Saving Christmas: Use of Analogy to Teach The Compensatory Mechanisms of the Heart in Heart Failure.

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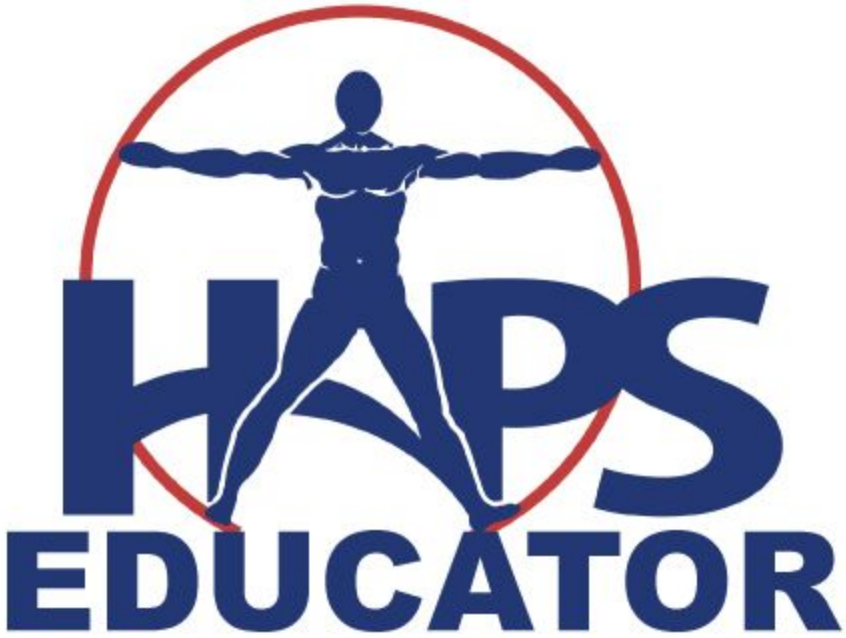

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Saving Christmas: Use of Analogy to Teach The Compensatory Mechanisms of the Heart in Heart Failure

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Abstract
Students of physiology are taught that the body's homeostatic mechanisms are in place to maintain the body's internal environment. This is most often associated with maintaining health. Congestive Heart Failure represents a disease in which the body's homeostatic mechanisms worsen the progression of the disease. Using the analogy of Santa Claus delivering presents around the world in a single evening, students can gain a better understanding of how the body's attempt to respond to a deviation from homeostasis, the decrease in cardiac output, may drive the progression of the disease.
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Key words: cardiac output, stroke volume, heart failure, RAAS

Introduction
Students of physiology and biology are taught the concept of homeostasis. The term was coined by Walter Cannon in 1932 in *The Wisdom of the Body* and used to describe the internal constancy of the body. Decades earlier, the French physiologist Claude Bernard observed that the body's internal environment, or "*milieu interieur*", tended to remain relatively stable despite constant changes in the external environment (Fox 2016). Of course, a deeper understanding of homeostasis, such as the "set points" for various physiological measurements, developed over time. In most cases of injury, illness or disease development, homeostatic mechanisms prevent the onset of symptoms, or in serious cases, loss of life. Thus, when students are faced with a physiological scenario in which the body's homeostatic mechanisms actually exacerbate the very condition they were attempting to correct, confusion is reasonable and expected. In a2010 review article "Congestive Heart Failure: Where Homeostasis Begets Dyshomeostasis", the authors discuss the evolution of the understanding of homeostasis, and how homeostatic mechanisms can further contribute to the progression of a disease (Kamalov *et al*. 2010). The term, dyshomeostasis, was suggested by Richards to describe homeostatic mechanisms that were either inappropriate for the condition, excessive, or deficient to correct a dysfunction (Richards 1960).

The Dyshomeostasis of Heart Failure
Heart failure is defined as the inability of the heart to adequately meet the oxygen demands of the body on a persistent basis. In response to heart failure, homeostatic compensatory mechanisms take place that in the long-term cause further damage to the heart and worsen the progression of the condition (Porth and Gaspard 2015). These mechanisms include activation of the sympathetic nervous system, ventricular hypertrophy with chronic remodeling, and increased preload through activation of the renin-angiotensin-aldosterone system (RAAS) and the Frank-Starling mechanism (Porth and Gaspard 2015).

In the United States, heart failure is the leading cause of hospitalization among the elderly and affects over 5 million Americans, with 500,000 new cases diagnosed annually (Kamalov 2010). The worldwide prevalence for all types of heart failure in 2017 was 11.8% (Riet *et al*. 2017). Consequently, students taking physiology and pathophysiology will focus a great deal of time on the cardiovascular system and its common diseases. Students pursuing healthcare careers in particular will at some point inevitably encounter a patient diagnosed with heart failure.

Heart failure is a complex clinical syndrome that involves homeostatic responses from several other body systems. It presents a great opportunity for students to study homeostasis and integrate the function of several systems at once. In an undergraduate physiology or pathophysiology course, in which heart failure is a major topic, students must first recall and understand how cardiac output is determined. Then, they must understand the homeostatic responses to diminished cardiac output.

However, the responses to diminished cardiac output are the same homeostatic mechanisms that drive the progression of heart failure. In fact, today's standard of care for heart failure is focused on delaying disease progression, primarily by attempting to contain the body's homeostatic mechanisms

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(Kamalov *et al*. 2010). When the full mastery of a concept requires memorization, understanding and the ability to apply and analyze material, analogies may be a particularly helpful place to begin. Therefore, an analogy to a scenario where a mechanism should improve a condition in the short-term, but causes further problems in the long-term, is warranted.

In the activity that follows, students in an undergraduate pathophysiology course at Drexel University were presented with a simple analogy that should be easily understood. Moreover, it would be easy for them to explain to patients or others attempting to understand the condition. All images in the activity were hand drawn by the professor. Students in this course utilize Porth's Essentials of Pathophysiology as their primary text.

Student Activity

We have learned that heart failure is the inability of the heart to maintain sufficient cardiac output in order to meet the metabolic demands of tissues and organs. In other words, the body's tissues are not receiving the oxygen and nutrients they need to carry out normal function.

The most common cause of heart failure is atherosclerosis, which impedes blood flow through the coronary arteries supplying the heart. Other common causes are chronic hypertension, which makes it harder for the heart to pump blood through the arterial system, and valvular heart disease.

The human body is efficient at maintaining homeostasis. Nothing is more important to the body than meeting its oxygen demands and there are a number of homeostatic mechanisms in place to ensure that this is possible. These compensatory mechanisms of the heart/body are very helpful in restoring normal cardiac output.

To review:
Increased preload: According to the Frank-Starling mechanism of the heart, increased vascular volume (via activation of the RAAS system) will increase myocardial force of contraction, and therefore, the amount of blood ejected per beat.
Result: Increased myocardial contractility and stroke volume

Myocardial hypertrophy**:** Like any muscle of the body put under extra stress, the myocardium will grow larger, in an effort to get "stronger".
Result: Increased myocardial contractility and stroke volume

Sympathetic Nervous System activation**:** The sympathetic nervous system innervates the SA and AV nodes, as well as the myocardium. When cardiac output drops, SNS activation will occur. This will increase the rate and force of contraction.
Result: Increased heart rate, myocardial contractility and stroke volume

All of this sounds great, right? However, over the long term, adjustments are detrimental to the heart. Why?

To answer that question, let us recall what caused the heart failure in the first place. Since the most common cause of heart failure is atherosclerosis, we will start there. Atherosclerosis causes decreased perfusion of the myocardium, meaning the heart muscle itself is not getting the oxygen it needs to work effectively. People with heart failure due to atherosclerosis have often had myocardial ischemia, meaning that part of the heart muscle is incompetent (angina) or no longer functioning (infarction). Each of the three compensatory mechanisms listed here increases the heart's need for oxygen and nutrients, which it could not get enough of in the first place. Current management of heart failure is directed towards reducing the harmful consequences of these compensatory responses.

Now, what does all of this have to do with Saving Christmas? Analogies to things we all understand and can easily imagine can be powerful learning tools. In this exercise, we will use the example of Santa Claus attempting to deliver presents to all the children around the world in one evening to better understand heart failure. As you might have guessed, in this analogy:

Cardiac Output = All the children getting their gifts on Christmas morning.

Recall that Cardiac Output is defined as: Heart Rate x Stroke Volume. Let us establish what would be the analogous components in our Christmas story.

Presents to the children are the blood, filled with oxygen (Stroke Volume)

Santa and his sleigh (The heart)

The speed of the reindeer pulling the sleigh (Heart rate)

Imagine that one Christmas, Santa fails to get all the children around the world the gifts on their wish lists (decreased cardiac output). Santa is not being very forthcoming with Mrs. Claus about what is going wrong. Mrs. Claus, who has to receive all the sad letters the next year, decides to add some reindeer to Santa's sleigh to speed him up. Instead of four reindeer, Santa now has five:

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Figure 1. Santa has to carry extra reindeer food.

While this speeds up the sleigh at first, Santa still comes up short, because now he has to carry extra reindeer food. Carrying extra food causes the reindeer to get tired more quickly. So, the compensatory mechanism to speed up the sleigh (SNS activation to increase heart rate!) ultimately does more harm than good.

Mrs. Claus then decides that perhaps Santa simply needs to carry more presents. Perhaps the problem is that he is not leaving the North Pole with the number of presents he needs. In this scenario, children who wrote to Santa are like the cells of the body not receiving adequate oxygen, or the kidneys not receiving adequate perfusion.

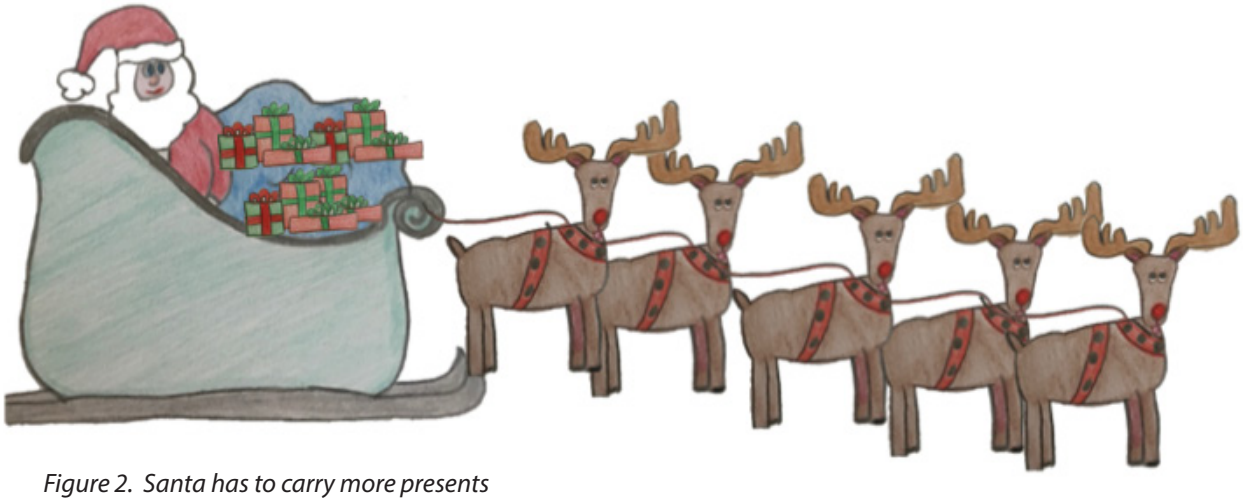

Figure 2. Santa has to carry more presents

However, just like the added reindeer food, these added presents weigh down the sleigh, and the reindeer get too tired. So, the compensatory mechanism to carry more presents (increase stroke volume) ultimately does more harm than good.

Finally, Mrs. Claus decides Santa's sleigh needs to be expanded. Now he can carry enough food for the reindeer, and all of the presents.

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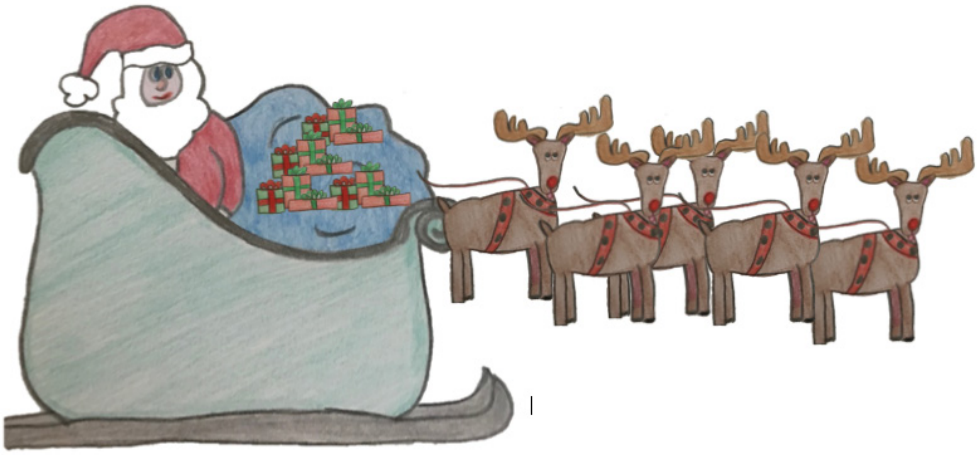

Figure 3. Santa needs a bigger sleigh.

Alas, Santa still fails to make his deliveries. Why? The bigger sleigh requires more gas, and that gas is Christmas spirit. What Santa and Mrs. Claus did not realize all along was that the lack of Christmas spirit was the problem in the first place, and nothing can replace it.

Imagine that Christmas spirit is the blood supply to the myocardium; it does not matter how the heart remodels if it cannot get the nutrients and oxygen it needs to meet the body's demands. Thus, the most common cause of heart failure, atherosclerosis, will always be the source of the problem.

What about chronic hypertension, another common cause of heart failure? Imagine hypertension like a strong wind blowing against the sleigh. It does not matter how much more powerful the sleigh is, or how much faster the reindeer are, or how many more presents Santa carries, if the wind that resists the sleigh does not stop blowing.

In your group, try to expand upon this story. For example, heart failure can be classified as systolic or diastolic dysfunction. What would be "wrong" with Santa's sleigh in these scenarios, and how would that impact cardiac output?

Review the common medications or other treatments for heart failure that we discussed in class such as beta blockers and diuretics. Do these measures target the compensatory mechanisms that Mrs. Claus tried, or do they actually assist the heart in maintaining cardiac output? What would be the consequences of surgical removal of excessive myocardial tissue?

Discussion

The story of saving Christmas is not for everyone. Any analogy in which the attempts to solve a problem (i.e. the homeostatic compensatory mechanisms) further exacerbate the condition would be appropriate. Challenge your students to create their own story. Whether as a formal assessment (perhaps a small group presentation) or just an activity to break up the normal class period, activities such as this can engage students in a creative process.

Furthermore, since this activity requires students to *remember* normal cardiovascular dynamics, *understand* how the heart responds to diminished cardiac output, and *apply* the compensatory mechanisms, it naturally takes students through Bloom's taxonomy of learning. If students then expand upon the story, or create their own, they are taking Bloom's taxonomy a few steps further to *evaluation* and *synthesis* (Krathwohl 2010). Students may appreciate the effort to bring humor into learning, and view the instructor as more approachable and invested in their learning. These factors have been shown to increase student self-confidence (Micari and Pilar 2012).

A note of caution is warranted in the use of analogies for complex physiological or pathological mechanisms. Brown and Salter (2010) recommend that analogies should be clear and well-understood by a student. Otherwise, explanation of the analogy can potentially lead to more confusion. They further suggest that analogies should also acknowledge the differences between the learned concept and the analogy and the limitations of the analogy's applications (Brown and Salter 2010).

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In conclusion, a simple analogy may improve student understanding of dyshomeostasis, as in the case of studying heart failure. Instructors are encouraged to challenge students to create their own analogies whenever a concept is particularly difficult to understand.

About the Author

Krista L Rompolski received a BS in Exercise Science and an MS in Exercise Physiology from Bloomsburg University, and PhD in Exercise Physiology from the University of Pittsburgh. She is a Certified Exercise Physiologist through the American College of Sports Medicine. Dr. Rompolski is the co-author and lead Digital Author of the 15th edition of Human Physiology, by Dr. Stuart Fox. She is an active member of the Human Anatomy and Physiology Society and the American Academy of Anatomists. Her teaching interests include pathophysiology, gross anatomy, and anatomy and physiology.

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