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Is it oxygen, or electrons, that our respiratory system delivers?

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ABSTRACT

Respiration involves the inspiration of atmospheric gases. Arguments are put forth that it is not oxygen gas that passes from the alveoli to the capillaries, but electrons extracted from the oxygen. Those electrons are theorized to bind to hemoglobin. They are then passed by the circulation directly to the tissues, where they support metabolism. Issues confronting the standard respiratory paradigm are identified, while various observations are put forth that seem consistent with the direct role of electrons in the respiratory process. If the hypothesis is validated, then a direct link will have been established between respiration and metabolism.

Introduction

Questioning whether we breathe oxygen must surely seem bizarre, for oxygen deprivation leads quickly to suffocation, followed by death. Could there be any question?

Yet, certain enigmas emerge from the widely held mechanism of respiration, which are rarely addressed. Here I consider several of them. I then go on to suggest a mechanistic variant by which those enigmas resolve in a natural way. The variant involves a central role of electrons. For sure, oxygen is critical for life, but I raise question whether it is the oxygen itself that is the critical agent, or electrons extracted from the oxygen.

I Begin by citing an issue that is not commonly considered by those dealing with respiration: The breathing of fish. At extreme depths, oxygen is in short supply; yet fish manage to survive. If vertebrate life requires oxygen, then how do those fish make it? this paradox is relevant to our consideration, and I will deal with it later

For now, we focus on humans.

Passing gas

Every few seconds we cycle between inspiration and expiration.

During inspiration, our lungs draw in atmospheric gases, comprising not just oxygen (~21 %), but also nitrogen (~78 %). We also breathe argon (~1%) and several trace gases [1]. Most of those inspired gases never make it past our lungs. Only oxygen manages apparently to pass from the alveoli (air sacs in lungs) to the capillaries that envelop them (Fig. 1). Nitrogen does not ordinarily pass, nor does argon. Hence, whatever mechanism supposedly facilitates passage of oxygen to the cardiovascular system must somehow block the other atmospheric gases. The inspiratory process appears to be selective.

Augmenting our understanding of the selectivity issue is the passage of various gases that are noxious. Examples include the halogens [2]. As poisonous gases, fluorine and chlorine presumably exert their toxic effects on body tissues, which implies they must first pass through the alveolar membrane to the blood. Yet, both molecules are substantially larger than nitrogen, which ordinarily cannot pass. Thus, size is evidently not the determining factor in the passage from alveoli to blood.

There appears to exist a complex selectivity paradigm: certain noxious gasses pass, oxygen passes, but other atmospheric gases including the most abundant one, nitrogen, do not pass. Clearly, the inspiration mechanism must be less simple than we often presume.

Nor can we fully rationalize the expiration process. Expired gas is presumed to contain carbon dioxide (Fig. 1). As an end product of metabolism, that gas is considered to follow the reverse course of inspiration, supposedly passing from tissues to blood, then from blood to lungs, where it gets released into the atmosphere. Thus, CO_2 is yet another gas to be added to the mix of those that do apparently manage to pass through the lung-blood barrier, notwithstanding that molecule's hefty size.

The mix of molecules that do or do not pass raises the obvious question: how? Why do some gases pass but not others? Or even more fundamentally, how does gas pass through any continuous membrane?

Confounding issues

For explaining the passage of molecules through membranes, the

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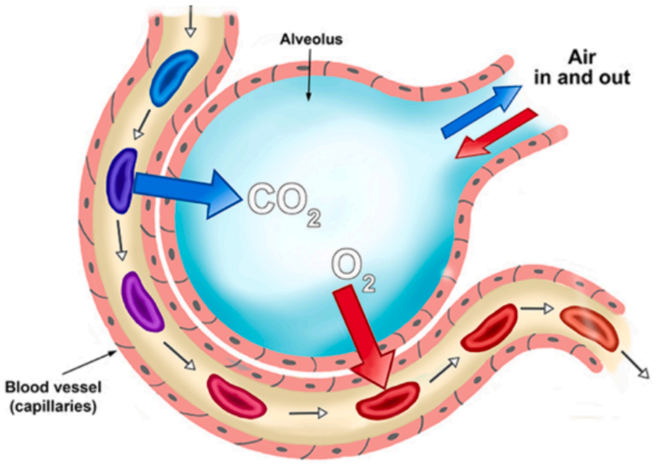

Fig. 1. Respiratory system, as commonly conceived. Alveolar oxygen passes from alveoli to red blood cells in capillaries. Carbon dioxide follows the reverse path. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

commonly accepted driving mechanism is diffusion. By the force of Brownian motion, gases are thought to pressure themselves through membranous barriers. On the other hand, gases such as oxygen form bubbles in liquids, shifting the question to how a bubble could diffuse through a continuous membrane.

A possible mechanism is the opening of pores. Since each balloon-like alveolus expands during inspiration, expansion could open membranous pores, allowing oxygen to pass. But if oxygen passes, then why not nitrogen as well? Not only does nitrogen have a smaller atomic mass, but also it is four times more prevalent than oxygen. With the pore-size explanation, then, you'd think we'd breathe mainly nitrogen — or at least a goodly amount. Hence, that potential mechanism appears unlikely.

A possible rescue: On theoretical grounds, some sources argue that notwithstanding its larger mass, oxygen's volume may be ever so slightly smaller than that of nitrogen [3], making its passage easier; so we breathe mainly oxygen. But that argument faces obstacles. Considering that the volumes of the two molecules are at least roughly comparable (they are neighbors on the periodic table) the rate of passage of the two gases should be of the same order. We should breathe both. Further, a slightly deeper inspiration that opens the alveolar pores a bit more would favor the struggling species: nitrogen. Yet, even with deep breathing, nitrogen fails to pass at all from alveoli to the blood.

Another rescue option circumvents all of those gas-based considerations. Since the alveolar membrane is a complex cellular system, thereby containing water, one could envision the dissolution of oxygen in membrane's water. Through such dissolution, molecular oxygen could theoretically pass through the alveolar membrane to the capillaries. The problem is quantitative. The solubility of oxygen in water is so extremely low (roughly 10 molecules of oxygen per million of water) that we'd be perpetually struggling to breathe. Hence, this option fails to solve the problem.

The question remains: How can we understand why some gases seem to pass easily from alveoli to capillaries while others do not? Relative molecular size apparently fails to provide a meaningful explanation. No obvious answer seems at hand.

A second and entirely different issue deals with efficacy. To capture as much oxygen as possible from those alveolar air sacs, blood ought to surround each alveolus as a continuous sheath, like a glove enveloping a hand. But it does not. The capillaries that surround the alveolus are separated. In most species they are quantitatively sparse: total capillary surface area enveloping each alveolus is estimated to be of the same order of magnitude as the surface area of the alveolus itself [4]. While this measure might at first seem generous, it is not. Only a small fraction of the vessel circumference can intersect the alveolar surface. Hence, only a modest fraction of alveolar oxygen should make its way into the capillaries, the rest wastefully diffusing beyond — a curious situation when nature might be expected to maximize oxygen transfer.

Continuing on this same (lack of) efficiency theme, a third issue concerns capillary diameter. An odd feature of capillaries is their narrow diameter, down to ~ 3 – 4 μm in in healthy young adults. This is half the diameter of the red-blood cells, 6—7 μm, that need to pass through them. The issue is especially prominent in alveolar capillaries, with roughly half of all capillaries narrower than red cells [5]. Squeezing erythrocytes through those vessels requires energy, and any such energy expenditure, one might think, ought to have some well-defined purpose.

Hence, the third enigma emerges: Why are capillaries so narrow as to require the appreciable energy expenditure in order to allow red-blood cells to pass through? Has mother nature erred? Or might the squeeze be necessary to facilitate some critical, yet-to-be-identified purpose?

In sum, an understanding of respiratory-system function appears to face a series of paradoxes. The first: how does the alveolus permit gas passage through its membranous shell, apparently with complex selectively rules? The second relates to the overall physical system design: while the gas should pass efficiently from alveoli to capillaries, why are alveoli-capillary interfaces as sparse as they are? The third question is energetic: why are pulmonary capillaries so narrow that erythrocytes must be energetically forced to contort in order to flow through?

It appears that aspects of the respiratory system seem to deviate from the simple designs anticipated from nature. These issues don't disqualify the conventional mechanism, but they do raise question whether some variant of conventional thinking might better fit the facts. Oxygen seems necessary for animal life. But might the underlying oxygen-utilization mechanism involve something beyond the framework of what we've come to accept?

The hypothesis

I suggest that it's not oxygen *gas* that our bodies require, but electrons drawn from that oxygen. That is, no gas flows from alveoli to capillaries, only electrons extracted from the oxygen gas.

To appreciate how this dynamic might occur, we must recognize that the oxygen molecule is highly electronegative, one of the most electronegative elements on the periodic table. That means it has a profoundly strong tendency to accumulate electrons.

Whether any tendency exists to give up those electrons in the right circumstance is less clear. Conceivably, some of those electrons could be drawn off by positioning a positive charge close enough. When opposite charges lodge near one another, electrostatic attraction can be of impressive magnitude. Any such positively charged entity could thus serve as a receptacle for oxygen's electrons. And if that positive entity happened to lie within a capillary, then it could transport those electrons directly to tissues downstream, where needed. Hence, drawing electrons from even the most electronegative substance would seem at least plausible.

Recognizing the ultimate need for electrons in tissue metabolism, one could envision the electrons initially transferred from oxygen to red blood cells, then delivered downstream to relevant sites in tissues. In such a way, the electrons required for metabolism could be delivered directly to the tissues, absent any intermediate steps.

Does reason exist to entertain such an hypothesis?

First, the mechanism averts issues associated with gas flowing through membranes. No gas flows in this mechanism; it's only electrons that flow. Those electrons could theoretically come from any source, oxygen being perhaps the most capable, though not unique (see *The Fish Conundrum*, below) donor.

Second, the erythrocyte squeeze can be rationalized. Those squeezed erythrocytes necessarily abut the capillary wall. In so doing, they ensure contiguous contact: erythrocyte – capillary wall – alveolar wall. Contiguity averts potential complications posed by any intervening insulating layers, ensuring high electrical conductance. In fact, the surfactant layer that lines the alveolus is recognized to have particularly high conductance. Thus, electron charge could transfer efficiently from oxygen gas on the alveolar side to erythrocytes on the vascular side. Why capillary diameter would need to be smaller than red-cell diameter can therefore be appreciated: erythrocyte squeeze may be critical for allowing electrons to flow readily from oxygen to abutting red-blood cells.

The third point deals with hemoglobin, the main constituent of the erythrocyte. To fit the hypothesis, hemoglobin would need to bear positive charge in order draw oxygen's negative electrons. Once the electrons are bound, hemoglobin's charge would neutralize, or even become negative.

Hemoglobin's two states are widely confirmed [6]. The so-called T form is associated with low pH, *i.e.*, positively charged, while the R form is associated with high pH, or negatively charged [7]. Evidently, the two distinct charge states required in the hypothesis do exist, and they are well documented. We easily detect them as red (arterial) and purple (venous) blood.

Hence, the proposed cycle would proceed as follows. First, positively charged hemoglobin draws electrons from oxygen. Then it delivers those electrons to the tissues, regaining its positive state and recovering its ability to extract electrons from the inspired oxygen.

A central feature of this hypothesis is that the hemoglobin must have the capacity to attract, and then surrender the electrons that it stores. The attraction (by positive charge) has just been dealt with. As for the electron surrender, it's noteworthy that hemoglobin has a tendency to easily oxidize [8], *i.e.,* to lose its electrons. That feature may be of concern for blood in storage, but it fits well with expectations of the proposed hypothesis. Hemoglobin can evidently attract, and then deliver electrons.

Hence, the hypothesis appears to enjoy at least modest foundational underpinning: it averts complications associated with passage of gas through membranes; it provides rationale for the bending of erythrocytes; it offers justification for hemoglobin's two states; and, it justifies the capillaries' relative sparseness, with positively charged hemoglobin serving as the attractor of oxygen's negative electrons.

A final consideration involves exhalation. The hypothesis implies that at the very least, the exhaled gas ought to include what remains of the inspired gas after electrons are extracted: namely, nitrogen and positively charged oxygen. The latter, now with positive charges, should be highly reactive. Hence, the exhaled gas might be expected to contain some product of oxygen and nitrogen, and indeed that is the case. Exhaled gas contains nitric oxide [9].

A possible objection to the proposed hypothesis: If the sole respirational requirement for survival is electrons, then why are gases such as hydrogen sulfide and carbon monoxide, which are electron-donating, so toxic? Shouldn't the proclivity for electron donation make them behave like oxygen?

A possibility is that those gases may lack the capacity to donate *multiple* electrons. Oxygen has five oxidation states, −2, −1, 0, +1, and + 2, implying a capacity to donate not just one but multiple electrons. If so, then the hemoglobin transition could require multiple electrons. That feature is consistent with the natural oxidation of hemoglobin, which favors the loss of two electrons rather than one [8]. If the natural respiration process indeed involves surrender of a pair of electrons, but the noxious gases can only surrender one, then that could perhaps explain why noxious gases cannot substitute for oxygen.

Matching supply with demand

The proposed respiratory mechanism's essential product is electrons. Electrons, meanwhile, are widely recognized as a supplier of fuel for running cells. Hence, correlation exists between supply and demand.

In the classical demand mechanism, however, exploiting that fuel involves an electron-transport chain, which ultimately produces ATP. That molecule's high-energy phosphate bond is thought to supply the ultimate energy required for powering cellular processes. A multiplicity of reactions is involved, and it is beyond the scope of this article to outline those steps in detail. Nevertheless, electrons are central features. Hence, electrons are central to the widely accepted metabolic mechanism, and those electrons are arguably produced directly from the proposed respiratory mechanism. Supply and demand match.

Given that correspondence, I'm drawn to mention a recently proposed mechanistic variant that connects supply with demand even more directly. That variant involves intracellular water. Water molecules, when lying adjacent to hydrophilic surfaces, split into positive and negative components [10]. The negative component has been labeled "fourth phase" or "exclusion-zone" (EZ) water, earlier termed "structured" water. Building adjacent to the cell's hydrophilic surfaces, EZ water largely fills the crowded cell [11]. In so doing, EZ's negative charge arguably accounts for the cell's well-recognized negative electrical potential [12]. That electrical potential remains well sustained over the long term.

The cell's electrical potential, however, is not the main point. The main point is the net charge underlying that electrical potential. Negative potential implies excess electrons inside the cell. Those crowded electrons amount to potential energy, expended as the negative charges naturally disperse. Such energy expenditure is arguably manifested as a so-called phase transition [11], wherein negatively charged EZ water transitions into ordinary neutral liquid water and the cellular proteins transition into folded proteins. Those two features do the work of the cell, *e.g.,* contraction, secretion, nerve conduction, *etc.* — powered by the potential energy of excess electron charge [12]. Electron charge is central.

Following cellular action, the cell must return to quiescence. Proteins must return to their extended configuration while liquid water must return to its negatively charged EZ state. The return, therefore, requires electrons. And therein lies the critical point: As proposed, those electrons may come directly from respiratory oxygen, arriving at the tissues via the circulation. Absent such an electron supply, the cell could not return to its quiescent state, subsequent cellular work could not be carried out, and the organism would quickly become dysfunctional.

That electrons can rebuild EZ, as required in this formulation, has been demonstrated. Laboratory studies have confirmed that electrons supplied by simple electrical current can directly convert neutral liquid water into negatively charged EZ water [13,14]. Hence, the critical step in the reverse phase transition has been experimentally validated.

In the above-described framework of cellular action, the linkage between supply and demand is particularly tight. Electrons are supplied by the respiratory system, and those electrons are directly consumed by the tissues. Simplicity prevails. In this context, the concept of electron donation from the respiratory system would seem to make sense.

The oximeter challenge

A potential objection to the above arguments lies in the workings of the common fingertip oximeter [15]. Clipped onto the tip of the finger, the oximeter is said to measure oxygen saturation, not electron saturation.

The device consists of two light-emitting diodes (LEDs), each emitting a distinct wavelength (color) of light. The emitted light passes through the finger, hence through the finger's blood. A photosensor on the opposite side of the finger detects the amplitude of each passing wavelength. Because the absorption spectrum of "oxygenated" (red) arterial blood differs significantly from that of "deoxygenated" (purple) blood, measuring the relative amplitudes of those two wavelengths can provide information on oxygen saturation.

But is it genuinely "oxygen saturation" that is being measured? The underlying assumption is that arterial hemoglobin is saturated with oxygen while venous hemoglobin is devoid of oxygen. Oxygen appears to make the difference. But we would not know if the difference in hemoglobin structure arises from electrons, rather than oxygen? The oximeter merely reports structural differences. It says nothing of the basis of those differences.

We cannot, therefore, assert that the finger-tip device constitutes disproof of the proposed mechanism. The device is thought to measure oxygen but it could just as well be measuring electrons.

The fish conundrum

A mechanism has been proposed that circumvents certain confounding issues arising from conventional understanding. The proffered mechanism involves the transfer of negative charge from the oxygen molecule to the tissues. The critical agent is the electron, not the gas.

To help judge the proposal's merit, one way is to see whether vertebrate life can persist in situations in which oxygen is in short supply. If life could be sustained by electrons instead, then this would lend support to the proposed hypothesis.

Consider deep-sea fish. Oxygen cannot readily diffuse from the atmosphere to depths at which those fish flourish, often several miles beneath the ocean surface. At those depths, responsibility for providing metabolic energy would likely not lie with oxygen [16]. Instead, the fish "breathe" the surrounding water.

In such breathing, water is first taken into the mouth. It then passes through the gills, eventually exiting the fish from its lateral gill slits. That exiting water is confirmed to be more acidic (positively charged) than the neutral intake water [17]. Since the exiting water has gained positive charge, accounting for the intake water's neutrality means that the gills must gain negative charge (OH^-). A mechanism for achieving that charge splitting has been proposed [18].

The gills, like the lungs, are invested with capillaries [19]. Those capillaries permit the gills' acquired negative charge to be directly exploited for metabolic needs. Thus, fish arguably use electrons in the same way proposed for humans and other vertebrates. Oxygen seems irrelevant for those fish because very little oxygen is present in their waters. Effectively, fish breathe electrons, not oxygen.

Recall that when out-of-water fish are directly exposed to the oxygen gas in the atmosphere, they cannot survive. Fish apparently have no capacity to deal with oxygen gas. Something else must suffice, and that appears to be electrons (from OH^-). The mechanism closely resembles what is proposed for humans, lending credence to that mechanism.

Evaluation: Respiring without hemoglobin

Another consideration deals with the role of hemoglobin. If the binding of oxygen needs hemoglobin and hemoglobin is removed from the blood, then any survival would imply that the critical agent cannot be oxygen.

That very procedure has been carried out. The story begins with the well-known 19th century French physician Rene Quinton, who collected ocean water in proximity of algal blooms in the Atlantic near the France-Spain border. That water seemed particularly efficacious: prior to the advent of antibiotics, routine infusions of that water directly into the vasculature successfully treated infections. Even today, "Quinton Water" remains available.

Quinton performed a series of audacious experiments even before recognizing the clinical value of this water [20], and recounted a century later [21]. In early experiments, he infused large amounts of isotonic seawater, up to 104 % of body weight, directly into the saphenous vein of dogs, substantially reducing the hemoglobin concentration. The dogs recovered. In a later experiment, Quinton withdrew from the femoral artery essentially all of the dog's blood (about 5 % of body weight, over four minutes) until the animal was fully exsanguinated and at death's door. Only then was the seawater infused, over a period of 11 min. Following inevitable functional difficulties, the dog eventually regained full function, notwithstanding substantial diminution of blood hemoglobin.

These remarkable experiments demand repetition. If the results are valid, then they throw important light on the perceived need for hemoglobin, and thus for oxygen. On the other hand, the substituted seawater does not require hemoglobin to store electrons. The negatively charged EZ water could deliver electrons to the tissues. As in the fish example, electrons can apparently substitute for oxygen.

Loss of hemoglobin also happens in accidents and in war, with loss of blood. The acute need for replacement has spurred considerable research for injectable blood substitutes. According the Pacific Heart, Lung, and Blood Institute [22], one of the most promising blood substitutes seems to be perfluorocarbons [23]. PFCs are derived from fluorine- and carbon-containing compounds. A salient feature of fluorine is its profound electronegativity: even more than oxygen [24]. One wonders, therefore, whether blood substitutes might work as effectively as they do because of their capacity to accumulate, and then donate electrons.

Perspectives

From logical and evidentiary arguments, I have tried to demonstrate the likelihood that the key figure in the respiration process might not be oxygen, but electrons drawn from oxygen. I did so by first identifying certain confounding issues with the accepted respiratory mechanism, and then introducing an alternative that appears consistent with multiple observations.

The proposed charge mechanism interfaces nicely with the recently proposed cellular action mechanism, in which intracellular water stands as central to function [10,11]. A pivotal feature of that mechanism is the exploitation of water's separated electrical charges. While that mechanism's existence is not a strictly necessary companion to the proposed respiratory mechanism, it does provide a natural adjunct, and that is why I mention it.

If the proposed thesis has merit, it should be testable beyond the already-mentioned considerations. One experimental approach tests whether expired air contains positively charged oxygen. According to the hypothesis, it should. A second approach tests whether electrons alone can transform erythrocytes from one state to the other, and the reverse when those electrons are withdrawn. A third test explores the plasma that surrounds the erythrocytes: according to conventional thinking, alveolar oxygen should diffuse not only into the red cells, but also into the space between those cells; thus, plasma should be oxygen rich. By contrast, the proposed hypothesis predicts that the plasma should contain no oxygen at all. Carrying out those tests can shed additional light on whether the proposed mechanism, though radical, has validity.

A general implication of the electron-based mechanism under consideration here is that in biology, electrical charges may reign supreme — *i.e.*, that electrons may be the dominant players [25]. Those electrons could well constitute the common agents of action not only in the respiratory supply system, but also in the cellular consumption system as well as the energy-transport system.

In other words, the human body may function less as a chemical machine than as an electrical machine [26]. Electrical phenomena are known to dominate numerous aspects of human physiology and beyond [18,25], ranging from cardiac electrophysiology to brain function. Here, I argue that the respiratory system may function electrically as well. Given the diversity of phenomena acting electrically, it may indeed be appropriate to think of the body as a premier electrical machine.

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Gerald H. Pollack: Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Conceptualization.

Declaration of competing interest

The author declares that he has no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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