Understanding basic vein physiology and venous blood pressure through simple physical assessments

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INTRODUCTION

Objectives and Overview

The importance of arterial blood pressure measurement and its interpretation is extensively taught in all health care courses. Students gain an appreciation of the normal physiological control of arterial blood pressure, the interplay between mean arterial pressure and total peripheral resistance in maintaining cardiac output, and subsequent organ perfusion. The emphasis on learning about arterial blood pressure is not surprising: we know that hypertension is a major contributor to cardiovascular disease and mortality world wide (2, 8), and long-term high blood pressure causes end-organ damage, such as heart failure (14). An understanding of venous function is necessary to comprehend overall cardiovascular physiology, including arterial blood pressure. Students need to appreciate that the venous system forms one side of a two-sided closed system, where venous return and cardiac output are equal in healthy individuals in steady-state conditions. Adequate venous return is essential for a normal cardiac output and contributes to the regulation of arterial blood pressure. Therefore, a knowledge of factors that affect both peripheral and central venous pressure (CVP) is essential to understanding the interplay between heart and vascular function and, therefore, the hemodynamics of the circulatory system in general. In this paper, we describe simple laboratory tests that aim to illustrate a number of important properties of vein physiology. At the end of the exercises, students will be able to understand, measure, assess, and interpret the pressure in veins in different body regions and relate their observations to the circulatory system as a whole. The aim of this paper is to improve student understanding of the basic physiology of veins in healthy adults through physical examination in the laboratory class.

Background

Veins as capacitance vessels. There are two principal functions of veins: 1) to act as conduit vessels, transporting blood back to the heart from the body’s organs and tissues (i.e., the venous return); and 2) to act as capacitance vessels, accommodating large volumes of blood. At rest, the venous structures contain approximately two-thirds of the total blood volume and thus act as a blood reservoir (6). The ability of veins to house this volume of blood at any given time relates to their structure. Veins have thinner walls and larger diameters than arteries with less muscle and elastic tissue. This means that they have high vascular compliance so that the rate of change in volume with changing pressure is high and, therefore, changes in venous blood volume produce relatively small changes in venous distending pressure. In fact, veins have a compliance that is 30 times that of arteries (5), a reason why veins can be used as arterial bypass grafts. Veins are, therefore, highly distensible, expanding easily to accommodate large volumes of blood.

It is known that venous capacitance vessels react to outputs from baroreceptors as well as reflexes associated with chemoreceptors and cardiac receptors (5). Neurohumoral mechanisms can mobilize the blood in veins to maintain filling pressure in the right heart when required. Examples of this include venoconstriction during exercise or hemorrhage where sympathetic
activity via adrenergic stimulation reduces venous compliance and capacitance of the splanchnic vessels, increases peripheral venous pressure, and propels blood forward to the heart. Since venoconstriction and venodilation have significant effects on the distribution of total blood volume, both can affect CVP, stroke volume, and arterial blood pressure.

Effect of posture on venous pressure. Postural changes represent a major physiological challenge to blood pressure, and normal physiological adjustments need to occur to preserve blood flow to critical organs. When standing from a supine position, gravitational forces “pull” venous blood to the lower limbs (this also occurs on the arterial side). Due to the high compliance of veins, ~500 ml of blood can be redistributed to peripheral veins (9); this is known as venous pooling. The term venous pooling is sometimes misunderstood by students. It does not refer to a stagnant pool of blood, rather it refers to the slower transit time of blood through the venous circulation. This venous pooling leads to an immediate drop in stroke volume of 40% and an overall drop in cardiac output of 20% (7). However, mean arterial blood pressure is normally preserved, as there are compensatory increases in vascular tone mediated by the autonomic nervous system. Nevertheless, even healthy humans sometimes experience lightheadedness due to a transient drop in arterial pressure that occurs in the initial few seconds of standing.

William Harvey was the first person to demonstrate that blood in veins flows in one direction only due to the presence of venous valves. Harvey’s experimental illustrations were published in 1628 in De Motu Cordis (6a). When the venous valves close to prevent retrograde flow, a column of blood extends from the heart to the limbs. The weight of this venous blood exerts the hydrostatic pressure. This hydrostatic pressure is referenced to right atrial pressure (RAP). Pressure in veins is dependent on the position of the vein in relation to the heart (see Fig. 1). The veins below the right atrium are exposed to positive hydrostatic pressure and are distended by the pressure within them. The greater the distance from the right atrium, the greater the column of blood and, therefore, the greater the pressure, with it being highest in the feet.

In a practical class, we investigate the presence of venous valves and the skeletal muscle pump as two factors that facilitate return of blood to the heart. Venous capacity is influenced by the functional state of both the valves and the muscle pump, and descriptions of the assessment of each (in addition to some excellent descriptions on the general physiology of veins) and their role in determining venous pressure date back over a century (15). Venous blood from the lower limbs is transported against gravity, and larger veins possess valves to prevent any backflow of blood. In many regions of the body, veins run between and near skeletal muscles. Contraction of the skeletal muscles surrounding veins increases the pressure within the veins, pushing open the proximal valve and forcing blood toward the heart. For example, when calf muscles contract during exercise, blood is forced toward the heart, thus increasing venous return. When the muscles relax, the distal valve closes as the retrograde flow presses against the valve leaflets, and blood from distal veins is sucked into the empty muscle veins. In an individual standing still, the mean blood pressure in the veins of the feet will be ~90 mmHg (~120 cmH₂O) (6, 9). This will be significantly reduced (as low as 20 mmHg; ~30 cmH₂O) when that individual walks around (9). In this way, venous pooling is limited, and blood is “pumped” back toward the heart. Reflex capacitance responses are essential to limit cardiac output decreases when standing, and the skeletal muscle pump must be employed to avoid fainting (11). It is important to note that an effective skeletal muscle pump depends on valves working correctly. Incompetent valves do not close effectively and lead to chronic distension of veins, known as varicose veins.

Veins above heart level experience negative hydrostatic pressure and will be collapsed as the surrounding tissue pressure exceeds vein pressure, with a trickle of blood flowing through them. Note, the dural sinuses have rigid walls and cannot collapse, and so the pressure within them is subatmospheric. When lying supine, the effects of hydrostatic pressure are abolished and regional differences in venous pressure are relatively small.

Venous return and central venous pressure. Venous return is the volume of blood that returns from the veins to the atria each minute and is ~5 l/min. Peripheral venous pressure is the pressure in the peripheral veins draining the body organs and tissues. It is the pressure differential between the peripheral and central veins that determines venous return. The venous side of the circulation is a low-pressure system compared with the arterial side. Pressure within the named veins is usually between 8 and 10 mmHg, and CVP is ~0–6 mmHg (3, 9). Therefore, the pressure gradient between the periphery and the right atrium is small.

The volume of blood contained within the peripheral veins at any given time is determined by the venous pressure and venous resistance. Venous return is facilitated by a number of factors, including inspiration, increased total blood volume, increased venomotor tone, the cardiac suction effect, the presence of venous valves and the skeletal muscle pump. The factors that enhance venous return do so by increasing the

![Fig. 1. Effect of gravitational forces on venous pressure in different body regions when standing (6).](http://advan.physiology.org)
venous pressure gradient, which increases preload and, therefore, cardiac output by the Frank-Starling mechanism. The Frank-Starling law of the heart describes the positive relationship between ventricular myocyte stretch and force of ejection. The greater the preload (usually measured as end-diastolic volume or end-diastolic pressure), the greater the force of contraction of the ventricles, and the greater the subsequent stroke volume.

CVP is the pressure of the blood in the thoracic vena cava near the right atrium. CVP is an approximation of RAP and is measured in millimeters of mercury (mmHg) or centimeters of water (cmH₂O) above atmospheric pressure; 1 mmHg = 1.36 cmH₂O. A normal CVP reading is 0–8 cmH₂O or 0–6 mmHg (3, 9). An upright posture causes a redistribution of venous volume to the peripheral veins and decreases CVP, so too does venodilation. The CVP drops to −3 to −5 mmHg when the filling pressure is greatly reduced (6). On the other hand, venoconstriction of peripheral veins shifts venous volume to the central veins and increases CVP. In addition, CVP can increase to as much as 20–30 mmHg in cases of severe heart failure (6). The pressure gradient between the MAP and CVP is responsible for capillary blood flow. If this gradient is diminished, as would take place with high CVP, then organ blood flow would be reduced.

Measuring CVP directly is an invasive practice. The main sites used for insertion of a catheter are the subclavian, internal jugular, or a peripheral vein, and complications such as discomfort, infections, and accidental puncture can inevitably occur (18). Noninvasive measurement of CVP is relatively common in clinical practice with the use of bedside ultrasound, which provides rapid and accurate assessments through measuring inferior vena cava diameter to assess a patient’s volume status (4). However, for basic learning purposes in this practical, we investigate CVP by assessing peripheral venous collapse and jugular venous distension. It must be appreciated that an isolated CVP measurement is not of particular clinical significance but can be useful in an overall assessment when taken together with other hemodynamic measurements (12, 16).

Using the jugular venous pressure (JVP) as an estimate of CVP was first described by Lewis in 1930 (10). A modified version of this technique is commonly used today. The Lewis method determines the CVP by measuring the vertical distance between a point 5 cm below the sternal angle and the top of the neck veins. Although this method is not suitable for CVP measurement in critically ill, unstable patients (13), it is a useful diagnostic tool for clinical estimation of high CVPs, in general, and certainly this method aids student understanding of how the cardiovascular system integrates overall. In this experiment, we assess the right external jugular vein because it is more easily visualized than the internal jugular vein and has also been shown to be reliable in CVP determination (1, 17).

The central venous pressure waveform. Pulses seen within the right external jugular vein do not arise from the vein itself but are reflective of pressure changes with the right atrium during the cardiac cycle. The waveform is more easily visualized in athletic individuals whose resting heart rate and body fat percentage are both low. Figure 2 depicts the venous pressure waveform. The “a” wave corresponds to atrial systole and correlates with the P wave of the ECG. It increases when right ventricular end-diastolic pressure increases. The “c” wave is difficult to see. It is due to the closure of the tricuspid valve and slight bulging of the valve leaflets into the right atrium during the isovolumetric contraction phase of right ventricular systole. The “c” wave correlates with the end of the QRS complex on the ECG. The “y” wave coincides with the time of maximal atrial filling before the atrioventricular valves open. It correlates with the end of the T wave on the ECG. Note: two descending waves occur: “x” corresponds to atrial relaxation, and “y” to the end of ventricular systole when the tricuspid valve opens. These may be visualized as slight inward impressions of the skin of the anterior triangle of the neck. Understanding the venous pressure waves and how they relate to the cardiac cycle is integral to the accurate interpretation of the CVP waveform overall.

Learning Outcomes

After completing this activity, the student will be able to:
1. Estimate venous blood pressures in different body regions.
2. Appreciate why venous pressure varies in different body regions.
3. Describe how venous blood pressure is affected by posture.
4. Briefly discuss a number of factors that influence venous return.
5. Assess the normal function of venous valves.
6. Determine the impact of the skeletal muscle pump on venous return.
7. Estimate CVP.
8. Appreciate the significance of high and low CVPs.

Activity Level

This activity is suitable for students studying a variety of courses including physiology, anatomy and physiology, human biology, biomedical science, medicine, nursing and the allied health sciences. Currently, this activity is undertaken by our first year undergraduate medical, dental, pharmacy, biomedical sciences and human biology students. We have been facilitating this practical for well over 20 yr.

Prerequisite Student Knowledge or Skills

Before doing this activity, students should have a basic understanding of:
1. Blood pressure regulation.
2. Cardiac output regulation.
3. Total peripheral resistance regulation.
4. The significance of preload and afterload to blood pressure control.
5. The arterial and venous systems, including the functional anatomy of each.
6. The concept of compliance.
7. Regulation of venomotor tone.
8. Physiological factors that govern venous return.
9. Factors regulating CVP and/or RAP.

Time Required

The overall duration is 2 h. The suggested breakdown of time is as follows:

- **Experiment 1**: ~20 min
- **Experiment 2**: ~15 min
- **Experiment 3**: ~20 min
- **Experiment 4**: ~25 min
- **Experiment 5**: ~25 min
- **Experiment 6**: ~15 min

METHODS

Equipment and Supplies

The following equipment and supplies are needed:

- 1. Reclining bed, if available, or bench
- 2. Ruler

Human or Animal Subjects

This noninvasive experiment, as performed during practical classes, does not require ethical approval at Queen’s University Belfast; however, all students are invited to volunteer as a subject for practical classes, including this one, and informed, written consent is obtained. Students are made aware that there is no displeasure or disadvantage should they not want to volunteer. Adopters of this activity are responsible for obtaining permission for human research from their home institution. For a summary of Guiding Principles for Research Involving Animals and Human Beings, please see https://www.physiology.org/author-info.animal-and-human-research.

Instructions

It would be prudent to inform student volunteers about the requirements of this practical class before it takes place.

Students with prominent veins make ideal subjects for these experiments.

Visualization of the venous waveform is more easily attained in athletic individuals whose resting heart rate and body fat percentage are both low.

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**Table 1. Note the degree of distension of veins in each region and make conclusions about the venous pressure**

| Body Part                  | Degree of Distension | Conclusion about Pressure in Veins |
|----------------------------|----------------------|-----------------------------------|
| Neck                       |                      |                                   |
| Back of hand               |                      |                                   |
| Dorsum of foot             |                      |                                   |

Any student with heart conditions should be excluded as a subject.

**Experiment 1: Veins in different regions.** The instructions for experiment 1 are as follows:

1. Loosen or remove clothing so that the subject’s neck, back of the hand, and feet are clearly visible.
2. Instruct the subject to stand as relaxed as possible, supported at a wall.
3. Observe the veins in the neck, on the back of the hands, and the dorsum of the feet.
4. Note the degree of distension in the veins of each region.
5. Palpate the veins to classify the degree of distension (distended/partly distended/collapsed) and pressure (high/medium/low) of the veins within each region.
6. Student observers should note their evaluations in Table 1. The student can then be instructed to lie down. Compare the degree of distension when supine with standing upright.

**Experiment 2: Venous valves.** The instructions for experiment 2 are as follows (see Fig. 3 for representational photographs):

1. Select a long vein in the forearm and let it fill by lowering the arm (Fig. 3A).
2. Apply pressure to a segment with one index finger. This compresses the vein. At the same time, with the thumb of the same hand swipe the blood out of the vein upwards, i.e., toward the heart (Fig. 3B). If there is a valve in the vein segment, the blood will not flow back when the thumb is lifted (Fig. 3C). If the blood does flow retrogradely, move to another section of vein or move the index finger along the vein, i.e., away from the heart. The position of the valve should be seen clearly.
3. Test the adequacy of the valve by trying to force the blood past it and away from the heart. To do this, swipe your thumb along the section of vein toward your index finger.
4. Release the index finger and observe the empty section of vein filling from below.

**Experiment 3: Effect of the skeletal muscle pump.** The instructions for experiment 3 are as follows:

1. Ask the subject to remove his/her shoes and socks.
2. Ask the subject to slowly complete a number of heel raises or walk on the spot slowly.
3. Observe the emptying of the veins on the dorsum of the feet, i.e., they become less distended as the subject completes the exercises.
4. Student observers should be able to discuss why this occurs.

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Fig. 3. Illustration of the presence and function of venous valves. A: identify a long vein in the forearm. In some individuals, valves (V) can be visualized through the skin. B: apply pressure to a segment of vein with the index finger and swipe the blood toward the heart with the thumb. C: remove the thumb. The valve prevents backflow of blood, and the vein segment distal to the valve remains collapsed.
Experiment 4: Peripheral vein collapse as an estimate of CVP. The instructions for experiment 4 are as follows (see Fig. 4 for representational photographs):

1. Allow the arm to hang limply down until the veins fill and are distended. If the observer cannot visualize the veins, then this technique cannot be used.
2. Slowly and passively elevate the subject’s arm. Supporting the arm ensures that the skeletal muscle pump is not active.
3. Observe the veins on the dorsum of the hand and note the level of the hand at which they collapse.
4. Measure the height between the sternal angle and the hand when the veins are collapsed.

Experiment 5: Pressure in the neck veins and central venous pressure waves. The instructions for experiment 5 are as follows (see Fig. 5 for representational photographs):

1. Loosen clothing around the neck.
2. Lie the subject semirecumbent at an angle between 30° and 45° from the horizontal with the head resting on a pillow.
3. Adjust the reclining angle until the jugular vein becomes distended with the top of the column of blood (the meniscus) visible in the neck.
4. Note the point at which the jugular vein collapses, which is just above the meniscus.
5. Note that the sternal angle (an indicator for the right atrium) is 5 cm above the midpoint of the right atrium in an adult.
6. Use a ruler to measure the height between the right atrium and the point of vein collapse, i.e., CVP or RAP is equal to the vertical height of the column of blood above the sternal angle plus 5 cm (see Fig. 5B).
7. A JVP of 9 cmH₂O is considered to be the upper limit of normal (13).

Experiment 6: The venous pressure waveform. The instructions for experiment 6 are as follows:

1. Leave the subject in the same position as for the previous experiment.
2. Feel for the radial pulse at the wrist, and for every arterial pulsation you should discern two of the three venous pulsations in the jugular vein in the neck.
3. Student observers should be able to explain these waves.

Troubleshooting

Subjects should try to remain relaxed at all times.

Background noise should be kept to a minimum during measurement, and students should not speak, whether they are observers or subjects, during blood pressure measurement, as this can have a noticeable effect on venous pressure and confound observations.

When observing the venous pressure waveform, care should be taken to identify the jugular venous pulse and not the carotid pulse, which is located close to the internal jugular vein. The carotid pulse can be palpated, the venous pulse disappears on palpation. The venous pulse changes with respiration; the arterial pulse does not.

Safety Considerations

Standard hygiene measures should be followed, including hand-washing procedures before and after palpating the subject.

RESULTS

Expected Results

Inquiry applications. QUESTION 1: COMMENT ON THE DEGREE OF DISTENSION OF VEINS IN DIFFERENT REGIONS AND EXPLAIN THE OBSERVED DIFFERENCES. YOU MAY WISH TO USE YOUR NOTES FROM TABLE 1 TO INFORM YOUR OBSERVATIONS. In this experiment, students observe the degree of distension of veins at the neck,
back of the hand, and the dorsum of the feet. The veins in the neck will be undistended, reflecting low or negative pressure in veins above the heart. Those in the hands are distended, and in the feet even more distended, reflecting the increasing effect of gravity and hydrostatic pressure on the blood in the vessels below the heart. Palpation enables students to roughly compare the degree of distension and, therefore, the venous pressures at each region. Students should have an awareness of how posture affects hydrostatic pressure at each region.

**QUESTION 2:** WHAT WOULD BE THE CONSEQUENCES OF AN INEFFECTUAL SKELETAL MUSCLE PUMP? HOW DO VENOUS VALVES CONTRIBUTE TO THE WORK OF THE SKELETAL MUSCLE PUMP? Students should demonstrate an appreciation of hydrostatic pressure in the upright person, the tendency for venous pooling in extremities below the heart, and the relative difficulty in overcoming the low pressure gradient returning blood to the heart. They should recognize the mitigating role of the combination of venous valves and skeletal muscle contraction (the muscle pump) that propels the blood back toward the heart, reducing venous pooling. With an inadequate skeletal muscle pump, venous pooling may lead to extravascular edema, reduced blood volume, and limited venous return to the heart. Limiting venous return leads to reduced cardiac output and mean arterial blood pressure.

An inactive person standing still is subject to the full hydrostatic pressure gradient in the venous system, and pressure in the foot veins will be ~90 mmHg. Valves are integral to alleviating this maximum pressure when movement occurs. Incompetent valves lead to venous distension and the formation of varicose veins: unsightly, tortuous and swollen veins.

**QUESTION 3:** AT SOME POINT IN A PERSON’S LIFE, HE/SHE WILL EXPERIENCE LIGHT-HEADEDNESS WHEN MOVING FROM A SUPINE TO UPRIGHT POSITION. COMMENT ON WHY THIS MAY OCCUR. Without reflex compensation, moving from a supine to a standing position (with venous pooling) reduces venous return, CVP, preload, stroke volume, cardiac output, and, consequently, arterial blood pressure. Students should be familiar with the normal compensatory mechanisms that maintain blood pressure (principally via the baroreceptors and autonomic nervous system) and appreciate that, if these are overwhelmed, then arterial blood pressure declines and there is an immediate decrease in blood flow to the brain. The transient drop in arterial pressure (within 30 s or so) is thought to be due to a temporary mismatch between cardiac output and systemic vascular resistance responses. Orthostatic/postural hypotension or syncope occurs when there are inadequate adjustments to postural change. Prolonged standing may also lead to transcapillary filtration, which depletes effective circulating blood volume and further compounds a diminished venous return.

**QUESTION 4:** WHY IS FAINTING COMMON WHEN STANDING STILL ON A WARM DAY? If a person is not using his/her skeletal muscle pump, then venous pooling occurs. In warm weather, this venous pooling is exacerbated due to venodilation, transcapillary filtration, and potential dehydration, which can lead to cerebral ischemia and syncope or fainting. Soldiers on guard duty are particularly susceptible to fainting if they do not contract their calf muscles regularly to activate the skeletal muscle pump, as are other professionals, for example, teachers and hairdressers who may be standing for long periods of time. Older people, especially when dehydrated, may also be so affected.

**QUESTION 5:** LIST AND EXPLAIN SOME CAUSES OF HIGH AND LOW CVP. Produce this list in conjunction with the background information given in the main body of the article. Note: a high CVP indicates increased passive diastolic filling of the right ventricle, but can also indicate reduced venous inflow. The opposite is true for low CVP.

High: hypervolemia, increased venous volume, large blood transfusion, right-sided heart failure, pulmonary congestion, Valsalva maneuver, pneumothorax.

Low: hypovolemia, venodilation.

**QUESTION 6:** WHY IS CVP AN APPROXIMATION OF RAP AND PRELOAD? HOW MIGHT A KNOWLEDGE OF CVP BE USEFUL IN DETERMINING THE CAUSE OF AN ARTERIAL BLOOD PRESSURE DROP WHEN CARDIAC OUTPUT IS DECREASED? CVP is the pressure in the central veins where they empty into the right atrium and is regulated by the balance between the blood entering and subsequently leaving the right side of the heart and right ventricular function. CVP is an indicator of right ventricular end-diastolic pressure and hence the degree of stretch on cardiac myocytes, i.e., right ventricular preload (when ventricular compliance is normal and in the absence of tricuspid stenosis). If, for example, blood pressure falls, by an approximation of Ohm’s law, we know that this is due to either a drop in cardiac output or peripheral resistance. If cardiac output is decreased, CVP allows us to determine whether this is due to a decrease in cardiac function or venous return. If the decline in cardiac output is accompanied by an elevation in CVP, then this would be due, in general, to a decline in cardiac function and diagnostics would focus on determining why cardiac function was reduced. If CVP is low, then principally the venous return is reduced and treatment would focus on providing more volume.

**QUESTION 7:** CENTRAL VENOUS PRESSURE IS ELEVATED IN PATIENTS WITH RIGHT VENTRICULAR FAILURE. WHY IS THIS? EXPLAIN WHY ONE OF THE TELL-TALE SIGNS OF HEART FAILURE IS EDEMA OF THE LOWER EXTREMITIES. Right ventricular failure means that there is a failure to maintain adequate cardiac output from the right side of the heart. The most common cause of right ventricular failure is pulmonary hypertension, which is associated with respiratory disease and/or with left-sided heart failure. When the right ventricle cannot pump enough blood to the lungs, the CVP rises and blood backs up into the body’s veins. This increases capillary hydrostatic pressure at the venular end of the capillary and alters the balance of Starling forces, such that more fluid is filtered out of the capillary than normal. This excess filtered fluid accumulates in the interstitium and leads to the swollen lower limbs associated with right-sided heart failure. The accumulation of fluid in the interstitium is known as edema. Elevation of the feet helps to reduce edema by lowering venous pressure and hence reducing venous filtration. Compression stockings can also be worn to relieve swelling. They work by increasing interstitial hydrostatic pressure and forcing the excess filtered fluid back into the plasma compartment.

**QUESTION 8:** HOW MIGHT YOU EXPECT THE CVP WAVEFORM TO VARY WITH THE FOLLOWING PATHOLOGIES: 1) ATRIAL FIBRILLATION, 2) TRICUSPID REGURGITATION, 3) TRICUSPID STENOSIS, AND 4) PULMONARY HYPERTENSION? The answers are as follows:

1. Absence of “a” waves.
2. “c” and “v” waves fuse together.
3. “a” wave elevated.
4. “a” wave will be elevated; “v” wave may also be affected.

**Wider Educational Applications**

A knowledge of the venous system can form a broader understanding of the role of venous return in maintaining cardiac output via the Frank-Starling mechanism, along with the maintenance of blood volume and Starling’s forces along blood vessels. In terms of cardiovascular dynamics, the content of this paper could be extended by including a study of cardiac function curves and venous return curves to further explore how cardiac and vascular function are coupled. This understanding is essential to discussions of the pathophysiology and consequences of cardiovascular disease, such as heart failure and edema formation, and the basis for treatment.

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**DISCLOSURES**

No conflicts of interest, financial or otherwise, are declared by the authors.

**AUTHOR CONTRIBUTIONS**

E.A.T., S.M.R., and C.D.J. conceived and designed research; E.A.T., S.M.R., and C.D.J. interpreted results of experiments; E.A.T. and C.D.J. prepared figures; E.A.T. drafted manuscript; E.A.T., L.E.A.M., J.G.Q., S.M.R., and C.D.J. edited and revised manuscript; E.A.T., L.E.A.M., J.G.Q., S.M.R., and C.D.J. approved final version of manuscript.

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