MEDICAL EDUCATION/CME

Using Portmanteau to Create Meanings in New Physiology Words

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Abstract

Words and definitions frame our understanding. In learning physiology, precise words used that explain specific physiology help to conceptualize essential homeostatic events in the body. The combination of two keywords into one (portmanteau) can effectively aid the appreciation of physiological mechanisms. For example, the portmanteau "natriuresis" expresses the dual responses in the urinary excretion of excess water and sodium during renal compensation for both positive sodium and positive water status. Physiology teachers can create their own portmanteau to engage their classes. Students can also try integrating words to make physiology portmanteau to stimulate and summarize their own understanding.

Keywords: Definitions, Mechanisms, Physiology, Portmanteau, Understanding.

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Introduction

When a young person is nagged by her parents to study diligently for her tests, a common exasperated reply could be "chillax!." Here is a new generation portmanteau formed from "chill" and "relax." A recent discovery of the brain glial cells-associated drainage of the brain interstitial space was named with a portmanteau "glymphatics."[1] The word "portmanteau" was used by Humpty Dumpty explaining to Alice in Lewis Carroll's Through the Looking Glass.[2]

Here are some samples of how a carefully coined portmanteau can be useful for both educators and students in teaching and learning physiology, respectively.

"Osmotonic" (Osmolarity and Tonicity)

Osmolarity is a physicochemical parameter while tonicity is defined with respect to effects on water flux across borders including cell membranes and capillary walls. The value of osmolarity alone does not tell us the tonicity of a solution or fluid.

A 300-mOsm/L solution can be isotonic or hypotonic. A 300-mOsm/L solution is isotonic when all the 300-mOsm/L solutes in the solution are "osmotonic." We coined this "osmotonic" portmanteau to echo what is in the textbook, the definition of an isotonic solution.

"An isotonic solution will contain 300 mOsm/L of non-penetrating solutes, regardless of the osmotic concentration of penetrating solutes."[3]

So, a solution of 0.15-M sodium chloride will contain 300-mOsm/L of *osmotonic* solute and will be isotonic. Both the sodium cation and the chloride anion are non-penetrating in a laboratory experiment with red cells. *In vivo*, sodium and its accompanying anions are the predominant "osmotonic" solutes in the extracellular fluid (ECF). A change in ECF sodium concentration is the main determinant of the net water flux between the intracellular and extracellular spaces.

At the capillary, the electrolytes are freely diffusible across the capillary wall and are then not "osmotonic," but not osmoactive or do not exert an effective osmotic pressure. At the capillary, the plasma proteins restricted in the vascular compartment become the principal "osmotonic" solute. The osmotic pressure exerted by the "osmotonic" proteins is also named the oncotic or colloid osmotic pressure and is an essential Starlings' force that determines the distribution of fluid between the plasma and the interstitial fluid.

Thus, the "osmotonic" solutes are "non-penetrating," osmoactive, and exert an osmotic pressure at the separating interface.

"Sodiumolarity" (Sodium and Osmolarity)

Changes in ECF sodium concentration are accompanied by parallel changes in the ECF osmolarity. When the body normalizes the ECF sodium concentration, the ECF osmolarity is also restored. The detection of ECF sodium concentration thus also means the detection of ECF osmolarity. The ECF sodium concentration sensors detect ECF osmolarity changes. The hypothalamic osmoreceptors are also the hypothalamic sodium concentration sensors.

With regards to water balance, changes in the water balance alter the ECF sodium concentration as well as the ECF osmolarity. When the body normalizes the water balance, it also normalizes the ECF "sodiumolarity."

In negative water balance (dehydration), there is an increased "sodiumolarity." Compensation occurs by homeostatic increased

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reabsorption of water by the kidneys, with the production of concentrated urine. The water balance is restored and so is the "sodiumolarity."

Thus, the anti-diuretic hormone (ADH) from the hypothalamus/pituitary regulates the ECF osmolarity, the ECF sodium concentration (sodiumolarity), and the water balance. Note that when a large volume of water is consumed (positive water balance), the "sodiumolarity" is decreased, but the total body sodium or sodium balance is unchanged.[4]

"Potenssium" (Potential and Potassium)

"The potassium cation has great Potential." The resting membrane of excitable cells such as neuron and muscle fibers are determined by the trans-membrane potassium gradient. There is a steep potassium gradient with the intracellular concentration at least 30 times higher than in the ECF. The ECF potassium is homeostatically regulated closely at around 4 mmoL/L. Hypokalemia (low potassium level in plasma, which is a component of ECF) hyperpolarizes the neuron and muscle cells. The membrane excitability is reduced. Hypokalemia is then associated with symptoms of muscle weakness. The kidneys are the key controller of the ECF potassium level. You might say the kidneys have an exciting role in maintaining the normal responsiveness of excitable cells.

Importantly, in hyperkalemia, cardiac function is affected. The increased cardiac membrane excitability is not a desirable event since the heart is depolarized normally in an ordered sequence of impulse transmission *via* specific conducting fibers from the sinoatrial (SA) node to the ventricles. Irregular excitability of non-conducting myocardial fibers would be undesirable. Critically, the initial stimulation of cardiac muscles to a partial depolarization by the hyperkalemia subsequently can inactivate the voltage-gated sodium channels. The heart can cease to beat and contract.

The kidneys respond to the adrenal mineralocorticoid aldosterone by secreting potassium. Hyperkalemia is a direct, rapid stimulus for the release of aldosterone from the adrenal cortex. The potassium sensors are located on the adrenal glomerulosa cells that secrete aldosterone. You can say that kidneys have the potential or "potenssium" to keep our hearts beating.

The pancreatic insulin increases cellular uptake of potassium by stimulating cell membrane Na/K ATPase. Postprandial hyperkalemia thus also stimulates insulin release besides the hyperglycemia following a meal. A combi administration of insulin/glucose is used to manage serious hyperkalemia.

Potenssium, the exciting potassium cation with a wonderful potential.

"Sodinflux" (Sodium and Influx), "Chlorinflux" and "Potenflux"

Physiology is an electrifying subject. Many physiological processes involve the movement of charges, both cations and anions, across cell membranes. The influx and efflux of ions trigger physiological events. The direction of the ionic flux is predictable as the passive diffusion follows the transmembrane chemical gradient. For example, in the case of sodium cation, when it occurs, it is always sodium influx into the cells from the ECF since in the ECF the sodium concentration is around 10 times higher than inside the cells.

Hence the coined term "sodinflux." In excitable cells such as nerve and muscle, "sodinflux" depolarizes the cells.

For the other ions, here are the new fusion words:

- *Calcinflux*: ECF calcium can be about 100 times higher than cytosolic calcium. In cardiac and smooth muscles, "calcinflux" activates the muscle contraction.
- *Chlorinflux*: At some inhibitory synapses, "chlorinflux" of chloride anions into the postsynaptic neurons hyperpolarizes it, reducing the membrane excitability.

For potassium cations, it is the major intracellular electrolyte. "This ion sleeps inside" (a potassium pun adapted from the popular song chorus "The lion sleeps tonight"). The transmembrane potassium gradient is directed outward since the potassium concentration is at least 30 times higher inside the cells. Thus, when potassium ion moves or diffuses as the membrane becomes suddenly permeable, it is always an efflux, a "potefflux."

At synapses, "potefflux" at the postsynaptic neuron, after an inhibitory neurotransmitter binds, generates an inhibitory postsynaptic potential. The action potential that arrives at the presynaptic terminal has a repolarization phase as for all action potentials. This phase is due to "potefflux" to normalize the membrane potential after depolarization by "sodinflux."

"Compliancitance" (Compliance and Capacitance)

A physiological structure like the blood vessels or the lungs is stretchable or distensible. The more easily the structure is stretched by blood or air pressure, the higher would be its compliance; vascular compliance or lung compliance, respectively. A more compliant blood vessel/lung will have a greater volume or capacitance for the same distending pressure.

We use the portmanteau "compliancitance" to link the two related meanings.

Venoconstriction by sympathetic action or venous compression by skeletal muscle contraction reduces the venous "compliancitance."[5] The word vasoconstriction is used with reference to the arteriolar constriction when the vascular resistance is increased by sympathetic action. For the veins, the venous resistance change is a minor event compared to the change in the "compliancitance." A decreased venous "compliancitance" increases venous return, ventricular filling, and better cardiac output.

For the lungs, the compliance is the change in lung volume per change in transpulmonary distending pressure at the alveolar wall. In the pathophysiology of emphysema. the elastic tissues are diseased and the normal lung elastic recoil is diminished. The abnormal increased compliance or "compliancitance" results in an enlarged functional residual capacity.

"Dialasticoil" Diastolic Pump (Diastole and Arterial Elastic Recoil)

The cardiac pump is a rhythmic pump, ejecting in systolic cycles from the fixed volume of circulating blood. The ventricles are refilled with blood during diastole when the intra-ventricular pressure is near 0 mm Hg, and yet the blood flow that perfuses the peripheral tissues is not interrupted but continues optimally during diastolic relaxation of the ventricles.

What then sustains the continuous uninterrupted blood flow during diastole? I shall call this the "diastolic pump." This diastolic blood-driving pressure is due to the elastic recoil of the aorta and large arteries that are stretched during systole. If the blood vessel

conduits are rigid structures like water pipes, the blood flow will be interrupted during diastole.[6]

The fusion of three words diastole, elasticity, and recoil into "dialasticoil" describes this complementary diastolic pump when the ventricles are not contracting. One main factor that affects diastolic blood pressure (DBP) is total peripheral resistance (TPR). Imagine an elastic balloon, filled up, and then air is released by the elastic recoil of the balloon through the narrow opening. This opening can model the TPR. Therefore, when the TPR is increased the DBP tends to be raised and when the TPR is decreased, this will lower the DBP.

Apply this to the scenario during exercise. The extensive vasodilation in the skeletal muscles for metabolism and skin for thermoregulation will decrease the TPR. The systolic blood pressure (SBP) during exercise is elevated by the greater cardiac stroke volume. A greater arterial stretch should increase the DBP in parallel. However, the decreased TPR during physical activity has the opposite effect on DBP. So, the DBP does not rise as significantly as the SBP during exercise.

Another example of this TPR/DBP relationship is seen in hyperthyroidism. Thyroid hormones increase cardiac output *via* a synergistic action with the sympatho–medullary system on the myocardium. The tachycardia and increased cardiac contractility raise the SBP. Thyroid hormones increase the metabolic rate. This leads to peripheral vasodilation for heightened metabolism and also for thermoregulation. The decreased TPR lowers the DBP. Thus, in hyperthyroidism, the pulse pressure (SBP–DBP) is bigger.

So thankfully you can relax and recline when your heart also relaxes during diastole because there is a "diastolic pump" that ensures your brain continues to be supplied by uninterrupted life-giving blood. Amen!

"Proncotic Pressure" (Proteins and Oncotic Pressure)

The oncotic or colloid osmotic pressure is dependent on the plasma protein concentration. Hence, the fusion portmanteau "proncotic pressure" or "protoncotic" pressure. The osmolarity of plasma proteins is only a tiny fraction of the total blood osmolarity. However, at the microcirculation, when all the electrolytes are freely diffusible, the non-penetrating plasma proteins become the dominant osmoactive solutes that exert the effective osmotic pressure; the "proncotic pressure."

This capillary "proncotic pressure" is one of the four Starling's forces that direct the capillary fluid dynamics. Here are two examples of this applied "proncotic pressure":

- Proteinuria lowers the "proncotic pressure" and peripheral edema develops as net filtration into the interstitial fluid is increased. The "proncotic pressure" is a reabsorptive force at the microcirculation.
- During hypovolemia, the capillary hydrostatic pressure is reduced. The decreased arterial pressure as well as the arteriolar baro–reflex vasoconstriction drops the capillary hydrostatic pressure. The "proncotic pressure" is, however, unchanged. There is then a higher net reabsorptive force and a transcapillary shift of fluid from interstitial space helps to preserve the vascular volume.

The "proncotic pressures" at the glomerulus and the peritubular capillary are functionally unique.

- The glomerular capillary also has the "proncotic pressure." The "proncotic pressure" in the glomerulus increases along the capillary since at rest, the renal filtration fraction is a significant 0.2. The plasma proteins are more concentrated toward the postglomerular efferent end of the capillary.
- The peritubular capillaries are in series with the glomerulus and the high resistance efferent arteriole produces a low peritubular hydrostatic pressure. The end-glomerular, raised "proncotic pressure" becomes the peritubular "proncotic pressure." Thus, along the peritubular capillary, the "proncotic pressure" is higher than the hydrostatic pressure. The net reabsorptive force serves the essential role of the peritubular capillary in renal tubular reabsorption of fluid.[7]

When we consider the overall function of the intestines in recycled reabsorption of a huge volume of digestive aqueous fluid besides the dietary fluid, reabsorption of water by the intestinal enterocytes is greatly favored by a net reabsorptive force at the capillaries that supply the gut. Certainly, the "proncotic pressure" is the essential capillary force involved.

In the interstitial fluid, there is also a smaller "proncotic pressure." The interstitial "proncotic pressure" is kept low to prevent the accumulation of fluid. The lymphatic drainage has a dual function in keeping both the hydrostatic and "proncotic pressures" in the interstitial space at a minimum value.

"Floxigenation" (Bloodflow and Oxygenation)

This new term "floxigenation" reminds the students of the integrated role of the cardio-respiratory mechanism to provide adequate oxygen (O_2) supply (mL O_2/time) to the cells. The delivery rate of O_2/min to the cells is calculated by the product of "bloodflow" (cardiac output) and the "blood oxygen" content (lung oxygenation).

The well-oxygenated blood from alveolar–capillary exchange must flow to the tissues. When circulatory flow is diminished as occurs in cardiac pump failure, the O_2 delivery is reduced. The "floxigenation" is decreased. The cells do not receive adequate O_2 supply. This decreased "floxigenation" is named "stagnant hypoxia."

In "histotoxic hypoxia," the "floxigenation" can be normal but the cells are metabolically inhibited and cannot use the O_2. The blood flow and the arterial blood O_2 content are normal. The venous partial pressure of O_2 (PO_2) and O_2 content are characteristically high in "histotoxic hypoxia."[5]

"Floxigenation" is decreased if the blood flow is normal but the arterial blood O_2 content is low. In this case, the reduced arterial blood O_2 content can occur in two ways:

- First, the PO_2 is decreased, and hence the hemoglobin (Hb)-O_2 saturation. Any situation that is associated with a lower PO_2 is named "hypoxic hypoxia".
- Second, the arterial blood O_2 content is low because the Hb concentration is low, or the Hb binding for O_2 is reduced (e.g., competitively by carbon monoxide or genetically by mutation). The total arterial blood O_2 content is reduced and this Hb-associated hypoxia is named "anemic hypoxia." Note that the dissolved PO_2 is unchanged.

Let us humorously apply "floxigenation" to physiology educators! Our constant role as teachers is to ensure that the physiology knowledge that we have (O_2 content) is well communicated (flow) to our students. We are responsible to have sufficient Physiology

Content (our own reading and understanding). Then also as well, we improve and find the best teaching delivery mode for our students.

"Saturaffinity" (Saturation and Affinity)

The Hb-O_2 saturation and the Hb-O_2 affinity are the focus here. Consider this statement:

The Hb-O_2 saturation is determined by the PO_2.

In the oxy-Hb dissociation graph, the *x*-axis is PO_2 (in mm Hg) and the *y*-axis, the Hb-O_2 saturation (in %). Note that the PO_2 changes itself does not alter the natural affinity of the Hb for O_2.[8]

Changes in affinity are indicated when there is a shift of the PO_2/Hb-O_2 saturation curve to the right or to the left. Obviously, when the Hb-O_2 affinity is affected, it will also change in parallel the Hb-O_2 saturation for any given PO_2. For example, the normal Hb-O_2 saturation at PO_2 of 40 mm Hg is 75%. If any factor reduces the Hb-O_2 affinity, then at the same PO_2 of 40 mm Hg, the Hb-O_2 saturation can decrease to 70%. This benefits the cells which will download more O_2. Therefore, any factor that changes the affinity of Hb-O_2 will result in a corresponding change in Hb-O_2 saturation. This is the use of the portmanteau "saturaffinity."

In active muscles, changes in the local factors decrease the "saturaffinity" of Hb-O_2. During exercise, the lower tissue pH, higher tissue PCO_2, and warmer muscle tissue all decrease the Hb-O_2 "saturaffinity." Bohr's effect is the reduction of Hb-O_2 "saturaffinity" by lower pH and higher PCO_2. The release of more O_2 from the Hb is enhanced with the decreased "saturaffinity" of Hb-O_2.

The rise in the red cell metabolite, diphosphoglycerate in hypoxic conditions also helps to compensate by reducing the "saturaffinity" of Hb-O_2. Note that when the concentration of normal Hb is decreased as in iron deficiency anemia, the blood O_2 content is decreased but the natural "saturaffinity" of the Hb-O_2 is unchanged.

When we teach our students, what are the factors that hopefully will decrease the saturaffinity of the physiology knowledge that we wish to impart or download to our students?!

"Hypoxcell" (Hypoxia and Cell)

Hypoxia is defined by the cell. It is a "cellfish" perspective. The cells in the peripheral tissues are the consumer targets of cardio-respiratory functions. For O_2, the resting tissue O_2 consumption is about 250 mL O_2/min. This is around 25% of the total O_2/min delivery to the cells. The total cellular O_2 supply is determined and calculated from the following: Arterial blood O_2 content × Cardiac output, that is, 200 mL O_2/L arterial blood × 5 L/min, giving 1000 mL O_2/min.

The cells can experience a lack of O_2 or hypoxia ("hypoxcell") even when the arterial blood content is normal.[9] There are two scenarios of this type of "hypoxcell."

- One is when the cells are prevented from using the O_2 as occurs in metabolic inhibitor toxicity. This "hypoxcell" is also termed "histotoxic hypoxia."
- "Hypoxcell" is also present when the blood flow is stagnant or sluggish. Thus, the rate of supply to the tissues is inadequate for cellular metabolism. The name of this "hypoxcellular" situation is "stagnant hypoxia."

There are two other categories of "hypoxcell" called "anemic hypoxia" and "hypoxic hypoxia." In anemic hypoxia, the "hypoxcell" is due to a reduced O_2 content caused by changes in the red cell Hb. The Hb abnormality can be just the decreased concentration or a genetic alteration in the Hb molecule. Note that in anemic hypoxia and also in the "hypoxcell" due to stagnant and histotoxic hypoxia, the PO_2 in arterial blood, dissolved in the plasma is unchanged.

Only in hypoxic hypoxia is the reduced arterial O_2 content due to a drop in the PO_2. The Hb-O_2 saturation is thus reduced by the lower PO_2. The tissues become "hypoxcellic."

"Prevoload Aftepressload" (Preload, Afterload and Volume, Pressure)

The use of preload and afterload is regular terminology for those familiar with cardiovascular physiology. The preload and afterload describe cardiac filling and cardiac work, respectively. The preload is the end-diastolic filling (EDV) and the afterload is the upstream pressure in the aorta that the rhythmic contracting heart has to overcome to eject the next stroke volume.[6]

I noticed with my students that there is some difficulty in distinguishing preload and afterload, where the unit for one is the volume (in mL) and for the other is pressure (in mm Hg), respectively. So, we think if we can embed the volume and pressure parameters into the terms preload and afterload respectively, this will help the students to utilize the names well. So, preload can expand to "prevoload" and afterload can become "aftepressload."

Then when describing Starling's intrinsic mechanism of cardiac muscle tension generation, we can say the following:

The greater the prevoload, the bigger will be the stroke volume.

Over a minute, the venous return that contributes to the "prevoload" is related to the cardiac output. For each diastole, the "prevoload" is the recycled blood volume that enters the ventricle chamber added to the end-systolic volume (ESV) remaining from the last systolic contraction. In a person with chronic hypertension, the cardiac workload is increased since the "aftepressload" is higher. The use of calcium channel blockers produces peripheral vasodilation and this helps to reduce the "aftepressload" for the hypertensive person. The stroke volume is improved.

Hope this modified "load names" will lift some confusion over the meaning of preload and afterload in cardiac physiology.

"Phyaiku" (Physiology and Haiku)

The popular Japanese Haiku has a general 5, 7, and 5 syllables in the poems or rhymes that encapsulate for children and students, enjoyable and content-filled lines. We have written over the years on Haiku with Physiology contents. Here are some *osmotonic Phyaiku (a double portmanteau).*

Milk intolerance
Enzyme lactase deficient
Osmotic diarrhoea

Hyperglycemia
Filtered exceeds reabsorbed
Sweet urine excrete

SIADH
Hypotonic expansion
Neuronal swelling

Protein in urine
Oncotic pressure reduce
Edema collects

Gastric emptying
Duodenal osmosensors
No dumping syndrome

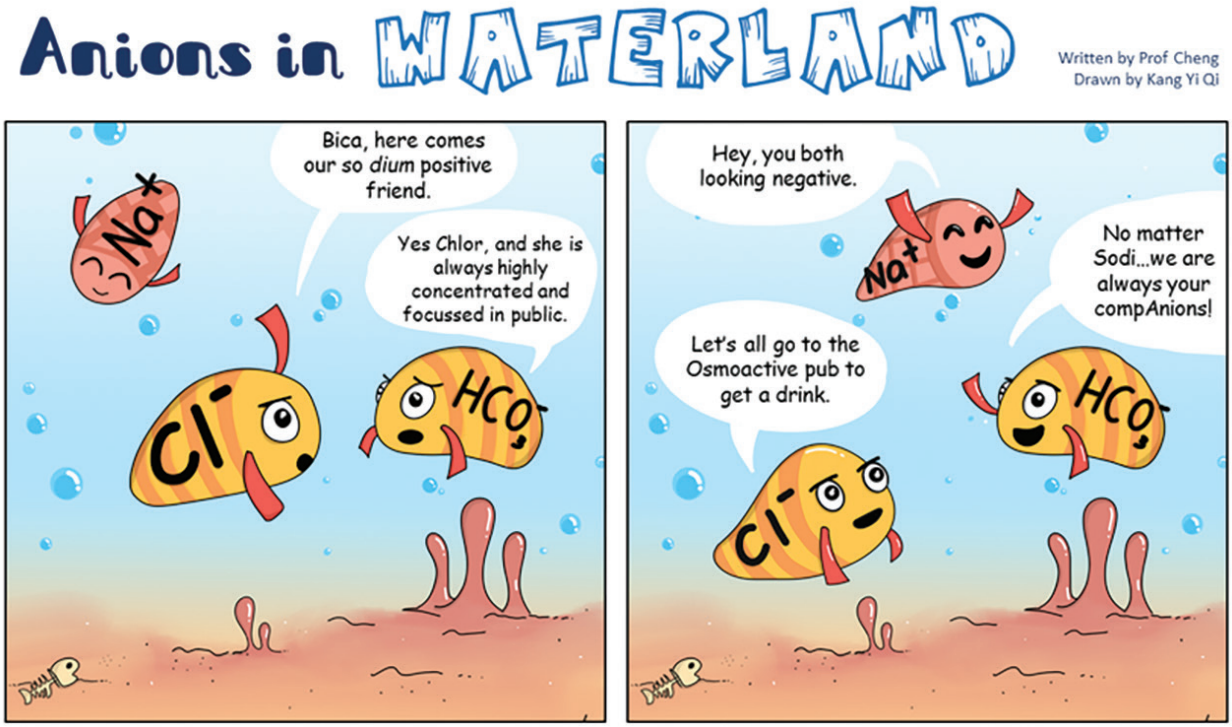

Fig. 1: Anions in waterland
The portmanteau "compAnions" are the two major anions, chloride and bicarbonate, associated with or that accompany sodium cation. Together NaCl and $NaHCO_3$ constitute the dominant "osmoactive" solutes in the ECF. Their effective osmotic pressure determines the direction of water fluxes across cell membranes.

Here for entertainment is a portmanteau "CompAnions" as part of a cartoon highlighting the "osmotonic" ions, sodium, chloride, and bicarbonate (Fig. 1).

Summary

The blending of two key physiology words into a portmanteau or "phyortmanteau" is a useful writing tool in teaching and learning physiology. Teachers can emphasize the portmanteau, mechanisms, and concepts that the two words are part of. This integration of words brings together main physiological points not merely to create a hybrid new term. Portmanteau combines physiological meanings from two words and provides a broader perspective of a specific physiological event. Students can review and create their own portmanteau according to their learning styles to summarize and clarify their understanding of physiology.

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