Reading physiology slowly

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Abstract:
Learning physiology requires unhurried time to understand and deepen the appreciation for whole-body homeostatic mechanisms. To read slowly is to read thinkingly, to read and imagine conceptually, the integrative workings of multiorgan physiology. It is reading beyond descriptive facts, to see the priorities of diverse organ responses during adaptation to challenges to maintain homeostasis. Teachers have a key role to inculcate slow physiologic reading in their classes with students.

Keywords:
Learning, physiology, reading, teaching, thinking

In our fast-paced, information downloading, and deleting generation, students can be inundated with a huge amount of knowledge from different authors and sources. It can be overwhelming to surf and sieve through the large volume of easily available texts. For students who have limited time outside their classes to diligently plow through even their preferred physiology text, it is quite natural to fall back on relying basically on the PowerPoint notes to revise when preparing for tests.

Students can read fast and superficially, just their lecture notes, and end up absorbing less “Physionutrients.” Absorption of knowledge, I would like to think is also an active process. Passive, rapid browsing through facts will often result in superficial benefits. The retention of essential knowledge will likely also be transient. Working through complex, interrelated mechanisms is like the repeated, energy-consuming piling of foundation for the construction of high-rise buildings.

Mere memorizing of PowerPoint information without focused thinking and a deeper understanding of the physiology will end up with the student powerless and slow to apply concepts to clinical settings. This article shares some observations and thoughts to help students learn and teachers to teach Physiology “slowly,” focusing on examples from the cardiovascular system.

How to Read Slowly, Consider Your Heart

Consider slowly the blood pressure (BP) equation.

All physiology students can state, almost as easily as the multiplication table, that BP is the product of cardiac output (CO) and total peripheral resistance (TPR), i.e., BP = CO × TPR.

Several essential instructions are necessary to unpack this seemingly straightforward cardiovascular relationship.

1. First, this is a physiological formula and not strictly a mathematical equation. The CO and TPR factors are determinants of the mean arterial BP. That said, the cardiovascular mechanism must be understood and applied in a right-to-left direction. To make the point, we cannot say that when the BP increases, the CO will also increase (incorrect left to right direction). Physiologically, an elevated BP represents a higher afterload and...
with time the myocardium can be weakened by the increased cardiac workload. Cardiac pump activity will be compromised eventually

2. When we slowly move to the pulmonary hemodynamics, the same BP equation (BP = CO × TPR) is instead unpacked in a different unique way. The pulmonary blood vessels are much more compliant, and intravascular mechanical pressure can produce vasodilation (in the systemic circulation, vasodilation generally occurs when there is diminished vasoconstrictor sympathetic activity to the arterioles). This means that in the lungs, increased pulmonary arterial pressure can directly vasodilate and lower the pulmonary vascular resistance. In other words, the equation has now proceeded in a left-to-right direction

3. Another feature to slowly appreciate is the systemic term, TPR. The students will appreciate that the “total” does not mean the involvement of all arterioles when the TPR changes during compensations for fluctuations in BP. The baroreflex-activated increase or decrease in sympathetic vasoconstrictor action does not functionally affect the arterioles in the cerebral and coronary circulation. This is critical to the homeostatic achievement of maintaining adequate perfusion to the brain and the heart when the driving BP changes.

**Slowly, My Starling**

The textbook description of the intrinsic ventricular contractile response called Starling’s law of the heart is well known among students. Often, teachers might have observed that the students’ appreciation of this myocardial property is limited to the relationship between the end-diastolic volume and the stroke volume in a single ventricle.

For students who ponder a little more, it will be seen that Starling’s mechanism applies to both the left and right ventricles regardless of the differences in intraventricular pressures during a cardiac cycle. The less muscular right ventricle pumps a CO that matches the left ventricular CO.

The right ventricular pump is in series with the left cardiac pump with the pulmonary circulation sandwiched in between. The important potential problem should then be realized in the event of unequal CO from the two myocardial pumping machines working in series.

Understanding would then be extended to the critical maintenance over time of equal right and left CO in order that there is no vascular congestion in either the pulmonary circulation or in the peripheral systemic circulation. Beat by beat variations in the two-stroke volumes do occur, and this is where Starling’s law of the heart takes physiological place so that no homeostatic balance is broken and either pulmonary edema or peripheral edema develops.

Over time, the venous return into the left or right ventricular chamber will be equal to the CO ejected by the left or right side, respectively…O my Starling, all is well.

**The Heart Defined Slowly**

Students can well appreciate that the myocardium has intrinsic properties to autoregulate its own blood perfusion. Read slowly and noticed that the strict definition of autoregulation by the heart (brain and kidneys also) describes the ability to maintain a relatively constant blood flow when there are fluctuations in the driving arterial BP.

When the heart is more active during physical activity, it no longer “autoregulates” its coronary blood flow. It “automatically” increases the blood flow parallel to the increased cardiac work of beating faster and stronger. The intrinsic autoregulation of a constant resting coronary blood flow operates via the myogenic and also the metabolite mechanisms. The hyperemia in the active heart is due to a large amount of metabolite vasodilating factors, including myocardial hypoxia, local hypercapnia, and adenosine. Tachycardia and active hyperemia are always associated.

Hence, we could summarize by saying that coronary autoregulation of blood flow is automatic and active hyperemia to sustain the greater metabolic demands of the pumping heart is also an automatic physiology event.

**Slowly, Appreciate the Concurrent Central and Local Controls**

The big picture of cardiovascular function in the whole body needs to be seen when understanding the mechanisms that are triggered concurrently at both the central regulatory centers and at the local tissues. During hypovolemia, there is the need to both maintain soonest adequate tissue perfusion and to eventually restore volume. Central control neurons located in the brain stem are activated by baroreceptor afferent impulses to increase the TPR (BP = CO × TPR) in an effort to normalize the hypotension. At the same time, in the brain and the heart, which do not contribute to the TPR changes, both arteriolar myogenic responses and local metabolite vasodilators improve the tissue perfusion to complement the central action of increasing TPR to raise the BP. For the students who do not go beyond regurgitating, BP = CO × TPR to slowly appreciate the collaborative central/local compensations, this
beautifully orchestrated physiology of the central neuronal conductor and the local tissue playing the right timely tune will be missed.

**Slowly, “A Lung” with the Heart**

The cellular customer is always right! The integrated purpose of the cardiovascular and respiratory functions is to supply oxygen (O₂) and energy substrates to the tissues. For oxygenation, we might call the rate of O₂ delivery to the cells as rate of tissue oxygenation. The units would then be ml O₂ per min (ml O₂/min).

At the alveoli, the diffusion of O₂ down its alveolar-capillary partial pressure gradient oxygenates the pulmonary blood and saturates the arterial blood with an O₂ content of about 20 ml% (per deciliter). Lung oxygenation (ml O₂/min) is not synonymous as lung ventilation (ml air/min).

For this well-oxygenated blood to benefit the cells, blood must flow well. Stagnant hypoxia is caused by poor tissue blood flow, even though the oxygenation in the lungs is normal.

Thus, the rate of normal O₂ delivery to the cells is given by the arterial O₂ content multiply by the CO. At rest, around 25% of the arterial oxygen content is extracted by the tissues.

One primary function of the lungs is also to remove the metabolic carbon dioxide (CO₂). Accumulation of CO₂ will acidify the blood, and acidosis desensitizes the neurons when in excess. The rate of CO₂ removal by the lungs can be worked out as simply the venous CO₂ content multiply by the venous return (equal to the CO).

**Slowly, Directly, and Indirectly Talk to the Kids**

The cardiorenal physiology is an integrated homeostatic phenomenon in the control of blood volume and pressure.[9] Interestingly, the pumping function of the heart is monitored by the kidneys. A decreased CO can result from a weak myocardium although the blood volume is normal. Hypovolemia by reducing the ventricle filling also tends to reduce the CO.

The mechanoreceptors located at the preglomerular afferent arteriole sense any changes in the renal arterial pressure. A decreased CO for whatever primary reasons will be sensed directly by these intrarenal baroreceptors. In response, the juxtaglomerular (JG) endocrine cells in the afferent arteriole released renin into the circulation. Renin, a hormone enzyme, triggers the intravascular chemical reactions that lead to antinatriuretic, compensatory activities, and BP-promoting actions.

If students do not give much thought to integrating organ functions, the cardiovascular events will be learned as a segmented part of what should be a homeostatic global event. Should the CO be lessened, the resulting hypotension will be detected by both the carotid/aortic sinus to produce a baroreflex. Indirectly, the activated sympathetic discharge will among its spectrum of actions also stimulate the JG cells to secreted renin. This indirect neural input to the JG cells enhances the direct effect by renal arterial hypotension on the JG cells.

The increased renal sympathetic actions on both the afferent/efferent arterioles also serve to raise the TPR; a determinant of arterial BP. Renal sympathetic action is also antinatriuretic and conserves sodium by reducing filtered sodium load and increasing tubular reabsorption of sodium.

**Teachers Who Teach Slowly Produce Students Who Read Slowly**

My younger colleagues in the department are frustrated by much time devoted to maintaining the external image of the faculty. By this is meant, the time spent on keeping up with the publication mill in order to contribute to hopefully a better university ranking. On top of this, there is regular quality assurance exercise, and most of this audit evaluates the management of department teaching rather than the actual engagement of the students and how deeply students have learned.

In it, any wonder that my young colleagues have much less unhurried time to think through and prepare their lectures. Of course, the move to self-directed learning gives some relieve for better class teaching. My observation is that most students do not do self-discovery learning well and far better to guide them in what I call “directed self-learning.” This includes teaching them to be able to sift out and discern essentials of any physiological events and not be overwhelmed by the fine details of cellular mechanisms in understanding whole-body homeostatic physiology. Instructing students to chew and digest physiology slowly to assimilate more usable information for active application in clinical settings.

Teaching slowly will involve meeting the objectives we have described above to help students to learn conceptually, integratively, and definitively (definitions).[3] “The student is not above his teacher” was a wise accurate statement made.[10] Teachers can help students to see pattern and principles that across different organ systems.[4] And naturally, the teacher must see these shared mechanisms first to reveal and hopefully delight
students learning physiology. A quick coverage of descriptive facts and tables of stimulatory/inhibitory factors during a class seldom attract the student to the "physiosymmetry" and beauty of homeostatic events.

Perhaps time should be allowed and allocated to have physiology reading sessions. Take a comprehensive good physiology book and as a few selected students are asked to read slowly, questions can be posed along the "Read." For example, when a respiratory control text is read, a question can be asked: "Is increased alveolar ventilation more important to oxygenate or "de-carbodioxidate" (remove CO\textsubscript{2}) the blood?" "Is CO\textsubscript{2} merely a metabolic product in Physiology?" [Table 1].

The title of this article is inspired by the book by Sire\textsuperscript{[5]} encouraging students to read well and understand the bigger picture and perspective into which the contents of what they read fit in.

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Conflicts of interest
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| Function                  | Event                                                                 |
|---------------------------|----------------------------------------------------------------------|
| Vasodilator               | Autoregulation of cerebral and coronary blood flow                   |
| Vasodilator               | Increases skeletal blood flow during physical activity               |
| Bohr's effect enhancer    | Promotes O\textsubscript{2} unloading in tissues                     |
| Regulation of blood pH    | A factor in major ECF buffer system with bicarbonate ions            |
| Control of bronchiole diameter | Stimulus for bronchial smooth muscle responses in ventilation/perfusion matching |

ECF=Extracellular fluid