CARDIOVASCULAR PRESSURE-FLOW RELATIONSHIPS: 
WHAT SHOULD BE TAUGHT?

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Cardiovascular physiology reflects the interrelations of flow, pressure, and resistance. Undergraduate students are often confused by the complexity of the system. This symposium presents a sequential presentation of the underlying concepts, building on analogies, past experience, and conceptual models to allow students to develop a physiologically appropriate understanding of cardiovascular physiology.


Key words: cardiovascular physiology; undergraduate; active learning; pedagogy

The existence of life depends on having substances, such as amino acids, nutrients, and ions, differentially distributed. This distribution is facilitated by the existence of barriers, such as the cell membrane at the microscopic level or the skin at the organism level. Physiology is the study of the movement and the regulation of movement across these barriers. There are no more basic, fundamental physiological concepts than gradients and conductance. Specific applications of these principles allow understanding of diffusion, osmosis, filtration, and the flows of blood, air, and urine. This update symposium is an introduction to the themes of gradients and conductance. It will be further developed in the next update symposium today, Barb Goodman’s “Respiratory and Renal Pressure-Flow Relationships,” (1) and a number of workshops on the following days.

One of the major difficulties reported by students who have difficulty in mastering physiology is that the information presented does not match with concepts that they have previously developed. This is particularly troubling for students, because not only do they have to learn the new material, but they have to “unlearn” the incorrect information first. Some of these misconceptions develop as a consequence of the instruction. For example, I regularly use analogies when I am explaining concepts, allowing the students to build on information that they already possess. This very powerful teaching approach, however, has a significant disadvantage. Analogies are not perfect, and extending them beyond the specific case often leads to confusing or incorrect conclusions. Joel Michael will present a workshop on identifying student misperceptions and ways to correct them (3).

Finally, students must be allowed to work with the new information that they have gathered. If students understand the material, they will be able to apply it to a novel situation and to draw the correct conclusion. This is an active process that solidifies the learning, making it more permanent. The specific topics covered in this update symposium will include the topic as well as teaching approaches and activities to help the students learn physiology more effectively.

Background and Definition of Terms

One of the first steps in opening a discussion is to establish specific definitions for the terms to be used. The terms used here are defined as specifically related to the cardiovascular system.

Pressure (P) is the force applied over an area. This force can act to raise the level of fluid in a tube and is termed a hydrostatic pressure. In the cardiovascular system, the contraction of the muscle of the heart both increases pressure and provides the energy to
propel blood out of the ventricles. Cardiovascular pressures are expressed as the height of a stationary column of fluid (mmHg, or millimeters of mercury). This resulted from the clinical use of mercury manometers to measure blood pressures in the early 1900s. Other pressures, such as that due to the osmotic movement of water, can be expressed in similar units to allow easy comparison.

**Flow** (Q) is the volume flow rate, for our purposes, of blood. Consequently, it is expressed as volume per time (e.g., ml/min). It is important to distinguish flow from velocity, which is expressed in distance per time.

**Resistance** (R) is a measure of the opposition to fluid movement in the cardiovascular system. For convenience, resistance is often expressed in peripheral resistance units, a derived value calculated from the relationship: 

\[
R = \frac{P \text{ gradient}}{Q}
\]

It is important to recognize that vessel radius is the primary determinant of resistance. The relationship is inverse, and the mathematical relationship is the fourth power of the radius. (Poiseuille’s Law). Decreasing a blood vessel radius to one-half of the starting radius will increase resistance to flow 16-fold.

**Cause and effect.** Physiology is an experimental science. Most of what we know about the body results from experiments in which only one variable is changed and all other variables remain constant. The variable that changes is termed the cause, or independent variable. Any alterations that occur subsequent to the intervention are termed effects, or dependent variables. By convention, experimental graphs are plotted with the cause on the x-axis and the effect on the y-axis. Many physiological changes are time dependent, so time often appears on the x-axis. If an increase in the “cause” causes an increase in the “effect,” the relationship is said to be direct and will be indicated by a proportionality symbol (\(\alpha\)). If an increase in the cause causes a decrease in the effect, the relationship is called inverse (one goes up, the other goes down). This will be indicated by the reciprocal of the proportionality symbol (1/\(\alpha\)).

Teaching: this material is fact based, rather than conceptual, and can be covered in assigned readings (handout) or in lecture. It is important that the entire class begins with these common definitions.

**Vascular–Ohm’s Law (Cardiovascular Variant)**

Stephen Hawkings shares a wry observation in the introduction to his book “A Brief History of Time.” His publisher felt that for every equation included in the book, the readership drops by 50%. Hawkings restricted himself to one equation: 

\[
E = mc^2
\]

The message for instructors is that less is more; whereas equations may be a useful shorthand, they do not of themselves bring clarity to a concept except for a minority of people (many of whom are science teachers, but that is another topic).

For the cardiovascular system, the key equation is

\[
Q = \frac{P \text{ gradient}}{R}
\]

This equation is particularly troubling for students, because it contains three variables, all of which can change simultaneously and which are interrelated. To introduce the equation to students, it is useful to artificially devise a system in which one of the variables is constant and to experimentally examine the interaction of the two remaining variables.

### 3.1 Implications if one variable kept constant.

Diagram: a river flowing through a canyon

#### 3.1.1 If inflow is held constant.

**Problem:** a landslide blocks a flowing river, creating a dam.

**What happens (immediately) to**

1) flow into the canyon
2) flow out of the canyon
3) the river level upstream from the landslide
4) the river level downstream from the landslide

**Will flow out of the canyon ever return to normal, and why?**

Concept: when resistance is increased, flow past the resistance is immediately diminished. Flow into the canyon is unchanged. Pressure (water level) upstream from the constriction rises, and pressure downstream falls. The \(\Delta P\) continues to increase until flow past the
constriction resumes. Ultimately, a balance is reached where the flows entering the canyon, past the resistance, and exiting the canyon are equal. When that happens, the system is stabilized at a higher \( \Delta P \) and higher resistance but normal flow.

Cardiovascular implication: constricting arterioles raises arterial pressure and lowers capillary pressure. The sympathetic nerves are widely distributed in the periphery, and sympathetic constriction of arterioles is a major mechanism for controlling blood pressure. If the constriction is maintained, flow past the arterioles will return to normal if the arterial-capillary \( \Delta P \) is sufficiently high.

Possible misconceptions: it is INFLOW that is constant, not flow past the resistance. Also, the model assumes that all fluid entering the system must go past the point of constriction. This viewpoint is valid when looking at total peripheral resistance, such as controlled by the sympathetic nerves. It does not, however, apply in situations in which resistances are in parallel and flow may be shunted to another vascular bed.

3.1.2 If \( \Delta P \) is held constant, \( Q \) is \( 1/\alpha \) to \( R \). Problem: municipal distribution of water

Why does water come out of the faucet?

What problems could stop the water from flowing?

Concept: when resistance is increased (at the faucet), flow past the resistance is diminished (or stopped if resistance increases sufficiently). When resistance decreases, flow past the resistance is increased. In this case, flow is inversely proportional to resistance.

This system depends on an adequate pressure head (the municipal water tank). If water pressure at the house decreases, then the flow will not be as great for any reduction in resistance. If water \( P \) falls below the height of the faucet, opening the faucet will actually cause air to go into the water pipes.

Cardiovascular implication: constricting arterioles reduces flow to the downstream vascular beds. Local control systems use this mechanism to match tissue blood flow to metabolic needs.

Possible misconceptions: the local arteriolar dilation will cause arterial pressure to fall. This normally does not occur, because the arterioles involved represent only a small portion of the total number of arterioles. However, if all arterioles are dilated by local mechanisms (septic shock), then arterial pressure would fall much as would occur to municipal water if everyone took a shower in a city at 6:53 AM.

3.1.3 If \( R \) is constant, \( Q \) is \( \alpha \) to \( \Delta P \). Problem: If you are taking a shower when the toilet is flushed, what happens to:

water flow out of the showerhead?

the water temperature?

Concept: as the water tank on the toilet fills with cold water, the pressure within the cold water pipes falls. This reduced \( \Delta P \) (only on cold water side) reduces cold water flow out of the showerhead even though the resistance (in the bathtub) remained constant. Hot water pressure, however, is buffered by the volume in the hot water tank and remains somewhat constant. Cold water flow diminishes, and hot water flow remains constant, and the water temperature increases.

Cardiovascular implications: physiologically, resistance is the least stable of the variables. However, there is one pathology, coronary artery stenosis, in which the narrowed coronary artery provides a fixed resistance. In this case, myocardial blood flow falls if arterial pressure falls. If the stenosis is severe, the stenosis becomes the major site of resistance to blood flow, and the arteriolar dilation, such as occurs during exercise, does not allow myocardial blood flow to increase (resistances connected in series). This is why individuals with coronary artery stenosis are at risk for myocardial ischemia during exercise.

3.2 Variables change simultaneously—your sibling and the water hose. Diagram: a long cylinder, the middle of which can be constricted.
3.2.1 Increasing velocity of water exiting at the end of hose. Problem: you are washing the car. Your sibling, who is 9 ft away, desperately needs to be squirted.

Concept: velocity of water movement is dependent on the flow ($\Delta P$ and $R$) and the cross-sectional area. Occlusion of a portion of the end of the hose with your thumb will decrease the cross-sectional area. The reduced cross-sectional area increases velocity of water flowing past that point if flow remains constant. Occlusion, however, will acutely increase the $R$ and, consequently, decrease the flow. Because of the reduced outflow, the pressure on the “house” side increases and $\Delta P$ increases until flow out the end of the hose recovers toward the starting point. Whether flow recovers all of the way to the starting point is determined by the magnitude of the increase in the $\Delta P$.

Cardiovascular implications: increasing resistance on a flowing system will cause a reduction of flow past the site of $R$ and an increase in the $\Delta P$. During times of intense sympathetic activation, the increase in arterial pressure comes at the expense of tissue blood flow. Certain vascular beds, such as the heart and the brain, respond poorly to the increased sympathetic nerve activity and are consequently spared the reduction in blood flow during sympathetic activation.

High velocity of blood flow can lead to turbulence, which is audible with a stethoscope. The sound caused by turbulent blood flow past a narrowing in the heart is a murmur or, if the narrowing is in a major artery, a bruit.

Possible misconceptions: one common misconception is that the Korotkoff sounds heard while using a sphygmomanometer to estimate arterial blood pressure come from turbulent Q. The real cause is a “water-hammer” pulse, generated when a moving column of water hits a stationary column of water, such as the clanging of the pipes in a house heated with steam radiators. When taking a blood pressure, the column of fluid is interrupted when cuff pressure is between systolic and diastolic pressures, and the water-hammer pulse occurs. Flow, however, is not reestablished until cuff pressure falls below venous pressure, because flow to the arm requires both inflow and outflow tubes.

Teaching Options

Interactive lecture. This material was presented in an interactive lecture format, in which problems are posed and students are given time to develop answers before the lecture continues. It is possible to use photographs, videotape or audiotape to enliven the presentation of the problem.

Inquiry. The American Physiological Society has cooperated with high school teachers to develop an inquiry-based exercise that examines these relationships, called the “Elvis experiments.” It is a component of the “physiology of exercise” module. The same exercises are appropriate for undergraduate and professional students.

P and Q generation in the heart

One measure of work in a fluid system is (change in $P$) $\times$ (change in volume). In the cardiovascular system, myocardial contraction provides the work that propels blood through the body. The energy is stored as a pressure (potential energy) and is converted to kinetic energy as blood flows from the aorta/pulmonary artery through the vascular tree. The energy is dissipated by the blood flowing past a resistance and is released as heat due to friction.

4.1 Generating pressure–strength of myocardial contraction. Cardiac muscle is striated and relies on the same actin-myosin cross-bridge sliding-filament mechanism that is found in skeletal muscle. That includes the use of preload to determine the overlap of the actin and myosin filaments. Cardiac muscle, however, wraps around a chamber, and the contraction compresses that chamber. In the heart, preload is measured as the resting volume (rather than length). Contraction generates pressure (rather than tension). Afterload is measured as a pressure (rather than a weight).

4.1.1 Preload. Preload is the amount of stretch imposed on the myocardial muscle before the contraction. The volume in the cardiac chamber before contraction sets the preload. This volume reflects the distensibility of the myocardium and the pressure that is forcing blood into the chambers. The resting (end diastolic) volume is difficult to quantitate, so venous pressure is traditionally used as an index of the filling of the heart, or the preload. The connective tissue in
the myocardium helps prevent overstretching of the heart by the preload. Consequently, at physiological levels of pressure, the heart never passes the “optimal” cross-bridge alignment and operates only on the ascending limb of the length-tension curve.

4.1.2 Contractility. Unlike skeletal muscle, the rate of cycling of the cross-bridges is variable, controlled by intracellular calcium. As intracellular calcium rises, the rate of cycling increases and the rate of pressure generation within the ventricles increases. This rate of pressure development, $\Delta P/\Delta t$ (dP/dt), is a measure of contractility.

The myocardium, then, has two independent mechanisms to regulate pressure generation, preload, and contractility. To use an analogy, it is like the Roman slave galleys in the “Ben-Hur” movies. These ships had rows of slaves manning the oars, and the cadence was kept by matching the rowing cycle with the drum beat on a kettle drum. There are two separate ways to increase the movement of the ship: change the number of oars in the water or increase the cadence. Increasing the number of oars in the water is analogous to the alignment of the actin and myosin; increasing the cadence is analogous to increasing contractility. Because these are independent mechanisms, they can operate in unison, where both preload and contractility can increase simultaneously to increase cardiac performance.

4.2. Generating flow–afterload and cardiac output. Flow requires a $\Delta P$ and a finite resistance. For the cardiac output, the flow of concern is the flow from the left ventricle into the aorta. The pressure generated by ventricular contraction (described above) determines left ventricular pressure. Arterial pressure is the “downstream” side of the $\Delta P$. Arterial pressure, together with any resistance to flow through the aortic valve, represents the afterload. Normally, resistance to flow through the aortic valve is low, and arterial pressure is the major determinant of afterload.

4.3. Cardiac cycle (Wiggers) diagram–integrating pressure, volume, and flow. This material is generally difficult for students to master without guidance. An interactive lecture format works to introduce the students to the concept of a pump and provide an accurate, predictive model of the cardiac cycle (Fig. 1).

Valve movements

What happens if $P$ in chamber 1 > $P$ in chamber 2?
Valve M is open, and volume moves from 1 into 2

What happens if $P$ in 2 is greater than $P$ in chamber 1?
Fluid starting to move from 2 into 1 will close valve M,
No fluid will move when the valve is closed

What happens if $P$ in chamber 2 > $P$ in chamber 3?
Valve A is open, and volume moves from 2 into 3

What happens if $P$ in 3 > $P$ in chamber 2?
Fluid starting to move from 3 into 2 will close valve A,
No fluid will move when the valve is closed

What happens to the $P$ in chamber 1 when it is compressed from the outside?
P in chamber 1 increases

What happens to the $P$ in chamber 2 when it is compressed from the outside?
P in chamber 2 increases

Assume $P$ in 3 is higher than $P$ in 1. What would happen if both valves M and A were open at the same time?
Fluid would flow from 3 through 2 into 1
Relating to the body. Cardiac cycle is the sequential contraction of the atria and then the ventricles. Follow the generation of P and movement of the valves during the cycle.

After the students are comfortable with the model, relate the concept to the cardiac cycle figure in the textbook.

Possible misconceptions: atrial contraction is not essential for normal cardiac output to be achieved and becomes significant only in the high cardiac outputs characteristic of moderate and extreme exercise.

Summary Of Cardiovascular P-Q-R Relationships

When arterial pressure is constant, arteriolar resistance controls tissue blood flow. Cardiovascular regulatory systems are designed to maintain arterial pressure. Tissue blood flow is then controlled by tissue metabolic needs (Fig. 2).

5.2.1 Arterial pressure is the regulated variable.
From the figure, cardiovascular regulation is centered on maintaining an appropriate arterial blood pressure. Cardiac output is important in that it is one of the two determinants of arterial pressure. Stroke volume is important in that it is one of the determinants of cardiac output. Diastolic volume is important in that it is one of the determinants of stroke volume. This logic can be applied to any of the components in the above diagram.

From a control systems approach, the arterial pressure is the regulated variable. Arterial pressure is sensed in the baroreceptors of the aortic arch and

FIG. 2.

Sympathetics. Figure modified from Rushmer (Cardiovascular Dynamics, Saunders, 1976). In the original figure, the analysis is expanded to include a number of disease states.
carotid sinus. A decrease in arterial pressure causes a reflex activation of the sympathetic nervous system. Sympathetic nerves, acting on a variety cardiovascular target organs, act to return arterial pressure toward normal.

This approach is somewhat of a simplification and focuses on the short-term, homeostatic, regulation of arterial pressure. For example, the cardiopulmonary receptor and renal control of blood volume is ignored. The ability to adapt to changing demands, such as the feed-forward regulation of blood P during exercise is not considered.

Summary
For the cardiovascular system, the relationships among pressure, flow, and resistance must be taught and explored. These interrelated variables determine the behavior of the entire cardiovascular system. The key to understanding cardiovascular control is to focus on two major points: 1) if arterial pressure is sufficient, local (metabolic) control will determine the flow to any individual capillary bed and 2) all nervous system and endocrine control systems are designed to insure that arterial pressure remains sufficient.

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