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Adaptations to deep breath-hold diving: respiratory and circulatory mechanics

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Leith DE. Adaptations to deep breath-hold diving: respiratory and circulatory mechanics. Undersea Biomed Res 1989; 16(5):345–353.—Respiration and circulation in diving mammals are characterized by interrelated adaptations of structure, function, and behavior that are incompletely described and understood. This speculative survey touches some of them. a) Arterial blood flow can be controlled by vasoconstriction not only in arterioles but also in large arteries. The latter physiology is not well known. b) Mechanisms that might regulate and limit nitrogen uptake are not clear, although Scholander's suggestion that airspaces become gas-free during deep dives is still accepted. c) Systemic arterial retes may be able to store oxygenated blood in some diving mammals. If so, O_2 in the lung might be "skimmed off" early in a dive, leaving the N_2 behind. d) Variable clusters of interdependent adaptations in diving mammals include compliant chest walls that avoid thoracic squeeze; inspiratory breath holds that maintain high lung volumes; large tidal volumes that nearly empty the lung at end-expiration (so there is near-complete turnover of lung gas with each breath); airways that are "armored" by cartilage rings all the way out to the airspaces (so that they do not close and trap gas in the lung and do permit high expiratory flow rates even at very low lung volumes); submucosal vascular retes that may prevent airway squeeze; a puzzling difference in the cross-sectional areas of trachea and bony nares; and very large lungs in shallow divers (sea otters). Study of mammalian adaptations to deep diving promises to illuminate basic issues in physiology.

diving mammals
comparative physiology
respiration
circulation

CONTROLLING ARTERIAL RUNOFF

In 1724, the year before he died in the shoddy Baltic city that he built, Peter the Great gave terse instructions to a Danish naval commander named Vitus Bering:

1) At Kamchatka or other place there you are to build one or two boats with decks.
2) [You are to sail] on these boats along the land which goes to the north, and according to expectations (because its end is not known) that land, it appears, is part of America.
3) You are to search for that [place] where it is joined with America, and to go to any city of European possession, or if you see any European vessel, to find out from it

> what the coast is called and to write it down, and to go ashore yourself and obtain first-hand information, and, placing it on a map, to return here. (1)

Bering went. His two expeditions spanned 17 years (2), and in the end he did not return. He died in midwinter, shipwrecked on a nameless island in the sea that bears his name.

With Bering was Georg Wilhelm Steller, who was thus the first naturalist to visit Alaska. An accomplished and relentless observer, Steller survived, and so did his journals. Condensed here from a wonderful biography by Stejneger (3) is Steller's account of the hungry castaways' approach to the delicious (therefore soon to be extinct) Northern manatee—Steller's sea cow:

> The harpooner stood in the bow of the boat with the hook in his hand and struck as soon as he was near enough to do so, whereupon the men on shore, grasping the other end of the rope, pulled the desperately resisting animal laboriously towards them. Those in the boat . . . wore it out with continual blows, until, tired and completely motionless, it was attacked with bayonets, knives and other weapons and pulled up on land. [It] breathed heavily, as if sighing. From the wounds in the back the blood spurted upward like a fountain. As long as the head was under water no blood flowed, but as soon as it raised the head up to breathe the blood gushed forth anew. . . .

It took me a long time to appreciate the significance of Steller's observation: bleeding from severed arteries cannot be regulated by downstream mechanisms. If the blood that "spurted upward like a fountain" was arterial, then we have to look for local or upstream mechanisms, and they have to be capable of turning flow on and off quickly, on demand. Three points:

1. *Central circulatory mechanisms* provide no explanation for Steller's observations if we assume that central arterial pressure was maintained; and if that assumption is correct, then
2. *distal arteriolar constriction* must not be the only way to control arterial runoff, because it cannot be the direct mechanism here; and finally
3. *rapid reversible closure of large arteries under central neural control* is the only apparent explanation for these observations in Steller's sea cow.

Gooden and Elsner (4) review some of the evidence and issues surrounding the idea of Folkow et al. (5) that blood flow to muscles during diving is regulated by arteries rather than by arterioles. Critical closure can stop flow in small arteries, but is there an upper limit to their size? Do neural mechanisms suffice, or is there neural modulation of underlying humoral vasoconstriction? When and to what extent are arterial rather than arteriolar mechanisms used, by what animals?

REJECTING THE NITROGEN

Nitrogen uptake may be regulated to offset the neural effects of high hydrostatic pressures, but excessive N_2 uptake holds the risk of decompression sickness for deep divers (not for shallow divers). The deep breath-hold diver can take at least three independent approaches to limiting N_2 uptake:

1. Dive at low lung volumes so that there is not enough N_2 on board to be a problem. Let it all be absorbed early in the dive, whereupon N_2 uptake stops.

2. Stop the blood flow through the lung early in the dive, whereupon N_2 uptake stops.

3. Allow lung parenchyma to become gas-free by compressing the gas into conducting airways early in the dive, whereupon N_2 uptake stops (6).

It seems to me that all three mechanisms could lead to a peak of P_{N_2} in the blood early in a deep dive, so the existence of such a peak (7) does not help us to choose among them. Note also that rejecting N_2 in gas phase seems to deny access to oxygen stored there. Now let us examine these three possibilities further.

There is little evidence that the first approach is used. Although some deep-diving mammals have relatively small lungs in relation to their bodies' capacitance for dissolved N_2 and dives are thought to begin at less than full inspiration, still, where these difficult measurements exist, the amount of nitrogen taken up is thought to be less than the amount in the lungs (7). Furthermore, when diving mammals are seen to exhale first upon surfacing, we know that the lungs were not gas-free at the end of the dive, as would have been expected if all the N_2 had been absorbed.

Available evidence does not suggest that blood flow through the lungs stops entirely during diving, and it is doubtful that reduction to low but finite flows would sufficiently limit N_2 uptake at very high pressures; so the second approach does not seem likely to be important.

If the third approach were used, then one might expect that during ascent, gas that had been compressed into the conducting airways would expand again into the lung parenchyma, and N_2 uptake should start again near the depth at which it had stopped during descent. But no second rise, or peak, of blood P_{N_2} is seen at that time in Weddell seals (7), which seems to cast doubt on the operation of this mechanism in these animals. Still, cartilage rings that keep airways from closing are ubiquitous, I believe, in deep diving mammals, and they do permit the airspaces to empty completely, so that gas exchange there presumably ceases; they are not found in the lungs of terrestrial mammals, whose small airways close at low lung volumes, trapping gas in airspaces distal to the points of closure.

But cartilage rings are found around airways of shallow divers, too, who are less at risk from N_2 uptake. Such a remarkable example of convergent evolution suggests strong evolutionary pressures. Are there other possible functions for the cartilage rings that might relate to problems shared by deep and shallow breath-hold divers? Yes; they permit high expiratory flows down to very low lung volumes; *see* below.

SKIMMING OFF THE OXYGEN

We noted above that avoiding N_2 uptake seems to imply that diving mammals must forego access to O_2 stored in the lung. But that is not necessarily true. Perhaps it is possible to skim off the O_2 early in the dive, and only then to shut off N_2 uptake. How might O_2 and N_2 exchange be separated in time?

Some diving mammals have central systemic arterial retes that seem to be capable of storing arterial blood. If, at the start of a dive, the animals could stop distal systemic arterial runoff but allow venous return and cardiac output to continue, they might be able to transfer a lot of blood from venous to arterial reservoirs via the lung. If this process were completed at modest depth, then essentially all of the O_2 could be picked up before the P_{N_2} in alveolar gas became very high; one or more of the mechanisms

outlined above could then operate to stop N_2 uptake. Is this sequence of circulatory events plausible? I think so.

When arterial inflow to a peripheral region is stopped, its venous bed can continue to drain, and some volume of blood that was stored there is transferred to other regions. If the occluded region is a large one with a long time constant for venous return (e.g., abdominal viscera), then a relatively large volume of blood can be shifted to regions with shorter venous time constants (e.g., muscle). This mechanism by itself can increase venous return and left heart cardiac output ($\dot{Q}$) in terrestrial mammals (8).

But what would happen if arterial inflow to almost all vascular beds were stopped abruptly? Assuming that their venous beds continued to drain into the central circulation, we can see at least three possibilities. First, left ventricular ejection and thus $\dot{Q}$ might fall abruptly. Right atrial pressure (Pra) would rise quickly toward the "mean systemic pressure" (8), quickly slowing venous return. Second, blood might be stored in capacious central venous reservoirs. Such capacious reservoirs seem to exist in some diving mammals, along with sphincters that might control venous return from them into the right heart. Again, both venous return to the right heart and $\dot{Q}$ would decrease abruptly, but in this case Pra would fall.

The third possibility is more interesting. If venous blood were permitted to return to the right heart, *and if there were a place for it to go from there,* then as peripheral venous capacitors drained through venous resistances we would expect a quasiexponential decay of venous return, Pra, and $\dot{Q}$. Where might the blood go? I do not know of evidence (or a physiologic rationale) to suggest that the lungs are a big vascular capacitor, and the dilated aorta of diving mammals is not big enough (9). But perhaps systemic arterial retes have the necessary capacity. If so, then widespread interruption of arterial inflow to peripheral vascular beds at the start of a dive, *by itself,* might suffice to initiate transfer of blood from venous reservoirs through the lung into arterial capacitors, in animals that have them, and thus to operate an O_2-skimming and storage mechanism.

It would be nice to know more about those retes. Is their runoff solely to the brain? What is the magnitude and timing of their volume changes during diving? What are they for, if not to store oxygenated blood? What is happening when blood flow stops in major arteries *before heart rate falls* (10)?

Not all mammals have extensive arterial retes, and so the initial circulatory events of the "diving response" are not likely to be the same in all. That is, detailed description and comparison among species (including humans) of these initial circulatory adjustments and their mechanisms might be interesting.

A CLUSTER OF ADAPTATIONS TO DEEP BREATH-HOLD DIVING

Diving mammals' respiratory and circulatory systems display interesting mechanical and behavioral adaptations to deep diving. These are not isolated, but rather form clusters of linked adaptations whose interdependences are perhaps not yet fully appreciated.

Compliant chest walls

Big, adult terrestrial mammals like humans have stiff chest walls, which means they can relax with an open airway without having their lungs collapse (which

presumably would interfere with gas exchange). That is to say, the relaxation volume of their respiratory system (Vrel) is fairly great. But during deep breath-hold diving, thoracic gas volume (VTG) is inexorably reduced by compression as ambient pressure increases. The stiff chest wall resists deformation. As VTG diminishes, outward recoil of the chest wall increases, making pressure inside the chest become progressively lower in relation to pressure exerted on the rest of the body. This tends to drive blood into the chest from extrathoracic reservoirs and also impedes ejection of blood from the left heart. Blood shifts into the chest, tending to overdistend the central circulation. The potentially catastrophic results can be lumped under the divers' term "thoracic squeeze": cardiac arrhythmias, pulmonary edema, and caval rupture.

Those problems disappear if the chest wall is compliant, and that seems to be the solution used by deep divers. (But other solutions are conceivable, and there is a range of chest wall compliance among diving mammals.)

Inspiratory breath holds

Compliant chest walls are good for going deep, but they do not apply passive elastic forces to keep the lung inflated during breathing at the surface; Vrel is essentially zero. Do diving mammals then have to use inspiratory muscle activity to keep their lungs inflated so that gas exchange can occur?

No; there is a simple, actually elegant, behavioral solution: diving mammals close the airway and relax during inspiratory breath holds at the surface. This takes little or no energy; at least some animals' nostrils or blowholes are tightly closed in the relaxed state. At relatively long intervals, diving mammals quickly exhale to near zero lung volume and then immediately inhale a volume that may approach the maximum possible. Thus the tidal volume (VT) may approach the vital capacity (VC), and both may approximate the total lung capacity (TLC), as defined in humans.

So the flexible chest wall is an anatomical solution to the functional problem of thoracic squeeze; but it creates another functional problem, namely, keeping the lung inflated during breathing at the surface. The solution for this problem, in turn, is behavioral: the inspiratory breath hold.

Note that many small and newborn terrestrial mammals have compliant chest walls and low relaxation volumes; they too have behavioral methods of maintaining lung volume above Vrel, e.g., glottal "braking" of expiratory flow. Human infants with lung diseases sometimes use what is called "grunting respiration": quick expiration and inspiration followed by a brief inspiratory breath hold using the glottis. So the functional problems of a compliant chest wall have some generality among mammals; they can find expression in disease, and they can call forth similar behavioral responses, with unknown control mechanisms, even in humans.

"Armored" airways

Cartilage rings keep small airways from closing, so they do not trap gas in distal airspaces. Thus, these rings permit the lung to empty completely; the parenchyma can become gas-free at low lung volumes, perhaps with the effect of stopping N_2 uptake from the lungs at great depth (*see* above). While this may be a persuasive explanation for the evolution of such armoring of small airways in diving mammals,

it is a doubtful reason for making large airways very stiff (noncollapsible) as well, for these are unlikely to be subject to critical closure and in any case would not tend to close until after the parenchyma was gas-free; that is, closure of the large airways could not trap gas in airspaces. But perhaps their stiffness ensures that they maintain a volume big enough to accept gas "compressed into" them from airspaces at depth.

It was noted above that some shallow divers also have armored airways, so we need to look for explanations other than preventing N_2 uptake. In at least some species, armored airways also seem to permit high expiratory flows to be achieved at very low lung volumes. This has two quite different implications that need to be considered separately.

First, high maximum inspiratory and expiratory flow rates seem to be advantageous for diving mammals to minimize the ratio of time spent in ventilation to time spent in breath holding. But the *peak* expiratory flow rates of diving mammals are not especially high in comparison with those of terrestrial mammals. Terrestrial mammals achieve high peak flows, not by stiffness (high specific elastance) of the airways, but by large airway cross-sections and perhaps stiffer lungs than whales. So stiff airways are not essential for high peak expiratory flows, and do not seem to serve that purpose in diving mammals.

But there is more to exhaling fast than high peak flows. In terrestrial mammals forced expirations tend to follow roughly exponential time courses; that is, maximum expiratory flows are roughly proportional to lung volume above residual volume (RV), which might be 20% of TLC. Exhaling to RV requires a long *time* spent on the tail of the exponential, breathing out at low flow rates. Thus hyperpnea for terrestrial mammals is best performed with fairly small tidal volumes, at fairly high lung volumes, so that high flows are achievable throughout expiration.

If, however, other considerations make it desirable, first, to breathe with very large tidal volumes, and second, to approach zero lung volume during expiration, then quick expiration requires that high flows be available *at low lung volumes*. So I think the armored airways of diving mammals may have these physiologic consequences:

1. Stiff central airways
   - provide a reservoir with a gas capacity sufficient to receive all the alveolar gas early in the course of a dive
   - can be relatively small in relation to the maximum flows required
2. Stiff peripheral airways
   - allow airspaces to become gas-free
   - permit high flows at all lung volumes throughout expiration, so that
     a. high peak flows are unnecessary, which allows smaller airways generally (it may be hard to discover whether this is true in any sense);
     b. expiratory time can be short;
     c. large tidal volumes can be achieved without a time penalty;
     d. end-expiratory lung volume can be very low, again without a time penalty, so that near-complete turnover of alveolar gas can be achieved with every breath (*see* below).

Submucosal vascular rete lining airways

Armored airways presumably evolved as a solution to some functional problem(s), but they create another one; they seem to present a squeeze problem similar to that

of the stiff chest wall. At very great depth, the gas in the respiratory system occupies a volume much less than that of the unstressed airways. But the airways in at least some whales are so stiff that airway transmural pressures on the order of minus 100 cmH_2O would exist if they were compressed to low volumes; and similar transmural pressures would probably be applied (in the opposite sign) to engorged mucosal vessels in the airway lumen, enough to damage them. Making the airways more compliant would presumably defeat the purpose for which they are made stiff. But there is another solution. A thick, dense vascular rete underlies the mucous membrane of sperm whales' airways. Perhaps it can accept a volume of blood sufficient to offset much of the volume decrement (and the associated airway deformation) due to gas compression, so that airway (thus vascular) transmural pressures stay in a tolerable range.

Big airspaces, no lobes

Reexpanding gas-free lung regions can be a problem for terrestrial mammals. Big inflation pressures may be required to open small airspaces against the forces of surface tension. Reopening is nonuniform, and some regions may fail to reopen promptly, for example thin margins of lung lobes. Such atelectatic regions may interfere with gas exchange or be susceptible to infection. But we think that deep-diving mammals routinely allow their airspaces to become gas-free. Do they have adaptations that permit easy uniform reexpansion?

Apparently so. The great whales have big airspaces, perhaps a millimeter in diameter, more than 5 times the size of human alveoli. So while surface tensions are not thought to be different, opening pressures are low—around 5 cmH_2O in some great whales—and the lungs appear to open uniformly even at low distending pressures. Furthermore the lungs of at least the whales and phocid seals have no external lobation, hence no sharp lobar margins. Even their diaphragmatic margins have a large radius of curvature, so that even superficial airspaces are mostly surrounded by other airspaces. I think this probably makes local elastic forces more effective in maintaining uniform airspace expansion through mechanical interdependence with adjacent airspaces (11).

Big tidal volumes, low end-expiratory volume, breath-hold breathing pattern

Do the big airspaces solve one problem but create another? We can ask whether diffusion equilibration of gas concentrations within big airspaces might be incomplete, especially when gas fluxes across the lung are high. During a long breath hold this does not seem likely; even during recovery hyperpnea at the surface, the pattern of near-complete emptying (permitted by armored small airways) and refilling, followed by a breath hold, makes gas-phase diffusion disequilibrium seem unlikely, in part because the relevant distances are across the airspaces not along the length of the acinus. The situation contrasts with that in the horse, for example, where, in effect, a long alveolar duct with a high functional residual capacity is ventilated with fresh gas only at one end.

So continuous breathers and intermittent breathers (generally corresponding respectively, as it happens, with terrestrial mammals and with diving mammals, both shallow and deep) seem to use their lungs in different ways: continuous fractional

turnover of alveolar gas on the one hand, contrasted with intermittent complete turnover on the other. Not all of the implications to gas exchange are clear to me; and perhaps the differences between the two systems diminish during exercise or recovery hyperpnea when ventilations and gas fluxes are high.

Big trachea, small nares, relaxed expiration? Some puzzles

Suppose that expiration in a great whale is relaxed, driven by a large and nearly constant hydrostatic pressure—say 100 to 200 cmH_2O, depending on size and posture—plus the smaller and volume-dependent elastic recoil of the respiratory system. Taking a middle value, we would expect that a difference of about 160 cmH_2O between alveolar and atmospheric pressures could accelerate the gas to velocities around 15,000 cm/sec (about half the speed of sound in air) if it were all available for that use. (Of course, some pressure is dissipated in frictional losses.) Higher reported expiratory velocities (17,000–22,000 cm/sec) at the external nares of small gray whales (12) imply even greater unrecovered convective accelerative pressures around 300 cmH_2O, suggesting active expiration.

The tracheas of some cetacea seem to have 2–3 times the minimum cross-sectional area of the upper airways. If that is true, then 80–90% of the convective accelerative pressure drop referred to above occurs as gas enters the upper airways from the trachea, rather than along the intrapulmonary airways. It would also follow that neither the flow velocities in the central intrathoracic airways nor the collapsing pressures applied to them during expiration are very great. This suggests that whether expiration is passive or aided by effort, the achieved expiratory flows are not maximal in the usual sense of being flow-limited in the lung; so perhaps intrathoracic airways are never subjected to significant dynamic compression, and whales do not cough. Flows throughout passive expiration might be nearly constant, driven, in effect, by a nearly constant hydrostatic pressure across the fixed orifice presented by the upper airways.

It is not clear why expiration should be throttled in the upper airways like this, if indeed it is. The velocities there seem to approximate or exceed the velocities seen during a cough in compressed regions of the tracheas of terrestrial mammals; they are higher than the velocities thought necessary for removing even viscid materials from the airways. It would be useful to have measurements, especially in cetacea, of airway cross-sections (nares, bony nares, glottis, trachea) along with measurements of pressures and flows.

Big lungs, small lungs?

Sea otters have high metabolic rates and immense lungs, at least 5 times as big as expected for their size (13). They are shallow divers, presumably free to take advantage of O_2 stored in the lungs. They also have armored airways. The existence of even one such species, and its contrast with deep diving mammals, suggests several things. First, cartilage rings have other functions than allowing airspaces to become gas-free during deep diving. Second, deep divers do not depend much on lung stores of O_2. Presumably they could have evolved bigger lungs, with more stored O_2 (like the otter). Why have they not done so? Perhaps other considerations set limits. We

can ask whether the lung size that suffices for gas exchange at the surface just happens to carry enough but not too much N_2 for deep diving.

CONCLUSION

This speculative account has emphasized interesting principles and possibilities. It is not a review. It is limited by its general approach to a diverse group of animals with widely varying structure, function, habitat, and behavior. It represents the thinking of a respiratory mechanic who is on the sidelines of diving mammalogy. I should be grateful to have called to my attention relevant information that has been overlooked.

Interdependent respiratory and circulatory mechanical adaptations to deep diving, and their neural behavioral correlates and functional significances, are as yet incompletely described and understood. They are susceptible to laboratory and field studies whose design is usefully influenced by mechanistic hypotheses as well as by descriptive goals.

The extent to which these adaptations are shared among mammals including humans is not clear; their study promises to illuminate basic issues in physiology.

Most of these ideas have been the subject, and product, of discussions with friends and associates over the years, including M. Bradley, G. Kooyman, E. Lanphier, E. Sinnett, H. Van Liew, and W. Zapol and their colleagues, among many others. They are among the authors not cited whose thinking is reflected here. Supported in part by grants HL14580, HE11918, and HL19170 from the National Institutes of Health.—*Manuscript received May 1989; accepted July 1989.*

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