

Pathology is Physiology with Obstacles. The Unsuspected Capacity of Human Cells to Oxygenate Themselves

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ABSTRACT

The basis of gas exchange in the pulmonary alveoli is that oxygen passes through the pulmonary alveolar walls by simple diffusion. This is a dogma that dates back to the mid-eighteenth century, and has no physical, chemical, or even experimental basis.

Our observation that human eukaryotic cells possess intracellularly located molecules, capable of transforming the power of sunlight into chemical energy, through the dissociation of water molecules, as in plants; It is a disruptive discovery that will necessarily modify our current concepts about cell biology and therefore human physiology.

In this paper, we collected observations from researchers that suggest the ability to dissociate water from various human tissues, but which were ignored or misinterpreted.

Keywords

Blood, Hemoglobin, Diffusion, Dissociation curve, Hydrogen, Oxygen, Water dissociation.

Background

Every branch of science has its elementary unit: for physics is the atom, for chemistry the molecule, for astronomy the planets, for medicine the cell. Before the invention of the microscope, diseases were explained by causes such as spells, the introduction of foreign objects and substances into the body, divine punishment, or even loss of the divine spark (soul).

In Greece, it was blamed on breath or wind, or on an imbalance of the 4 humors: blood, phlegm, yellow bile and black bile. For them, all diseases occurred when the delicate harmony between the 4 basic humors of the body was disturbed. And this idea persists to this day, for example with the purges of grandmothers, or suction cups, even sangria. It is interesting that the Greeks did not have a

word for infection, since it comes from Latin.

Over time, poisonous substances were discovered and eventually used, and some are still used as medicines. And this without knowing almost anything about anatomy and physiology. So, the results were limited since it is not possible to repair or rescue a diseased organism without knowing how it is built and how it works.

The first dissections took place in Alexandria, in the year 300 BC, The words prostate and duodenum (twelve fingers) date from that time. Although the Greeks working in Alexandria did not have microscopes, they did have the mind's eye. So much so that the microscopic concepts of **tissue** and **parenchyma** date back to that time [1].

Galen, dissecting living and dead animals, discovered the function of the recurrent laryngeal nerve, also observed the effects of the

partial and complete section of the spinal cord, finally banishing the idea that arteries carried air.

The conditions for advancing in the study of anatomy were not the best once the Roman Empire collapsed. Until the thirteenth century A.D., a small book on anatomy written around 1316 by Mondino de 'Luzzi, in Bologna, was published in Italy, which reached 39 editions. At that time, it was a formidable challenge to dissect corpses, as it was difficult to work on a partially decomposed body, without gloves; without antiseptics, without fixatives; running water, and no notion of what an infection is, not to mention religious issues, perhaps because of the future resurrection.

At the end of the Middle Ages, dissection became part of the medical curriculum. The first pathologists were anatomists. And gradually the dissection of a sick corpse was called an autopsy. The first book on the subject was written by Giovanni Battista Morgagni, and was called; On the seats and causes of disease investigated by anatomy, which was published after his death in 1771.

Perforations or obstructions of the intestine, jaundice due to compression of the bile duct, abscesses, etc., were masterfully described, although it was not known what an abscess itself was. By 1850, surgery had made great strides in European medicine but had to be halted by frequent infections. Then the microscope came into play, and the word cell was coined by Robert Hooke in 1665, when he observed cork cuts.

Theodor Schwann (1810-1882) wrote in his book Microscopic research on the similarity in structure and function of animals and plants, the following sentence: All animal and plant tissues are made up of individual microscopic units, which provided to biology (and medicine) with its elemental unit: the cell.

In 1859, just 19 years after the cell theory emerged, Rudolf Virchow published 20 papers in a book entitled: Cellular pathology as based upon physiological and pathological histology, in which it was firmly established that disease cannot be understood unless it is realized that the ultimate (or first) abnormality must lie in the cell.

Our body can be conceptualized as a state, community, or commonwealth in which each cell is a citizen. And Rudolph Virchow is credited with the definition with which this article begins: Pathology or physiology with obstacles.

Clinicians now think in terms of cellular receptors and organelles, and they treat with drugs that select specific cellular targets. Even shock, considerate ultimate total disease, is now recognized as a generalized cellular disease. Thereby, disease ultimately makes sense only in the context of the elementary patient: the cell.

Most cellular malfunctions are accompanied by structural changes, which in turn guide us to the underlying functional problems. And the next thing is to discard countless functional, biochemical,

and histochemical tests. And although an athlete may weigh 3 or 4 times as much as an ascetic monk, the difference is only the adaptation of the same cells to the lifestyle.

Blood

Complexes of hemoglobin and carbon dioxide have been known for a long time; [2], however, so far, all researchers have regarded the oxygen uptake of the blood and its carbon dioxide uptake as two independent processes. Bohr found instead in the cited discussion that even though the carbon dioxide-uptake in the presence of oxygen remains uninfluenced, the oxygen uptake of the blood is usually reduced if a certain amount of carbon dioxide is present. However, from a quantitative point of view, the results were only reproducible with a relatively large error which may be due to great variability of the hemoglobin molecule.

No.	Pressure (mm)		O ₂ in 100 ml	Percent oxygen amount
	CO ₂	O ₂		
1	6.2	10.7	4.60	25.1
2	8.6	25.4	12.52	68.4
3	13.0	46.9	16.18	88.4
4	8.9	151.1	18.31	100

In this table, taken from the work of Christian Bohr [3], we can see at least two details, that the pressure of oxygen is always greater than that of CO₂, which is explained by the fact that CO₂ is 70 times more soluble in water than oxygen, and the second detail is that the pressures of oxygen that Bohr describes, they cannot be reached by simple diffusion. It has always been accepted that CO₂ is produced inside cells, which explains the pressure in mm Hg that is described, and if we consider that oxygen is also produced inside cells, through the dissociation of water, then the pressures that Bohr describes in his experiments begin to be coherent.

The pressures in mm Hg of oxygen are always higher than those of CO₂, because due to the poor solubility of oxygen in water, it is even repelled by it, which means that oxygen cannot leave the interior of the cells, but it cannot enter either, since the barriers are the same in both directions.

Then, then, oxygen (O₂) can neither pass through the walls of the pulmonary alveolus and reach the relatively high pressures that have been experimentally found, let alone by simple diffusion, as has been handled to date.

Likewise, Bohr says that the amount of CO₂ significantly affects the amount of oxygen, but it is because the unsuspected ability of hemoglobin to irreversibly dissociate water molecules is very sensitive to CO₂ levels. On the other hand, Bohr also refers to the great variability of the hemoglobin molecule, since as cited in this and other works, the amount of light significantly modifies the results of experiments. Since in both plants and mammals, light is the source of energy used by hemoglobin, chlorophyll, myoglobin, cytochrome P 450, and bilirubin (all derived from protoporphyrin IX).

The molecules that dissociate the water molecule do not use ATP as an energy source, nor do oldest molecules with enzymatic activity, for example: carbonic anhydrase, and other synthetases. Enzymes that use ATP as an energy source are called synthetases.

The role of sunlight

In the literature we could find examples that tell us about the fundamental role of light in the biology of the human body. It has been reported that children under 4 1/2 years of age can reconstitute the tip of the finger when it is sectioned above the distal section of the phalanx [4]. (Figure 1) Similar results have been reported in mice [5]. These works do show that repair in young age is excellent, and optimal regeneration occurs if the raw surface is not occluded by dressing (and therefore exposed to light), since the lack of light inhibits growth.



Figure 1: Accidental amputation of the tip of the pinky finger in a 10-year-old infant.

Giulio Bizzozero, one of Golgi teachers who named the platelets, proposed that cells can be classified in three categories [6]: 1) Labile, those that continue to multiply throughout life (bone marrow, most epithelia). 2) stable, those that can multiply but are normally quiescent (hepatocytes, fibroblasts, endothelium smooth muscle), 3) Permanent, those that cannot proliferate, (neurons, DNA does not replicate in post neonatal life, except the “song center” in canary brain, mouse cortex *in vitro*, fat cells do not multiply but can revert to a fibroblast-like phenotype). There are permanent cells with capability to duplicate DNA, this is: striated muscle, myocardium, and glomerular podocytes. In these cells, nuclei retain the capability to multiply (muscle), or to become polyploid (myocardium), or to multiply *in vitro* (podocytes) [7].

The "labile" cells of the bone marrow multiply throughout life. The formation of a red blood cell takes about 2 days (from stem cell to proerythroblast). The body makes about two million red blood cells every second! [8]. Thereby, the bone marrow makes more than 220 billion new red blood cells daily. And the most active

part of the bone marrow, the central portion, lacks blood vessels, and instead contains sinusoids. Between the hematopoietic cords, run the sinusoids, which have discontinuous endothelium, through which newly differentiated blood cells and platelets enter the circulation.

The bone marrow sinusoids provide the barrier between the hemopoietic compartment and the peripheral circulation. The sinusoid of the red bone marrow is a unique vascular unit. It arises from blood vessels that supply the cortical bone tissue at the corticomedullary junction. The sinusoidal wall consists of an endothelial lining, a discontinuous basement membrane, and an incomplete covering of adventitial cells. The endothelium is a simple squamous epithelium [9]. When blood cells formation and the passage of mature blood cells into the sinusoids are active, adventitial cells and the basal lamina become displaced by mature blood cells as they approach the endothelium to enter the sinusoid from the bone marrow cavity.

The sinusoids of the bone marrow are not blood vessels per se, that is, they do not have a continuous connection with the circulatory system, so that there is a bloodstream that provides glucose and other nutrients. Rather, they are like small storehouses where newly formed red blood cells accumulate before they pass into the bloodstream. So how is one of the most metabolically active tissues in the body provided with the necessary nutrients? (Figure 2, 3).

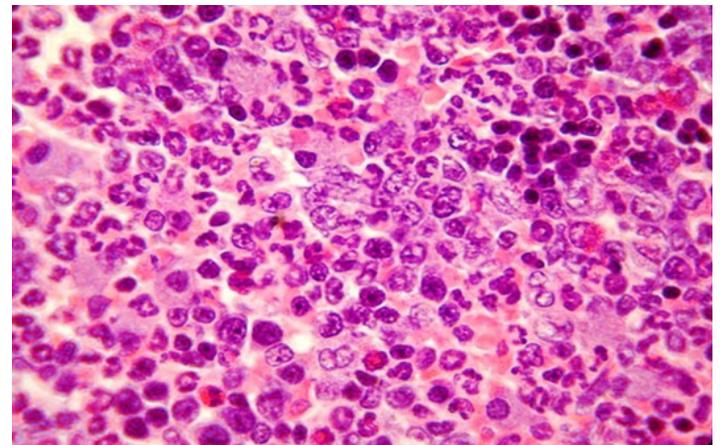


Figure 2: Wistar rat medullary smear, stained with hematoxylin and eosin. 40 X. Hematoxylin has a deep blue-purple color and stains nucleic acids by a complex, incompletely understood reaction. Eosin is pink and stains proteins nonspecifically. In a typical tissue, nuclei are stained blue, whereas the cytoplasm and extracellular matrix have varying degrees of pink staining.

The generation of oxygen and hydrogen, from the dissociation of water, occurs mainly in the perinuclear space, where water, a non-compressible liquid, exerts a positive pressure (push) towards the interior of the cell; but when it reaches the perinuclear space, the liquid water dissociates into its gaseous components (H_2 and O_2), which causes this positive pressure to disappear completely and then a negative pressure or vacuum is formed that attracts the water

into the cell. So, when studying histological sections, we must see cell nuclei as tiny vacuum or suction pumps that continuously attract fluids from the cell's environment into the cell (Figure 3).

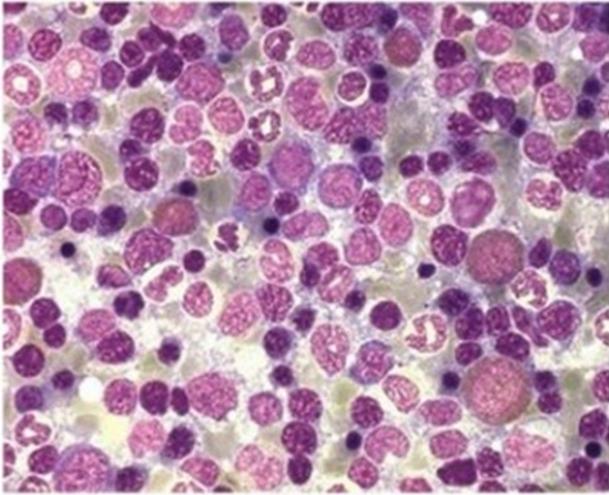


Figure 3: Bone marrow core biopsy, the core biopsy specimen obtained in this procedure provides for analysis of bone marrow architecture and processed for routine H&E slide staining.

Figures 2 and 3 show the absence of blood vessels in the central part of the bone marrow. And despite the absence of such vessels, the central portion of the bone marrow is one of the most metabolically active tissue in the human body. The energy that drives the mini-vacuum pump, which is the basis or principle of the functioning of all cells, is the power of light, just as in plants.

The interior of the bone, that is, the bone marrow, is not totally dark, because although visible light is very scarce, the bone is crossed by non-visible wavelengths, even healthy, living bone is known to fluoresce [10].

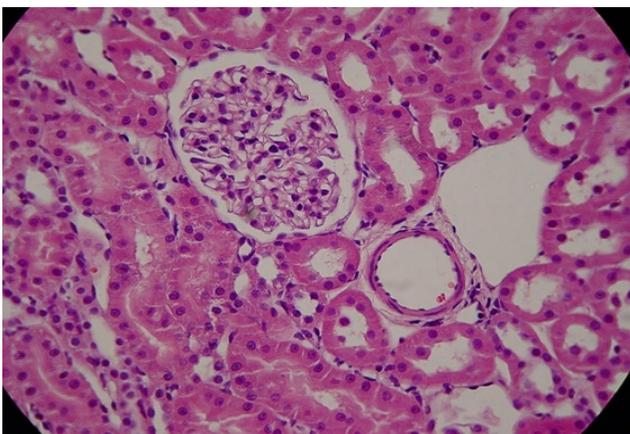


Figure 4: According to Bozzizero's classification, glomerular podocytes are permanent cells that cannot proliferate, but their DNA can replicate, forming polyploid. The formation of polyploids in the human body tends to increase as age advances.

When looking at the histological section of the Wistar rat kidney in Figure 4, stained with H&E, one can see the very numerous mini-suction pumps (cell nuclei) that constitute the histology of the tissue, which we must consider from now on when trying to explain the movement of water and electrolytes through the different types of cells [11]. The mechanism leading to polyploidy seems straightforward, but why it should occur in animal tissues is not at all clear. Especially intriguing is the fact that the megakaryocyte does not begin to make platelets until is octoploid [12].

The Heart and the Unsuspected Capacity of Human Cells to Oxygenate Themselves

High altitude anoxia causes hypertrophy of myocardium [13]. And according to Bozzizero's classification, myocardium is a permanent cell with capacity to duplicate its DNA, forming polyploids. Hypertrophy at high altitude can be explained largely by the increase in the number of red blood cells, supposedly to transport more oxygen from the lung to the tissues, but the lung does not allow oxygen to penetrate. Thereby, anoxia cannot be the cause of pulmonary arteries to constrict [14]. Because oxygen and the lung have nothing to do with each other, we must think that the increase in CO₂ inside the cells and bloodstream is what causes vasoconstriction of the pulmonary arteries.

When the air becomes rarefied, the lung cannot dilute the CO₂ that is continuously produced inside our cells and therefore tends to remain inside the alveolus and rise in the bloodstream. The various intracellular localization molecules that have the intrinsic property of dissociating the molecule from water, e.g. melanin, hemoglobin, myoglobin, cytochrome P 450, etc., [15] are especially sensitive to elevated CO₂ levels, which results in the dissociation of water molecules being altered, resulting in intracellular oxygen levels becoming insufficient to meet the metabolic requirements of the cells.

In fact, all molecules derived from protoporphyrin IX have the intrinsic property of irreversibly dissociating water molecules. Theoretically, it is in the microcirculatory system that oxygen (wrongly believed that comes from lungs) passes to the parenchymal cells by passive diffusion and the cells engage in gas exchange [16]. Which does not make sense, because in proportion the concentration of oxygen inside the cells is about 5 times higher than the concentration of oxygen in the atmosphere, which is inexplicable from passive diffusion.

There are about 5 million RBCs per cubic millimeter of blood which is equivalent to about 250 million RBCs in every drop of blood. The binding oxygen to hemoglobin is a reversible process that occurs rapidly and continuously for maintenance of adequate cellular respiration [17]. Supposedly, Oxygen carriage by blood can exist in two possible forms: as free or dissolved molecules in the plasma and RBC cytoplasm or bound to hemoglobin molecules in the RBCs. Because dissolved oxygen constitutes only 2% of the total oxygen content of blood, but this enormous difference is since hemoglobin, rather than transporting oxygen, generates it

by irreversibly dissociating the water molecules present inside the erythrocyte.

It is already known the oxygen's low solubility in the plasma, which impedes the diffusion of oxygen down its gradient from the RBCs through the surrounding plasma sheath and plasma gaps and into the tissue, and from the alveolar space in the lungs to the bloodstream [18].

The hypoxia of high altitude stimulates ventilation and thereby alkalosis ventilatory [19]. The iso-oxic hypercapnia and hypoxic hypercapnia studies, have shown that the pattern of cerebral blood flow (CBF) and ventilatory responses to hypercapnia were almost identical. CO₂ responses were augmented to a similar degree in both systems by concomitant acute hypoxia and acclimatization to sustained hypoxia [20]. The hypercapnia induces similar responses because the mechanisms that the body possesses to control are very efficient in controlling CO₂ levels in the blood, given its high toxicity. The high mountain disease is more due to the fact that the thinning of the air causes that the volume inspired into the interior of the lung is not sufficient to dilute the CO₂ that is continuously formed in the alveolar space, and this should not be greater than 4% of the inspired volume, otherwise the excess begins to be reabsorbed since the function of carbonic anhydrase is reversible, which causes functional alterations and symptoms derived from hypercapnia. But the problems that our body presents at high altitudes are not due to the lack of oxygen, because even at high altitudes the proportion of oxygen remains between 18 and 21%, but the total number of nitrogen and oxygen molecules is lower so the necessary intra-alveolar dilution of CO₂ (whose number of molecules is the same) is not carried out as it should be. and the proportion of CO₂ in interalveolar air begins to be greater than the normal upper range limit of 4%.

Conversely, the pattern of CBF and ventilatory responses to hypoxia were markedly different. Ventilation showed the well-known increase with acute hypoxia and a progressive decline in absolute value over 25 min of sustained hypoxia. When interpreting the previous paragraph taking into account that the primary function of the lung is to expel CO₂, but that it does not absorb oxygen in the slightest, since each cell of our body produces it inside it, we have that the elevation of CO₂ is the result of the decrease in the proportion of oxygen (O₂) in the inspired air, and although the proportion of nitrogen (N₂) is maintained, this combination in the inspired and already rarefied air, cannot dissolve the CO₂ adequately so that the lung can expel it with minimal or no reabsorption, and when the levels of CO₂ in the intra-alveolar space rise, it begins to be reabsorbed because the function of carbonic anhydrase is both ways, which is consistent with respiratory alkalosis and therefore the appearance of symptoms.

Our body's response to acute hypoxia is more of a defensive reaction, such as when a badly placed individual is pushed, and the person responds by maintaining the vertical. Something like this happens when the parameters of CO₂ are abruptly modified, as

the body responds by generating oxygen (through the dissociation of water) which restores the metabolic balance, preparing the individual to fight or run with acclimatization to hypoxia for 2 days, the absolute values of ventilation and O₂ sensitivity increased. By contrast, O₂ sensitivity of CBF or its absolute value did not change during sustained hypoxia for up to 2 days. The results suggest a common or integrated control mechanism for CBF and ventilation by CO₂ but different mechanisms of O₂ sensitivity and plasticity between the systems. Ventilatory and cerebrovascular responses were the same for all subjects irrespective of acute mountain sickness (AMS) symptoms.

Summarizing: Ventilatory and cerebrovascular hypercapnic response patterns show similar plasticity in CO₂ sensitivity following hypoxic acclimatization, suggesting a quite established integrated control mechanism developed over billions of years of evolution.

Conversely, ventilatory and cerebrovascular hypoxic responses differ, because our systems respond to CO₂ more than oxygen. Ventilation initially increases but adapts with prolonged hypoxia, this is an hypoxic ventilatory decline compatible with a defense mechanism, which characteristically tends to become extinct; and ventilatory sensitivity increases following acclimatization, because homeostasis has been restored but is not completely normal.

In contrast, cerebral blood flow hypoxic sensitivity remains constant over a range of hypoxic stimuli, with no cerebrovascular acclimatization to sustained hypoxia, suggesting different mechanisms for O₂ sensitivity in the two systems. What must be rethought but now considering that the oxygen that our body requires for its metabolism does not come from the atmosphere but from the interior of each cell, which generates it by dissociating the intracellular water molecules [21].

The paradoxical reaction of the pulmonary arteries (vasoconstriction) is congruent if the process is analyzed from CO₂ and not from atmospheric oxygen. We must bear in mind that both the generation of oxygen (O₂) as well as that of CO₂ are strict intracellular processes.

Effects of Light Exposure on the Release of Oxygen from Hemoglobin in a Red Blood Cell Suspension [22]

Measurement of oxygen transport in microcirculation has shown that about ten times more oxygen appears to leave the blood of arterioles than can be accounted for by diffusion. One possibility to explain the high oxygen loss is an increased release of oxygen due to exposure of blood to light.

The change of PO₂ in the sample chamber during light exposure was an average of 1.60 ± 0.9 mmHg (SEM). The contribution of photo-dissociation of oxygen from oxygenated hemoglobin molecules to the observed oxygen loss per RBC can account for only about 0.01% of the observed *in vivo* results. Therefore, light-associated oxygen release is negligible. Which was to be

expected, because just as atmospheric oxygen cannot cross the mainly aqueous barriers and various cell membranes to enter our body (much less by simple diffusion), it cannot leave either, since the barriers are the same in both directions. Therefore, the significant change in PO_2 occurs inside each cell, which is not easy to measure, but the fact that a detectable change occurs in a suspension of red blood cells, indicates that the greater the amount of light, the greater the dissociation of water at the intracellular level. If light had nothing to do with oxygen levels, the change detected would have to be zero.

Just as the lung cannot absorb oxygen from the atmosphere, because this gas is repelled by water, neither can the oxygen that is generated inside the cells, because it cannot cross the cell membranes in any way.

Cells get rid of the excess oxygen generated by the dissociation of water, solubilizing it by adding a carbon atom to form CO_2 , which is almost 70 times more soluble than oxygen, allowing it to leave the interior of the cells. The origin of the word oxygen is "acid-forming", and the acid they form is carbonic acid or CO_2

On average a normal blood concentration of hemoglobin is about 15 g/100 ml of whole blood and each molecule of hemoglobin is able to carry 1.34 ml O_2 /g Hb. Therefore, the total oxygen carrying capacity of human blood is 20 ml O_2 /dl [23]. But oxygen is an extremely toxic element, and the stated concentrations of 1.34 ml O_2 /g Hb and 20 ml O_2 /dL, are very high and would destroy hemoglobin molecules quickly, it is more congruent to think that they generate it continuously through the dissociation of water molecules, located inside the cells.

The dissociation curve of hemoglobin depends more on the amount of light and CO_2 levels than on its affinity for oxygen (if any), since hemoglobin is destroyed by oxygen, just like chlorophyll, hence it expels it as fast as they can.

The only intracellularly localized molecule that tolerates oxygen toxicity and is therefore capable of dissociating and reforming the water molecule is melanin.

Melanin forms bubbles when illuminated

Wolfgang Wöttinger, in his book *Senile Retinoschisis*, published in 1978 by George Thieme Publishers Stuttgart, Germany, in the page 4, describes the formation of bubbles when illuminating a tissue with a high amount of melanin such as the retina and choroid, although the author attributed it to the heating of the alcohol he used to preserve the tissues.

The paragraph in question:

"A razor blade was used to open the eye in the horizontal plane, beginning just over the point of exit of the optic nerve. The lens was left intact, and the two halves of the globe were separated by cutting the zonules. The interior of the eye was examined with a stereo-dissecting microscope (American Optical Company) with

halogen illumination. Important findings were recorded by means of sketch and photographed with a Tessar (Zeiss) on the Zeiss slit lamp. Since the information obtained from the photographs was insufficient, another method was developed using simultaneous stereo pictures taken with the Zeiss photo slit lamp. Each half of the eye was put in an optical glass cuvette (8 x 8 x 4 cm) filled with 50 % alcohol. A small glass plate was held at an angle to press each half of the eye onto the glass wall. A small support fixed onto the chin rest enabled the cuvette to be positioned at the right height. As a result, photographs could be taken from various angles and with different illumination, Small bubbles produced in the alcohol by the heat of the light sometimes made photography difficult. It was risky to remove these bubbles because of the danger of damaging the schisis."

To conclude, I would like to mention the well-known phenomenon of "freezing" sharks when the ventral part is placed above.

This response is technically known as "tonic immobility." When it strikes, there's usually nothing that the animal affected by it can do. Although it might seem like possums are "playing dead" by choice, the animals have no control over when the immobility hits them. It's the same for sharks, too. That said, there is a bit more of a controlled method to activate the tonic immobility in sharks, as they only need to be turned upside down.

For some reason, when a shark is turned upside down, it just freezes. It doesn't move. It doesn't bite or fight back. It's one of the best ways for scientists to handle sharks safely when studying them. But why do they do it? And does it help them in any way? (Figure 5).



Figure 5: The lower half of the shark contains less melanin than the upper part, so when they are turned upside down, the lower amount of melanin in the ventral part of the shark does not generate enough oxygen and hydrogen (from the dissociation of water) to support the complex functions inherent to the shark's life. so, suddenly, we "anesthetize" him. The well-known freezing reaction of sharks when they are turned upside down is explained congruently by the difference in the amount of pigment between the ventral and dorsal parts of this aquatic organism.

The greater the amount of melanin, the fewer blood vessels and vice versa

It is one of the key observations we made during the observational study, which we conducted from 1990 to 2002. Our working hypothesis was to correlate the morphological characteristics of the blood vessels entering and exiting the eyeball through the optic nerve (Figure 6).

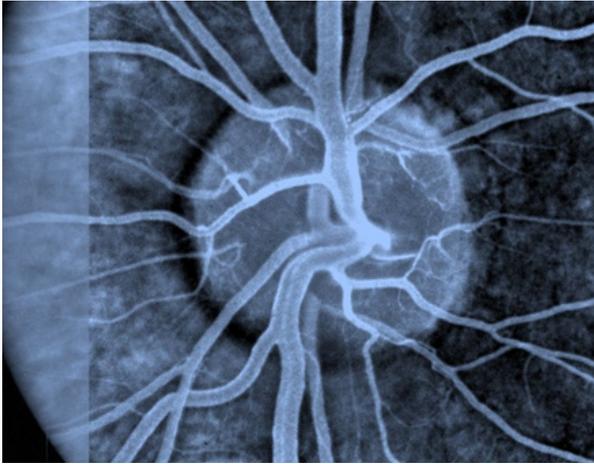


Figure 6: Photograph of the left optic nerve. Since our working hypothesis was about the vessels of the optic nerve, the magnifications we required were significant. This allowed us to appreciate important details.

The observation at the fundus level of the inverse relationship between the amount of melanin and the number and thickness of blood vessels is fulfilled at the macroscopic level (Figure 7).



Figure 7: The greater the amount of melanin, the fewer the number of blood vessels, and vice versa. Note the number of blood vessels in the arms of two famous tennis players.

Conclusion

Cell biology, human physiology, as well as medical practice, will gradually and significantly change, as the prevailing (wrong) dogma that our body takes oxygen from the atmosphere disappears and is replaced by the new paradigm that every cell in our body has molecules that allow it to produce its own oxygen. That is, they oxygenate themselves from the dissociation of the molecule from water, like plants.

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