (Kooyman and Sinnett, 1979) and California sea lion (Denison et al., 1971). Low compliance alveoli were modelled after that of a human (Francis and Denison, 1999) and high compliance alveoli were modelled after a sei whale (Leith, 1979). The total alveolar capacity (TAC), the maximum volume of air that can be held in the alveoli, was assumed to occur when the pressure inside the lung was 2.94 kPa greater than ambient (Denison et al., 1971; Kooyman and Sinnett, 1979). Minimum air volume (MAV), the air left in the alveoli when $P_{A,S} = 0$, was estimated to be 45% of TAC for a human (Francis and Denison, 1999), 18% TAC for an 'average' marine mammal (Denison et al., 1971; Kooyman and Sinnett, 1979) and 6% of TAC for a whale (Leith, 1979). The P–V curve of the alveolar space in a marine mammal was assumed to be sigmoidal and modelled by an equation similar to that used by Venegas et al. (1998):

$$V_{A,N} = a(1 + e^{-c(-b-P_{A,S})})^{-1} \quad (4)$$

where a, b, c, are fitting parameters and $V_{A,N}$ the normalized alveolar volume (V_A TAC^{-1}). The parameter a corresponds to the change in volume between the lower and upper asymptotes of the sigmoid, b corresponds to the pressure of the inflection point of the sigmoid and c is an index of the range of pressures where most of the volume change occurs.

2.3. Upper airways

Marine mammals have a closed or semi-closed cartilaginous ring which offers more structural support than the horseshoe like cartilage of a terrestrial mammal. Despite these structural differences, Cozzi et al. (2005) found similar stress–strain curves when

the trachea of goat, pig, and striped dolphin were compressed between two plates. We, therefore, assumed that $P_{D,S}$ in a diving marine mammal was similar to that of a terrestrial mammal. As Cozzi et al. (2005) did not measure the relationship between pressure and volume during their compression tests, we modelled the dead space as a compressible tube using data for terrestrial animals and an equation adapted from Aljuri et al. (1999):

$$V_{D,N} = (1 - P_{D,S}(0.0981Kp)^{-1})^{-1/n} \quad (5)$$

where $V_{D,N}$ is the normalized dead space volume ($V_D V_{D,o}^{-1}$), $V_{D,o}$ is the maximum dead space volume and Kp and n fitting parameters that define the stiffness of the trachea. In adapting this equation from Aljuri et al. (1999) we have assumed that the trachea does not compress longitudinally, and therefore, tracheal volume is proportional to cross-sectional area.

2.4. Diffusion rate

The rate at which gases diffuse ($\dot{D}$, mol m^{-2} s^{-1}), from the alveoli into the blood is a complicated interplay between alveolar and capillary surface area (S, m^2), pressure difference of the gas between alveoli and blood (ΔP, mol m^{-3}), alveolar membrane thickness (L, m) and permeability to gases (C, m^{-2} s^{-1}) across the alveolar membrane. It is often estimated by Fick's equation as:

$$\dot{D} = CS\Delta PL^{-1} \quad (6)$$

Due to the inability to predict changes in all variables we define a new variable ($C_{Dif} = CSL^{-1}$) that accounts for the overall changes in $\dot{D}$ at different V_A's. We also assume that C_{Dif} can be estimated after a compression from TAC to a new volume V_A as follows:

$$C_{Dif} = C_{Dif,TAC}(V_A\,TAC^{-1})^f \quad (7)$$

where $C_{Dif,TAC}$ represents alveolar properties when the alveoli are fully inflated and f is an exponent that describes how C_{Dif} is proportional to V_A. Only the overall effect on $\dot{D}$ due to changes in V_A were estimated and we did not attempt to quantify the magnitude of $\dot{D}$. For comparison with previous modeling attempts where gas exchange abruptly ceases at P_{coll} we assume $\dot{D}$ is proportional to ΔP when $P_{amb} < P_{coll}$ and $\dot{D} = 0$ when $P_{amb} \geq P_{coll}$.

3. Results

3.1. $P_{D,S}$ and $P_{A,S}$

The parameters a, b, c (Eq. (4)) were fit to give the best estimates of the alveolar compliances defined above and were found to be 1.04, 0.20, 1.21 for a human V_A, 1.1, 1.23, 1.33 for an average compliant V_A and 1.1, 1.62, 1.76 for a V_A estimated to be as compliant as a whale's (Fig. 1). In the simple balloon/pipe model the lung was assumed infinitely compliant, and therefore, $P_{A,S} = 0$ at all lung volumes.

We estimated the medium compliant $P_{D,S}$ by fitting pressure against cross-sectional area for 3 dogs and 2 sheep reported in Aljuri et al. (1999) using Eq. (5). It was assumed that $V_{D,N} = 1$ when $P_{D,S} < 0$ and for $P_{D,S} > 0$ Eq. (5) was used. Kp and n were, respectively, −10.82, 1.01. To account for variation in tracheal properties we increased Kp and n by 2 SE's from the fitted curve for a stiff trachea ($Kp = -19.5$ and $n = 1.55$) or decreased Kp and n by 1 SE for a compliant trachea ($Kp = -6.44$ and $n = 0.74$). In the balloon/pipe model we assume the trachea was incompressible, and therefore, $V_{D,N} = 1$ at all times.

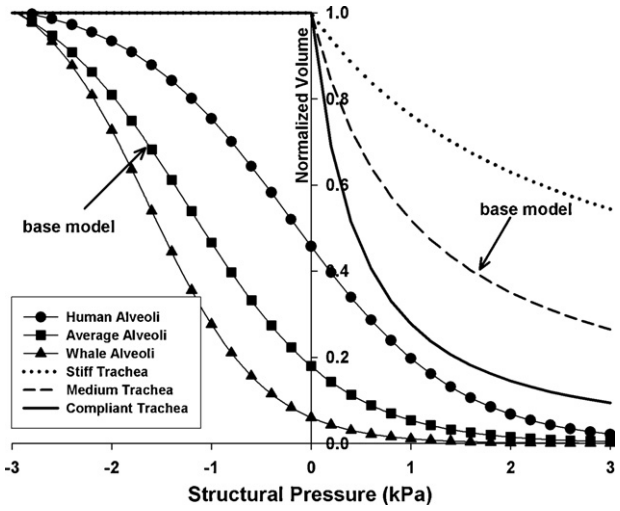

Fig. 1. Normalized volumes plotted against structural pressures for various alveolar and tracheal compliances. The average compliant alveoli and medium compliant trachea were used for the base model, and this model was used as a basis of comparison with all other simulations.

3.2. Relative volume of alveoli and dead space

The P–V curve loop of the alveolar space is $V_{A,N}$ TAC. Therefore, a change in TAC in this model is equivalent to a change in the structural properties of the alveoli (Eq. (4)). In addition, the output of our model is not as sensitive to the TAC:$V_{D,o}$ ratio as compared with the $V_{A,o}$:$V_{D,o}$ ratio (Fig. 2). Therefore, we use a constant TAC:$V_{D,o}$ = 10:1 in the current study. This is intermediate between the ratios reported for the Weddell seal diving on inspiration and expiration (Kooyman, 1973).

The model uses relative changes in volume since P_{int} is calculated based on Boyle's law. Therefore, our model predicts the same results as long as TAC:$V_{D,o}$ = 10:1 (i.e., the model is independent of absolute size). It is assumed that V_D is maximal at the surface, and therefore, we set $V_{D,o} = 1$. All ratios used in the simulations are defined relative to $V_{D,o}$. Consequently, the ratio TAC:$V_{D,o}$, therefore, defines the relative volume of the alveolar compartment as compared with the dead space compartment. In this model, TAC is used to define the alveolar P–V curve of the animal, and $V_{A,o}$ is the location on this curve. For example, if TAC:$V_{D,o}$ = 10:1, $V_{A,o} = 6V_{D,o}$ at

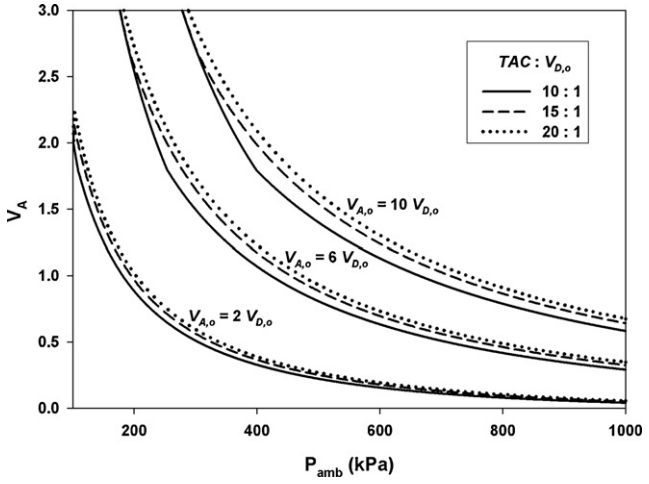

Fig. 2. Alveolar volume (V_A) plotted against ambient pressure (P_{amb}) for various ratios of $V_{A,o}$:$V_{D,o}$ and various ratios of TAC:$V_{D,o}$. The model is more sensitive to changes in $V_{A,o}$ than in TAC.

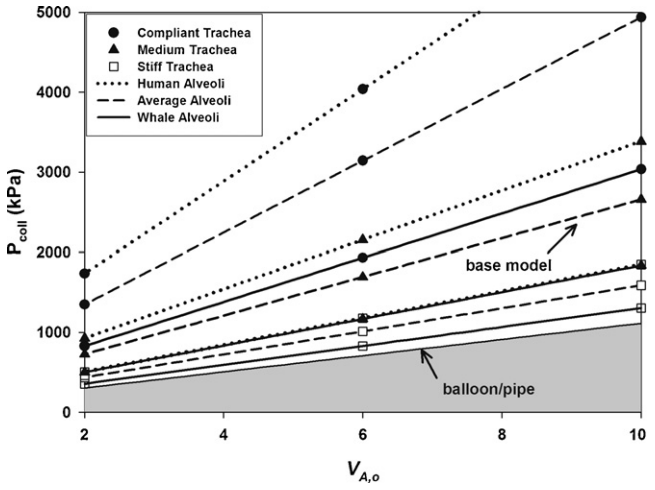

Fig. 3. Collapse pressure (P_{coll}) at various diving alveolar volumes ($V_{A,o}$) for all permutations of the structural pressure of alveoli and trachea. The balloon/pipe model represents an extreme case of our model.

the beginning of a dive represents an animal diving with 60% TAC remaining in its alveoli. This allowed us to explore the extent to which an animal can determine its lung collapse depth by diving on partial expiration.

3.3. Collapse pressure

The compliance of the alveoli and trachea, as well as $V_{A,o}$, affect P_{coll} (Fig. 3). We assumed that lung collapse occurred once $V_A \leq 0.01$TAC. For a dive on full inhalation ($V_{A,o}$ = TAC = $10V_{D,o}$), P_{coll} ranged from as low as 1303 kPa for an animal with a stiff trachea and a compliant alveolar compartment, e.g., sei whale, a situation approaching the balloon/pipe model, to as high as 6342 kPa for a human with a very compliant trachea. In the latter case, tracheal compression significantly reduces the available volume for the alveolar air. The balloon/pipe model, on the other hand, predicts P_{coll} = 1114 kPa. For an animal diving on partial exhalation with $V_{A,o} = 6V_{D,o}$, P_{coll} ranges between 831 and 4038 kPa, while the balloon/pipe model predicts P_{coll} = 709 kPa. For a medium compliant trachea and average alveoli, P_{coll} from our model is more than twice that predicted by the balloon/pipe model.

All subsequent simulations were run with a mid-compliant trachea and alveoli since these structural properties represent a reasonable estimate for a marine mammal.

3.4. Changes in V_A and V_D

Prediction of V_A at a given pressure for a mid-compliant trachea and alveoli closely matches the balloon/pipe model until MAV (Fig. 4a). When V_A is compressed below MAV, an increase in P_{amb} compresses both V_A and V_D. Tracheal volume, as measured by Kooyman et al. (1970), is plotted for comparison against our predicted tracheal volumes (Fig. 4b) and there is a close match between predicted and observed values for a $V_{A,o} = 6V_{D,o}$.

$P_{A,S}$ (i.e., $P_{amb} - P_{int}$) varies with P_{amb} and $V_{A,o}$ (Fig. 5). For low P_{amb}'s, when $V_A >$ MAV, we find $P_{int} > P_{amb}$. This is because alveoli naturally recoil to MAV, causing compression of air in the respiratory system. When $V_A <$ MAV, both the dead space and alveoli tend to expand outwards. In this case, some of the force that balances P_{amb} is taken up by the structural pressure and as a consequence $P_{int} < P_{amb}$. With decreasing $V_{A,o}$, the pressure inside the respiratory system equals P_{amb} at a shallower depth because V_A reaches MAV sooner.

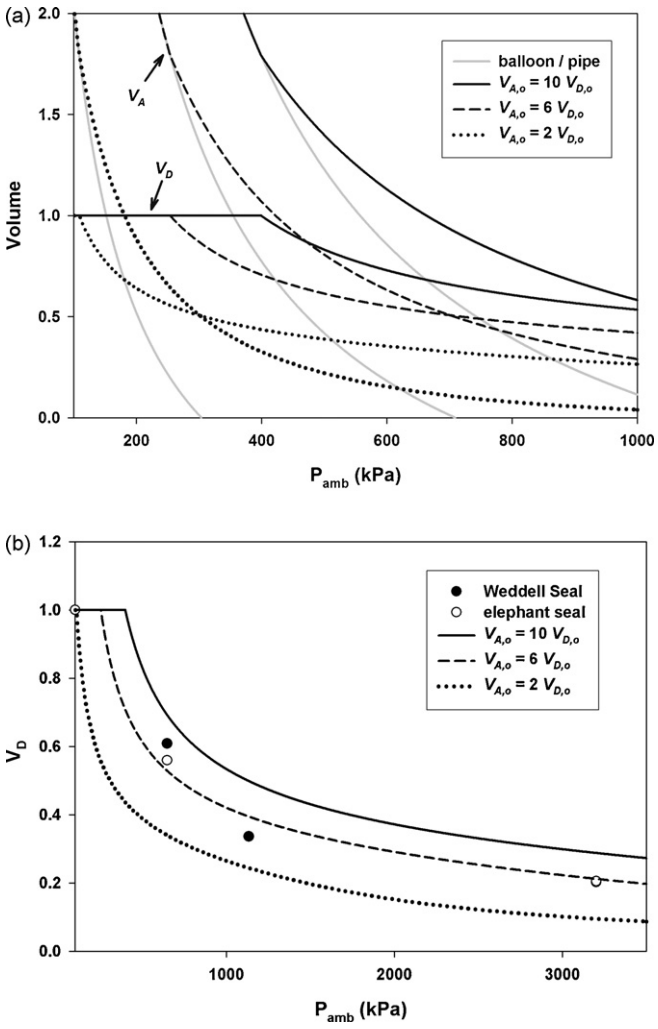

Fig. 4. (a) Alveolar (V_A) and dead space volume (V_D) against ambient pressure (P_{amb}) for various diving alveolar volumes ($V_{A,o}$:$V_{D,o}$). The balloon/pipe model is plotted for comparison. Our model predicts that the trachea starts to collapse once the alveolar volume reaches minimum air volume (MAV) and this causes V_A to become greater than predicted by the balloon/pipe model. (b) V_D plotted against ambient pressure (P_{amb}) for various diving alveolar volumes. Predicted tracheal volumes at depth as recorded by radiography in a pressure chamber are plotted for comparison (Kooyman et al., 1970). The experimental data show that the trachea of marine mammals is compressible.

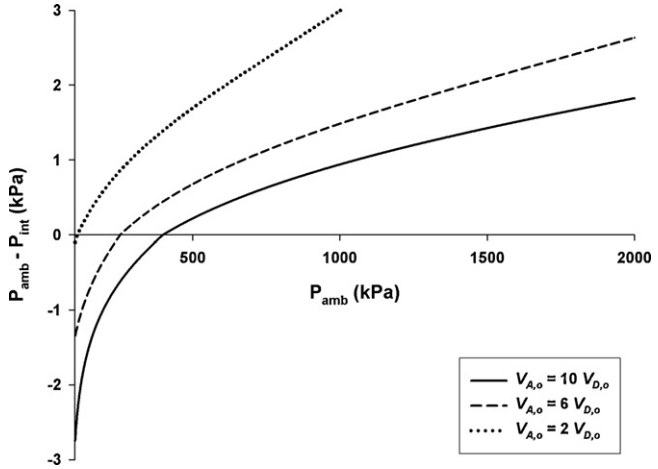

Fig. 5. Structural pressure due to compliance of the alveoli and dead space ($P_{amb} - P_{int}$) is plotted against ambient pressure (P_{amb}) for various diving alveolar volumes ($V_{A,o}$:$V_{D,o}$). When $P_{amb} = P_{int}$, V_A = MAV.

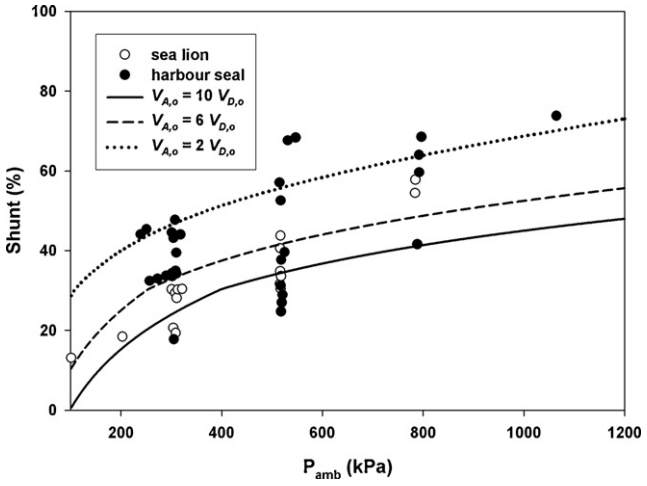

Fig. 6. Predicted and observed pulmonary shunt (%) vs. ambient pressure (P_{amb}). Observed pulmonary shunt data are from California sea lions and harbour seals (Kooyman and Sinnett, 1982).

3.5. Pulmonary shunt

Shunt fraction represents the volume of blood bypassing the gas exchange surface without exchanging gas. Kooyman and Sinnett (1982) published the only direct measurement of pulmonary shunt and $V_{A,o}$ at depth and this data was fitted to estimate diffusion rate at depth. Fig. 6 shows our model of diffusion that was fitted to Kooyman and Sinnet's (1982) experimental data for California sea lions diving with $V_{A,o} = 4.3V_{D,o}$ and harbour seals diving with $V_{A,o} = 3.3V_{D,o}$. Kooyman and Sinnet's shunt data for the two species were combined and converted to $1 - \text{shunt} = g\,(V_A\,\text{TAC}^{-1})^f$ where g is the proportionality coefficient. The exponent (Eq. (7)) was fitted to give $f = 0.21$ and this approximates the experimental data reasonably well from surface to $P_{amb} = 1000$ kPa (Fig. 6). At the surface there is a pulmonary shunt when $V_{A,o} < \text{TAC}$, because the surface area for gas exchange is less than when $V_{A,o} = \text{TAC}$.

4. Discussion

In 1940 Scholander proposed that marine mammals have stiffened upper airways that would stay open and receive air from highly compressible alveoli during diving. This movement of air was thought to collapse the alveoli and prevent gas exchange. However, this idealized system would tend to underestimate collapse depth if the trachea is compressible and the alveoli not infinitely compliant. This paper presents a mathematical model based on structural properties of the upper airways and alveoli that describes how a closed respiratory system behaves under pressure. Furthermore, we present a simple gradual shunt model and use it qualitatively to describe diffusion rate data from the literature. Our aim was to create a framework that could be used to describe how the closed respiratory system behaves while exposed to increased pressures during breath-hold diving. Using this model we have highlighted physiological variables that are important and that need further study to understand gas exchange during breath-hold diving.

4.1. The effect of $P_{A,S}$ and $P_{D,S}$ on P_{coll}

Very little work has been done on the respiratory mechanics of intact marine mammals. However, using *P–V* curves, Leith (1979) showed that the chest wall of a ribbon seal is very compliant and offers almost no structural support to the alveoli. Assuming that the chest wall does not resist compression, the *P–V* curve of an excised lung is a reasonable description of how the intact lower respiratory system will respond to changes in pressure. We, therefore, estimated $P_{A,S}$ in marine mammals using available *P–V* curves from excised lungs. As the *P–V* curves show similar features in both harbour porpoise (Kooyman and Sinnett, 1979) and California sea lion (Denison et al., 1971), we decided to combine the information for sea lion and harbour porpoise lungs to represent $P_{A,S}$ for a marine mammal with an alveolar compartment of average compliance.

To determine to what extent the structural properties of the upper and lower respiratory system have on P_{coll}, a sensitivity analysis was performed by varying $P_{A,S}$ and $P_{D,S}$ (Fig. 3). The average $P_{A,S}$ estimated from the data for a harbour porpoise and California sea lion (Kooyman and Sinnett, 1979; Denison et al., 1971) and the medium compliant $P_{D,S}$ estimated for 3 dogs and 2 sheep (Aljuri et al., 1999) were used as our base model to which all other variations were compared. A human *P–V* curve was digitized from Francis and Denison (1999) and fit with Eq. (4) to represent a stiff $P_{A,S}$ and we forced Eq. (4) to have a MAV occurring at 6% TAC (Leith, 1979) for a more compliant $P_{A,S}$. The compliance of the dead space was varied by adjusting the $P_{D,S}$ parameters for the base model as described above.

The sensitivity analysis suggests that both $P_{A,S}$ and $P_{D,S}$ are important in predicting how a closed respiratory system behaves under pressure. The results suggest that as alveoli become less compliant and the trachea becomes more compressible our prediction of depth for lung collapse varies further from the balloon/pipe model (Fig. 3). For the base model, $P_{coll} > 2$ times larger than that of the balloon/pipe model. This suggests that to further our understanding of how a marine mammal's respiratory system behaves under pressure future studies should be aimed at measuring the structural properties of the alveoli and dead space.

4.2. Collapse of the dead space

The structural properties of the dead space in a marine mammal are important in estimating P_{coll}. The more collapsible the dead space, the more the P_{coll} will vary from the idealized balloon/pipe model. As the compliant trachea compresses concurrently with the alveolar space, it reduces the volume for the alveolar air. Kooyman et al. (1970) measured compression of the trachea in both Weddell and elephant seals in a pressure chamber using radiography. At 650 kPa the diameter of the trachea decreased ~20% in both species. Obviously, the trachea must have experienced compression at depths shallower than 650 kPa. Upon further compression to 3200 kPa both species showed a decrease in diameter of 54% from surface values. Fig. 4b shows the model predicted and observed tracheal volumes. There is a close agreement between predicted and observed data for $V_{A,o} = 6V_{D,o}$. Kooyman et al. (1970) found that Weddell seals end voluntary dives with around 55% of their forced inspiratory lung volume. Therefore, by assuming that lungs and trachea collapse together when $V_A < \text{MAV}$, it is possible to estimate the experimental V_D's. This suggests that the alveoli need not be fully collapsed before tracheal collapse starts.

4.3. The effect of $V_{A,o}$ on P_{coll}

Another important variable in the model for predicting V_A and P_{coll} is the amount of gas in the marine mammal's respiratory system. In this model, the ratio between TAC and $V_{D,o}$ (TAC:$V_{D,o}$) as well as the ratio between $V_{A,o}$ and $V_{D,o}$ ($V_{A,o}$:$V_{D,o}$) affect V_A. While V_A was relatively insensitive to changes in the TAC:$V_{D,o}$ ratio, there were drastic changes as $V_{A,o}$:$V_{D,o}$ changed (Fig. 2). For example at a pressure of 500 kPa varying TAC:$V_{D,o}$ from 10:1 as in the base model to 20:1 causes an increase in V_A of 15% whereas changing $V_{A,o}$:$V_{D,o}$ from 6:1 to 10:1 is predicted to increase V_A by 73%. Consequently, a

diving animal has a behavioural ability to vary V_A by changing diving lung volume ($V_{A,o} + V_{D,o}$) while its inherent anatomy does little to affect V_A and thereby P_{coll}. To investigate the effect of changes in diving lung volume we kept the ratio constant as TAC:$V_{D,o}$ = 10:1 but varied $V_{A,o}$:$V_{D,o}$. Another property of the model is that V_A asymptotically approaches zero so that P_{coll}, and therefore, diffusion rate may be over estimated at high pressures.

4.4. Gas absorption at depth

We assume in this model that the total number of N_2,O_2 and CO_2 molecules in the lung does not change throughout the dive, i.e., we do not take into account absorption of gas into the body of the marine mammal. As O_2 and N_2 are taken up from the lung in exchange for CO_2, V_A and P_{coll} will be overestimated and the extent will depend on the descent rate and the length of the dive. However, accounting for changes in lung gas is a complicated problem that will require adding this lung volume model to a model of gas exchange at depth. Despite this shortcoming, our results clearly show that $V_{A,o}$:$V_{D,o}$ significantly affects V_A and P_{coll}. This is important as marine mammals can vary $V_{A,o}$ to adjust buoyancy, and therefore, $V_{A,o}$:$V_{D,o}$ and thereby P_{coll}. It is known that Elephant and Weddell seals commonly exhale before diving (Kooyman et al., 1970), thereby decreasing P_{coll}. Fur seals and sea lions on the other hand dive on inhalation (Kooyman, 1973; Fahlman and Hastie unpublished observation). Despite intra- and inter-species differences in TAC, varying $V_{A,o}$ will determine the extent of gas exchange at depth. Therefore, measuring $V_{A,o}$:$V_{D,o}$ in a range of species and during different dive behaviours (depth, duration, foraging, active swimming, gliding) may show how physiology shapes ecology. This ratio is also important in understanding how the respiratory system of a marine mammal behaves under pressure.

4.5. Trans-thoracic pressures

For the dead space to completely avoid compression as in the balloon/pipe model it must be able to withstand large trans-thoracic pressure gradients ($P_{amb} - P_{int}$). Brown and Butler (2000) analyzed the structural properties of the respiratory system in breath-hold diving mammals and predicted that a trans-thoracic difference >101 kPa would cause tissue failure. Following the logic that upper airways are incompressible they concluded that lung collapse is unavoidable during breath-hold dives. Our model shows that complete alveolar collapse can be prevented with a gradient of only 1–2 kPa if the dead space collapses along with the alveolar space (Fig. 5). Unfortunately, this trans-thoracic pressure gradient has not been measured.

Fig. 5 shows the trans-thoracic gradient that our model predicts will occur at depth. This figure is derived from estimated *P*–*V* curves and may be different than those of a living animal. However, this figure also represents measurable variables of the respiratory system throughout a dive. As gas management is paramount for efficient foraging in diving marine mammals, understanding how the lungs behave under pressure should receive high priority in future research. By experimentally measuring P_{int} and P_{amb} along with an estimation of $V_{A,o}$, it is possible to estimate the *P*–*V* curves of the trachea and lungs. For example, if the trans-thoracic pressure gradient sharply increased during a dive we would predict that the alveoli must have collapsed and the pressure gradient is a result of the upper airways resisting compression. Such an experiment would provide vital data of how the upper and lower respiratory system behaves under pressure and if and when alveoli collapse.

A recent paper by Hooker et al. (2005) found that Antarctic fur seals exhale continuously on ascent, starting at a depth that is 50–85% of the maximum dive depth. It is interesting to note that they do not exhale immediately on ascent and the reason for this can be explained by our model. The point where the trachea starts to collapse is the point where the pressure gradient between the outside and inside of the rib cage switches from negative to positive ($P_{amb} - P_{int} > 0$), i.e., the structure of the alveoli are starting to oppose P_{amb}. If the glottis was opened when there is this positive pressure gradient, sea water would enter the respiratory system since the alveoli and trachea would tend to expand. Conversely, if the glottis was opened when $P_{amb} - P_{int} < 0$ the respiratory system would tend to collapse and air would escape. If an animal has dived to a depth where V_A < MAV, the model predicts that $P_{int} < P_{amb}$ (Fig. 5). In this situation, water would tend to enter the respiratory system if the seal's glottis was open. The seal must, therefore, ascend until V_A > MAV without releasing any bubbles. After this point exhalation occurs passively because $P_{int} > P_{amb}$.

4.6. Lung compression and gas exchange

In this paper, we model the closed respiratory system of a diving mammal and predict that P_{coll} will be deeper than previously modelled. Consequently, we suggest that gas exchange should still be occurring deeper than the 1100 kPa predicted from a $V_{A,o}$:$V_{D,o}$ = 10:1 using the balloon/pipe model. Kooyman and Sinnett (1982) measured gas exchange in harbour seals and sea lions and their results conclusively show that alveolar collapse and termination of gas exchange happens much deeper than predicted in the balloon/pipe model. Their data from harbour seals and sea lions showed that the pulmonary shunt increased with compression and was estimated to be 100% at ~1700 kPa. This suggests that lung collapse does not occur, at least in harbour seals and California sea lions, until much deeper than previously predicted. Interestingly, this data also clearly shows a gradual collapse of the alveoli. Pulmonary shunt increased with depth in both species of marine mammals suggesting that the rate of gas exchange is gradually reduced as V_A decreases.

Alveolar surface area is vitally important in predicting gas exchange at depth (Eqs. (6) and (7)). We estimate that the alveolar properties that describe the rate of gas diffusing into the blood are proportional to V_A through an exponent f (Eq. (7)). Assuming everything is constant except surface area and in response to pressure alveoli are either open or closed, gas exchange would be expected to relate directly to volume so that f = 1. If lungs elastically compress, and remain spherical, the relationship should be to a 2/3 power and if they keep the same surface area but simply flatten out we would expect a power of 0. Since there has been no general consensus how the alveoli of marine mammals physically change under pressure we simply fitted pulmonary shunt data from Kooyman and Sinnett (1982). We found that f = 0.21 fits the data reasonably well (see Fig. 6). We used the 0.21 exponent to calculate a normalized diffusion rate ($\dot{D}$) at various P_{amb}'s. Our calculated $\dot{D}$ (Fig. 7) is an approximate value that may change in a complex way with pressure. However, our estimated value provides instructive information about changes in $\dot{D}$ with pressure that can be used in a qualitative comparison with available data. This exercise agrees with the idea of a gradual collapse as proposed by Scholander (1940) and shows that $\dot{D}$ initially increases to a maximum as pressure increases and then declines towards 0 as the alveoli collapse. By combining our model predicting V_A with the equation estimating $\dot{D}$, we can explain experimental data that has used blood and tissue N_2 tension as indirect evidence for shallow lung collapse.

Elephant and Weddell seals compressed in a pressure chamber showed no difference in arterial or venous P_{N_2} values between pressures of 405, 709 and 1480 kPa (Fig. 2 in Kooyman et al., 1972).

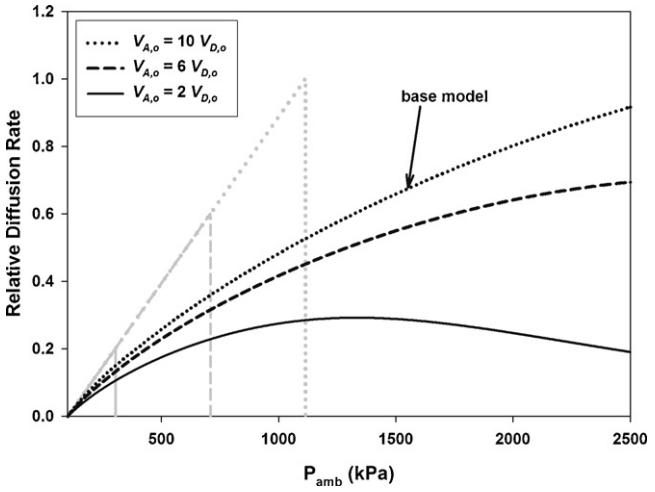

Fig. 7. Visualization of relative diffusion rate with depth plotted for the base model as well as a model with instantaneous termination of gas exchange at a depth predicted from the balloon/pipe model. The diffusion rate in the base model is estimated from experimental pulmonary shunt data and a number of caveats need to be applied as discussed in the text.

Arterial P_{N_2} should be an indicator of the diffusion rate of N_2 from the lungs into the blood. Consequently, with increasing P_{amb} the partial pressure gradient between the alveoli and blood increases and this increases $\dot{D}$ (Eq. (6)). A gradual alveolar collapse, in which decreasing gas exchange surface area compensates for increasing pressure gradient, is a simple explanation why arterial P_{N_2} is similar during compression to depths ranging from 405 to 1480 kPa. Fig. 7 shows that $\dot{D}$ could be similar at 405, 709, and 1480 kPa for low $V_{A,o}$ ($V_{A,o} = 2V_{D,o}$). This agrees with the suggestion that both elephant and Weddell seals dive on exhalation. If lung collapse had occurred arterial P_{N_2} would have been similar to mixed venous levels. As this was not the case, complete termination of gas exchange and alveolar collapse could not have occurred at a pressure less than 1480 kPa. In this same study harbour seals were dived in the compression chamber (see Fig. 4 in Kooyman et al., 1972). The data indicate that termination of gas exchange does not occur until well after 1480 kPa. In this case arterial P_{N_2} was higher at a depth of 1480 kPa compared with 405 kPa immediately after the start of compression. This shows that $\dot{D}$ was higher at the greater P_{amb} and again indicates that harbor seals may dive on inhalation, and therefore, have a higher $V_{A,o}$:$V_{D,o}$.

Ridgway and Howard (1979) suggest that N_2 washout curves measured in dolphins having dived repeatedly during 1 h to 1100 kPa indicated that gas exchange must have ceased at 810 kPa. Assuming that an animal dives at a constant rate of descent, the total N_2 absorbed during a dive is proportional to the area under the curve in Fig. 7 ($\dot{D}$ vs. P_{amb}). A certain amount of N_2 is absorbed before gas exchange terminates at 810 kPa. In the balloon/pipe model, this corresponds to $V_{A,o} = 6V_{D,o}$. Using our graded shunt model, the same area under the curve occurs at around 1100 kPa pressure. Thus, our model provides an alternative explanation for the data presented and shows that the washout rate of N_2 from the muscle is not unequivocal evidence for alveolar collapse at 810 kPa.

Falke et al. (1985) sampled arterial blood every 30 s during descent to depth in freely diving Weddell seals. The gradual decline in arterial P_{N_2}, which began at a depth of 405 kPa, was concluded to be evidence that the alveoli were collapsed and resulted from N_2 in the circulating blood being absorbed into tissues. Since this arterial blood was sampled from the aorta of the seal the blood P_{N_2} reflects what was happening at the lungs. The circulatory transit time is of the order of 2–3 min during a dive, as estimated from the delayed response in Weddell seal venous P_{N_2} 's during compression in a hyperbaric chamber (Kooyman et al., 1972). Hence, arterial P_{N_2} would make a considerable drop to near surface levels if gas exchange ceased within a few minutes of commencing the dive. Consequently, if the alveoli collapsed and gas exchange was prevented there would not be a gradual decrease in arterial P_{N_2} during the rest of the dive. Instead arterial P_{N_2} would be a reflection of mixed venous P_{N_2}, and therefore, a reflection of the overall blood P_{N_2} coming back from the body. Our model of a gradual shunt that develops as the animal descends deeper provides a plausible explanation. If we assume that arterial P_{N_2} is proportional to the estimated $\dot{D}$ in Fig. 7, our results suggest that $\dot{D}$ approaches a maximum well before $V_A = 0$. This suggests that maximum arterial P_{N_2} measured in the Weddell seal is at the point of maximum $\dot{D}$ and is not evidence for complete alveolar collapse.

Mathematical models are important tools that provide a framework for complex problems. However, these models are only as good as the data from which they are constructed and supported. The model presented in this paper is based on previous work that has measured pulmonary shunt (Kooyman and Sinnett, 1982) and tracheal compression (Kooyman et al., 1970). Our model agrees with data presented by Kooyman and Sinnett (1982) that complete alveolar collapse probably does not occur until a pressure > 1600 kPa. By considering a pressure-dependent compression of the respiratory system that results in a decreased alveolar–arterial diffusion rate and an increasing pulmonary shunt, our model also explains the discrepancy in estimated alveolar collapse pressure in the Weddell seal (400 kPa, Falke et al., 1985), the dolphin (800 kPa, Ridgway and Howard, 1979) and the harbour seal and California sea lion (>1600 kPa, Kooyman and Sinnett, 1982). This model has many limitations, but is the first comprehensive attempt to quantify how the physical properties of a marine mammal's respiratory system may be affected by pressure. It will be important for future studies that attempt to quantify gas exchange at depth.

Acknowledgements

We would like to thank Dr. Sascha Hooker, Dr. Gerry Kooyman, Dr. Anthony Farrell, Dr. Colin Brauner and James Kirkham for comments on the manuscript. B.B. and D.R.J. were supported by an NSERC Discovery Grant to DRJ. A.F. was supported by a grant from the Office of Naval Research (ONR award number N00014-07-1-1098).

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