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The absolute necessity of chest-wall collapse during diving in breath-hold diving mammals

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Abstract

It has been suggested that the chest-wall (rib-cage, diaphragm, abdominal wall) of a diving animal is nearly incollapsible, and at deep depths can sustain a considerable trans-thoracic pressure P_{TT} ($=P_{body\ surface} - P_{intra\text{-}thorax}$). In strong contrast, we argue on circulatory, blood pooling, biological material strength, and ventilatory grounds that this is incompatible with life. At a P_{TT} of more than a third of an atm, or that experienced at a depth of only 3.5 m if the interior of the thorax remains at $P_{sea\text{-}level}$, the heart is unable to perfuse tissues outside the thorax. To the extent that P_{TT} is substantially larger than at sea level, blood will pool in the thoracic vessels with potentially lethal consequences of vascular rupture. From estimates of supporting tissue thickness and geometry, a P_{TT} >1 atm would exceed the mechanical strength of known biological materials. Finally, a chest-wall sufficiently stiff to support a P_{TT} >1 atm is not compatible with breathing when the animal is at the surface.

Key words: diving, mammals, chest-wall, collapse, lung, mechanics, circulation.

Introduction

Van Nie (1987) suggests that during breath-hold diving in mammals, and specifically in cetaceans, the chest-wall surrounding the thorax containing the air-filled lungs would decrease only minimally in volume with increasing depth. Specifically, van Nie (1987) indicates that gas pressure within the thorax ($P_{intra\text{-}thoracic}$) of a diving cetacean would not rise above 1.9 atmospheres (atm) even to depths of 300 m, corresponding to 30 atm[1] of pressure applied to the body surface ($P_{body\ surface}$). In strong contrast, established fundamental principles of cardiovascular physiology, respiratory and soft-tissue mechanics imply that life is not possible when there is a large trans-thoracic pressure[2] difference P_{TT} ($=P_{body\ surface} - P_{intra\text{-}thoracic}$) compared with P_{TT} at sea level. Our arguments are divided into four main sections. We discuss the circulatory consequences of large P_{TT}s, the vascular consequences of intra-thoracic blood pooling, tissue rupture from stresses exceeding the known strength of biological materials, and the ventilatory consequences of a chest-wall sufficiently stiff to support large P_{TT}s. All of these consequences would be lethal.

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Discussion

The chest-wall and its collapsible surfaces

All body surfaces bounding the air-filled thorax are components of the chest-wall, composed primarily of the rib-cage (ribs and attached intercostal musculature) and the diaphragm. As the difference between $P_{body\ surface}$ and $P_{intra\text{-}thoracic}$ increases, the thoracic cavity will decrease in volume via the rib-cage pathway and/or the diaphragmatic pathway. In series with the diaphragmatic pathway is the abdominal contents and abdominal wall. To the extent that either pathway is compliant, trans-thoracic pressures will remain near zero as $P_{body\ surface}$ increases with increasing depth. By contrast, the support of a substantial trans-thoracic pressure difference demands that both pathways are correspondingly stiff.

Assuming the rib-cage is capable of supporting this substantial pressure jump, there are two possible modes by which the chest-wall could maintain a pressure difference of $P_{intra\text{-}thoracic} \ll P_{body\ surface}$ namely, a correspondingly large pressure difference:

[1]One atm $\approx$760 mmHg $\approx$10 m H_2O.

[2]We ignore trans-thoracic pressure differences of a few tens of cm of H_2O or less.

(1) across the diaphragm, or (2) across the abdominal wall. In the following sections we first define these modes more precisely and then discuss the physiological catastrophes that would accompany breath-hold diving if it were possible to maintain $P_{intra\text{-}thoracic} \ll P_{body\ surface}$ via either of these modes.

(1) $P_{intra\text{-}thoracic} \ll P_{abdominal} = P_{body\ surface}$ — In this case, chest-wall deformation secondary to an increasing $P_{body\ surface}$ is resisted by the rib-cage in compression and the diaphragm in tension. Further, the abdominal wall does not support a pressure drop and thus, $P_{abdominal}$ is essentially the same as $P_{body\ surface}$.

(2) $P_{intra\text{-}thoracic} = P_{abdominal} \ll P_{body\ surface}$ — In this case, chest-wall deformation secondary to an increasing $P_{body\ surface}$ is resisted by the rib-cage and the abdominal wall, both in compression, such that the thoracic and abdominal compartments are maintained at approximately the same pressure.

Heart-driven circulation

Common to both modes of chest wall support that we wish to show physiologically impossible are problems associated with heart-driven circulation. These arise from the following considerations. Note that the pressure within the pericardium (pressure applied to the heart's surface) closely approximates pleural pressure and is within a few cm H_20 of lung gas pressure (see Permutt and Wise, 1986). We denote all these nearly identical pressures as $P_{intra\text{-}thoracic}$. When a terrestrial animal is breathing, its intrathoracic gas is on average equilibrated with atmospheric pressure ($P_{body\ surface}$); thus, the pressure applied to the heart's surface is nearly identical to the pressure on the tissues lying outside the thorax. To circulate blood, the heart of a terrestrial mammal exposed to approximately the same pressure as are the tissues it perfuses needs only generate a pressure sufficient to overcome extrathoracic tissue pressure, vascular resistance, and gravity. The mammalian heart is capable of generating, on a sustained basis, a maximum systolic arterial pressure, $P_{body\ surface}$ to $P_{intra\text{-}arterial}$, of at most 3.5 m H_2O or 260 mmHg[3]. The pressure generated by the mammalian heart is independent of body mass and thus, while the heart's stroke volume is a function of body size the maximum systolic pressures found in a dog, elephant or giraffe will be nearly identical to those of small dolphins, large whales and pinnipeds (Torrance, 1998).

Circulation consequences to mode (1) of chest wall support: $P_{intra\text{-}thoracic} \ll P_{abdominal} = P_{body\ surface}$. If a trans-thoracic pressure difference ($P_{body\ surface} > P_{intra\text{-}thorax}$) developed during a dive and the heart were to continue to perfuse tissues outside of a 'pressure-protected' thorax it would have to generate a pressure approximately 100 mmHg[4] (1.31 m H_2O) greater than $P_{body\ surface} - P_{intra\text{-}thoracic}$. Assuming an animal was able to sustain $P_{body\ surface} - P_{thorax} = 1$ atm (equivalent to depth of 10 m), its heart would have to generate a pressure equal to a column of water 11.3 m high if any tissue or organ outside of its 'pressure protected' thorax were to be perfused. Important organs affected by this argument include the brain, eyes, mammary glands, abdominal viscera, and swimming muscles[5]. Such a cardiac performance is physiologically impossible and thus, $P_{body\ surface} - P_{thorax} > 0.3$ atm (let alone 30 atm; see van Nie, 1987) is not compatible with life because cardiac output would be zero.

Circulation consequences to mode (2) of chest wall support: $P_{intra\text{-}thoracic} = P_{abdominal} \ll P_{body\ surface}$. Moving the partition supporting the intra-thoracic pressure jump from the diaphragm to the abdominal wall does not make life more viable when $P_{body\ surface}$ is >3 m H_2O above $P_{intra\text{-}thoracic}$ or $P_{abdominal}$. While under this scenario the abdominal viscera could be perfused, importantly the brain, spinal cord, eyes, mammary glands and swimming muscles would not be perfused during dives deeper than about 3 m, when $P_{intra\text{-}thoracic}$ remains near 1 atm.

Conclusions for diving and heart-driven circulation

It is not possible for blood perfusion to continue outside of a 'pressure protected thorax' or 'pressure protected thorax and abdomen' when $P_{body\ surface}$ exceeds $P_{intra\text{-}thoracic}$ by more than about 3 m H_2O. By contrast, if an animal's thoracic cavity collapses in synchrony with increasing depth and the air retained in the lung at submergence is compressed such that both $P_{intra\text{-}thoracic}$ and $P_{extra\text{-}thoracic}$ approximate $P_{body\ surface}$, the load on the left heart remains the same as that for a terrestrial mammal and cardiac output is not compromised.

Venous pooling of blood

Similarly, common to both modes of chest wall support there are problems associated with blood pooling, and as above, these are addressed separately below. We begin with the observation that venous blood returning to the heart flows down a pressure gradient from peripheral tissues towards

[3]We consider the following two examples of carotid artery pressure to represent peak sustained cardiac performance for mammals: Giraffe with head up = 260 mmHg or 3.42 m H_2O (Van Critters *et al.*, 1968); and sprinting horses = 240 mmHg or 3.15 m H_2O (personal communication, Roger Fedde).

[4]Resting systolic intra-arterial pressure of terrestrial mammals.

[5]Ridgway & Howard (1979) showed that the dolphins' superficial locomotor muscles continue to be perfused at depths ≥ 70 m.

and into the thorax. Other considerations aside, whenever $P_{intra\text{-}venous}$ of the peripheral tissues exposed to $P_{body\ surface}$ is larger than $P_{right\ atrial}$ (which is approximately $P_{intra\text{-}thoracic}$), blood will flood into the thoracic vasculature. That is, blood will pool within the intrathoracic portion of the systemic venous system and the entire pulmonary vasculature as a result of the same $P_{body\ surface} \gg P_{intra\text{-}thoracic}$ difference that degrades and then prevents heart driven circulation to tissues outside of the thorax, as described above.

Venous pooling consequences to mode (1) of chest wall support: $P_{intra\text{-}thoracic} \ll P_{abdominal} = P_{body\ surface}$. Assuming the rib-cage and diaphragm do not collapse with increasing depth, blood would pool within the pulmonary vasculature, large intrathoracic vessels, and the heart's atria to the extent that the increase in intrathoracic blood-volume would compress the air within the lungs such that $P_{intra\text{-}thoracic} = P_{body\ surface} = P_{abdominal}$. The lungs' gas volume at any depth will follow Boyle's law, i.e., $P_{body\ surface} V_{lung\ gas} = \text{constant}$. For example, at a depth of only 10 m (where $P_{body\ surface} = 2$ atm), in the absence of chest-wall collapse and if blood pools sufficiently such that $P_{intra\text{-}thoracic}$ equals $P_{body\ surface}$, this volume of blood would be equal to half of the lung's gas volume at sea level. At 30 m the volume of blood pooling within the thorax to keep trans-thoracic pressure near zero would be 75% of the lungs' initial gas volume, etc. While it might appear that with sufficient blood to pool within the thorax such that trans-chest-wall pressures could be ameliorated and further blood pooling arrested, there are lethal complications from rupture of overdistended vessels or atria and associated cardiac arrhythmias (symptoms collectively referred to as 'thoracic squeeze' in human breath-hold divers, see Craig, 1987 and pers. comm., D. E. Leith).

If an animal expires to a smaller lung volume prior to submerging there will of course be a smaller absolute change in gas volume at any depth relative to diving from an initially larger lung volume. It might appear that this strategy would result in less thoracic blood pooling relative to a dive started from full lungs. However, at depths below that at which the chest-wall can continue to collapse without damage, the increase in the volume of blood pooling within the thorax, if trans-thoracic pressure is to be maintained near zero, is inversely proportional to the volume of gas retained at submergence (see Fig. 1). That is, in an animal having a chest-wall that can not collapse to very small volumes without damage, expiring to a low lung volume provides no protection relative to the volume of blood that will pool within the thorax. For example, the chest-walls of humans are known to be 'too stiff' to collapse sufficiently (minimum lung volume determined by chest-wall collapsibility is 20% of total lung capacity), such that blood pooling reaches dangerous levels at dive depths past about 60–80 m. We note that in competitive depth diving among humans (which can exceed 100 m), the dives routinely begin at total lung capacity, in large measure to limit blood pooling (see again Fig. 1).

At least some breath-hold diving mammals have contractile spleens that can expel significant quantities of blood (concentrated red cells) during a dive, e.g. Wedell seals (>300 kg) can eject >20 l of blood (see Hurford *et al*., 1996). Importantly, this 'extra blood' not only does not alleviate the potentially devastating effects secondary to thoracic pooling of blood, but in fact may exacerbate vascular overdistension.

For at least some cetaceans there are descriptions of muscular sphincters surrounding the inferior vena cava just cranial to where it passes through the diaphragm and another surrounding their vena cavae where they enter the right atrium (Harrison & Tomlinson, 1963). We suggest that any thickened fasciculi of the diaphragm surrounding the vena cava can not prevent venous flow into the thorax with pressure drops ($P_{body\ surface} > P_{thorax}$) of greater than a few tenths of an atm. A sphincter located where the vena cavae join the right atrium merely forces, with lethal consequences, the vena cavae to hold until it ruptures all blood pooling within the thorax.

Venous pooling consequences to mode (2) of chest wall support: $P_{intra\text{-}thoracic} = P_{abdominal} \ll P_{body\ surface}$. Assuming that the abdominal wall, in tandem with the rib-cage, is capable of supporting a large pressure jump, pooling of blood (arising from blood outside the 'pressure protected' thorax and abdomen) would still occur, distributed throughout the thoracic and abdominal vasculature such that the likelihood of any single vessel being dangerously over-distended would be substantially decreased. For example, enlarged or paired great veins found within the abdomen of at least some diving mammals could provide increased storage space for blood pooling within the abdomen. While this mode of chest wall support in large measure may avoid the rupture risk of overdistended vessels, such a stiff chest wall would prevent circulation to tissues outside the 'pressure protected' compartment (by analogy to the circulation arguments given above) and would severely compromise the ability to breathe (see below, Ventilatory consequences).

Conclusions for diving and venous pooling of blood

To the extent that chest-wall collapse is insufficient in magnitude to ameliorate the difference between $P_{intra\text{-}thoracic}$ and $P_{body\ surface}$, blood will, and in fact does, pool within the thoracic compartment of breath-hold divers. For example, the chest-walls of

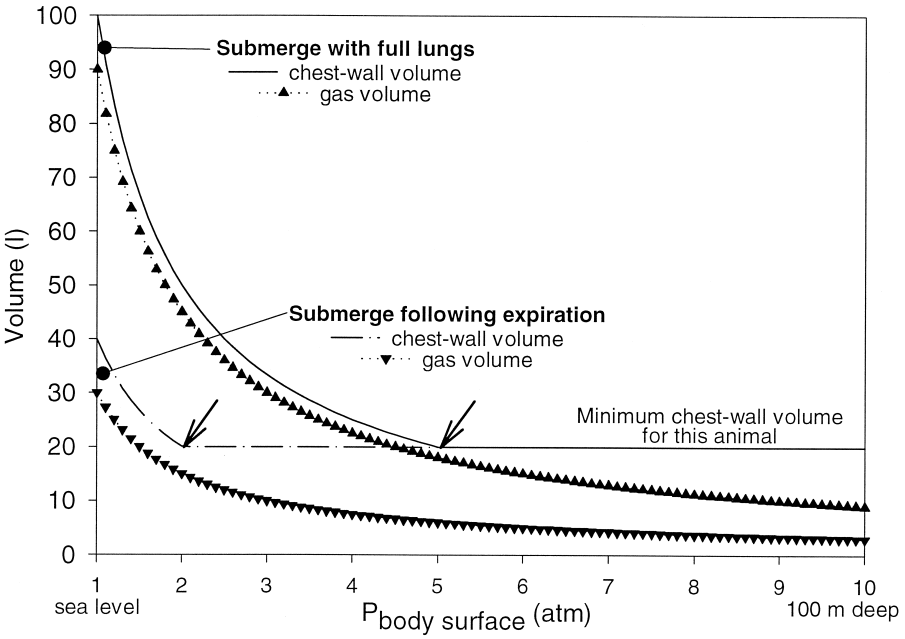

Figure 1. The effect of depth on chest-wall volume, lung gas volume, and blood pooling. Solid line: chest-wall volume beginning near total lung capacity. Dash-dot line: chest-wall volume beginning at a low lung volume. Triangles: lung gas volumes in both cases. Importantly, the difference between the chest-wall volume and the lung gas volume is the sum of non-blood tissue volume (constant at all depths) and blood volume (variable with depth). The volume of gas follows Boyle's Law ($P \cdot V_{gas}$=constant) if trans-thoracic pressures are to remain near zero. In this example, the animal has a chest-wall that without damage cannot collapse to volumes less than 20 l (arrows). At all depths above that, the volume of non-blood tissue plus blood is constant (10 l) regardless of the initial gas volume. At all depths below that, the increasing difference between chest-wall volume and lung gas volume is supplied by increased blood pooling. Note that at all depths below that at which the chest-wall reaches its minimum volume, the amount of blood pooling is larger in the animal beginning its dive at a low lung volume compared with beginning near total lung capacity.

diving humans are known not to be able to collapse to sufficiently small volumes such that at depths greater than about 60 m $P_{body\ surface}$ would remain greater than $P_{intra\text{-}thorax}$ if it were not for blood pooling within thoracic vessels, with all the attendant mortal dangers of such a situation (Craig, 1987).

Chest-wall mechanics

Any argument concerning mechanisms of collapse of the chest-wall during diving must take into account more than just bony ribs and their associated intercostal tissues. Chest-wall collapse in diving animals is dominated by the ease of inward collapse of the long, obliquely oriented soft-tissue diaphragm bounding the thorax's ventro-caudal surface and the soft-tissue abdominal wall (see Tuffy the dolphin at 300 m depth, figure 4 of Ridgway & Scronce, 1969). We may compare the ultimate breaking strength of a cetacean's body wall with the stresses induced as a function of the applied body-surface pressures with increasing depth as follows.

Mechanical consequences of mode (1) of chest wall support: $P_{intra\text{-}thoracic} \ll P_{abdominal} = P_{body\ surface}$. In the case where the abdominal wall is flaccid relative to externally applied pressure, and if the bony rib-cage is capable of supporting a pressure jump ΔP, then the tensile stress applied to the hemispherical diaphragm is, from the Laplace relationship (see Hoppin and Hildebrandt, 1977, p. 102): $\sigma = \Delta P(r/2t)$, where t is the thickness of the diaphragm, r is its radius of curvature and σ is the tensile stress on the diaphragm. Measurements of the diaphragm of adult minke whales[6] (*Balaenoptera acutorostrata*) showed an average thickness of about 2.8 cm and a radius of approximately 100 cm. Yamada (1970) reports that the ultimate tensile strength of skeletal muscle, the tissue from which

[6]Measurements from commercially harvested Minke whales, manuscript in progress.

the strength of the diaphragm is derived, is about 10^6 dynes/cm^2. At a depth of 10 m (1 atm), with no chest-wall collapse $\Delta P = 10^6$ dynes/cm^2. At that depth it follows that the tensile stress applied to the diaphragm is about 2×10^7 dynes/cm^2. Thus, at this very modest depth the stress applied to the diaphragm, if it were to resist collapse, would require a failure strength 20 times higher than observed in a broad spectrum of different muscles from different mammals.

Mechanical consequences of mode (2) of chest wall support: $P_{intra\text{-}thoracic} = P_{abdominal} \ll P_{body\ surface}$. As above for the diaphragm in tension, we calculated the compressive stress applied to the abdominal wall of a minke whale at a depth of 10 m. For thickness of the abdominal wall we used a value of 10 cm, which represents the approximate thickness of the fibrous collagen network interlaced with fat that forms the abdominal wall's stiffest layer; and a value of 200 cm for abdominal radius. The Laplace relationship then gives $\sigma = r\Delta P/2t = 10^7$ dynes/cm^2 for the compressive stress. We have no information for the compressive strength of the fibrous collagen network stiffening the minke's abdominal wall, but it almost certainly is less than the ultimate compressive stress reported for cartilage of 10^8 dynes/cm^2 (Yamada, 1970). Thus, it appears that if a minke's abdominal wall were reinforced with a 10 cm thick layer of cartilage it could possibly resist deformation from a trans-wall pressure equal to that found at a depth of 100 m. However, we suggest below that if the convex abdominal wall has a passive stiffness sufficient to prevent collapse, then breathing at the surface would be impossible.

Ventilatory consequences if the chest-wall is sufficiently stiff to prevent collapse through either mode 1 or mode 2. The abdominal wall musculature of cetaceans and pinnipeds is arranged similar to and appears no more substantial (relative to body mass) than that of terrestrial mammals, and thus cannot actively contribute to supporting the abdomen's convex profile against $P_{body\ surface} > P_{abdominal}$. Importantly, if a diving mammal's abdominal wall could passively resist deformation at a water depth of only 10 m its stiffness would make ventilation while at the surface all but impossible. That is, to the extent that the whale's body-wall is stiff enough to passively resist deformation from force applied to body surfaces, to effect ventilation by muscular driven deformation will require more than an identical force. The pressure necessary to overcome the elastic recoil of the respiratory system (ignoring flow resistance) and thus to drive ventilation in terrestrial mammals is at most a trans-thoracic pressure of about 100 cm H_2O. Thus, if a diving mammal's abdominal body-wall was sufficiently stiff to resist passive deformation at a depth of only 10 m, it would require that its active ventilatory muscles (intercostals and diaphragm) be 10 times stronger than that of terrestrial mammals. By extension, the muscles would have to be 100 times stronger if deformation could be passively resisted at a depth of 100 m. Observations of the thickness of cetacean (dolphin and whale) diaphragms and intercostal musculature suggest they are built no stronger than expected from mass-specific scaling. Additionally, we are unaware of any reports suggesting that pinnipeds' diaphragm or rib-cage are built any more substantially than those of comparably sized terrestrial mammals.

Do diving animals' chest-walls behave as thick-wall spheres?

Van Nie (1987) argued that a trans-thoracic pressure of some 30 atm can be supported across the cetacean's chest-wall. We argue that this is incompatible with life, whether the pressure drop is supported at either the diaphragm (Mode 1) or body surface (Mode 2). By contrast, van Nie's analysis (1987) follows from the thick-walled spherical solution to Navier's equation in elasticity, which displays no such pressure jump, i.e. pressure is a continuous function from body surface to body cavity (see Sokolnikoff, 1956). (The biological equivalent of a thick-walled sphere is a hypothetical animal in which the supporting body-wall is many, many times thicker than the radius of the body cavity it surrounds.) However, all our arguments remain valid in the sense that cardiovascular collapse, tissue rupture, and blood pooling will still occur but in a distributed, rather than localized, fashion. Similarly, our arguments regarding ventilatory inability at the sea surface in the face of a passively stiff chest-wall are independent of the mode of body-wall support.

Supplemental considerations

Not surprisingly, in light of the physiologic consequences that would occur if trans-thoracic pressures were non-zero during diving, it has been well established that the air retained in the respiratory system of breath-hold divers is compressed with increasing depth secondary to chest-wall collapse (see figure 4 of Ridgway & Scronce, 1969) and, in some species at deeper depths, a pooling of blood within the thorax (see Craig, 1987). While information concerning specific modes or mechanisms of chest-wall collapse during breath-hold diving is limited, our general arguments that compatibility with life requires keeping trans-thoracic pressures near zero remain valid.

The chest-walls of cetaceans and pinnipeds are known to be both more compliant and to span a considerably wider range of lung volumes (near zero lung gas volume to total lung capacity) than

those of terrestrial mammals (Leith, 1970). Preliminary examination of the chest-walls of several cetacean taxa suggest that diaphragmatic deformation (in series with abdominal wall deformation) represents the main mode of chest-wall collapse (unpublished observations). In photographs of a spontaneously diving dolphin at a depth of 300 m (figure 4 of Ridgway & Scronce, 1969) it appears that this cetacean's chest-wall deformation is via its abdominal wall, which itself can only deform in series with the diaphragm.

Abdominal viscera, except for any intestinal gas, are incompressible and overall have little shape stability; thus, stresses applied to the abdominal wall from increases in $P_{body\ surface}$ will be transmitted homogeneously to the diaphragm as if the abdomen and its contents did not exist (here we ignore any possibility that the abdominal wall could resist deformation (see above, Ventilatory consequences)). The cetacean diaphragm is very long (>50% of lung's cranio-caudal length) and lies nearly in a frontal plane, whereas the shorter diaphragm of nearly all comparably sized terrestrial mammals lies closer to a cross-sectional plane. The large area of contact between lung and diaphragm in cetaceans allows for the diaphragm to smoothly collapse the lung along the lungs' shortest dimension (i.e., ventral to dorsal).

It might be assumed that chest-wall collapse could compromise cardiac function. The cetacean heart lies atop the cranial-most aspect of the diaphragm, where its position and orientation is constrained by connections between diaphragm and pericardial sac. The heart's vascular connections are to the dorsal body wall via the aorta, to the area of the lungs immediately surrounding the heart via the pulmonary arteries and veins, and the vena cavae lying rather loosely within the mediastinal space between the two lungs. As the diaphragm collapses dorsally, any motion of the heart within the thorax would be identical to that of the surrounding. lungs and towards the aorta's connection to the dorsal body wall; thus, there are no obvious problems with cardiac function secondary to the chest-wall collapse of diving.

Cetaceans (and perhaps pinnipeds) are considered to routinely expire nearly to their lungs' gas-free state; thus, the lung's normal end-expiratory condition at the sea surface mirrors the configuration of the thorax, lungs and heart during diving when the chest-wall has been forcefully collapsed. The intact chest-wall of deceased cetaceans passively collapses (airway opened to atmosphere) to a position at which the lungs are nearly emptied of gas (<4% of the total lung capacity), which is further evidence of the high compliance of the chest wall in these animals (unpublished observations; Leith, 1970; Leith and Lowe, 1972). Additionally, that the chest-wall and lungs of cetaceans and pinnipeds can passively collapse to nearly the gas-free state demonstrates that the lung's airways remain patent to very small lung volumes. This is only possible if the lung parenchyma and airways *per se* are not severely distorted during chest-wall collapse (see Leith, 1970; Leith and Lowe, 1972; Denison *et al*., 1971; Denison and Kooyman, 1973). In contrast, the limit of collapse of the intact chest-wall of a terrestrial mammal without injury occurs at about 20% of the lungs' total gas capacity, and a lung removed from the thorax can only be collapsed to 15% of its total capacity.

Conclusions

The hypothesis that the cetacean chest-wall is nearly incollapsible is incompatible with life on the basis of circulatory, blood pooling, tissue rupture, and ventilatory consequences; these arguments apply even at depths as shallow as 10 m. Even if it were possible to construct a truly non-collapsible chest-wall that maintained intra-thoracic pressure less than body-surface pressure, such an animal would be unable to circulate blood to its extra-thoracic tissues and a potentially large volume of venous blood would pool within the thoracic compartment, threatening rupture of thoracic vasculature and the heart. Furthermore, trans-thoracic pressure differences >1 atm exceed the strength of known biological materials; moreover, body-walls sufficiently stiff to resist deformation even at 10 m depth are incompatible with breathing when the animal is at the surface.

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