

Relationship Between Pressure and Flow in the Arterial System of the Lower Extremity: A Discussion of Current Paradigms and Novel Concepts

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ABSTRACT This review summarizes a personal and practical experience with concepts and measurements of pressure and flow in the lower extremity. The anatomy and basic physiologic characteristics of blood circulation are described. Mean pressure and mean flow lead to the concept of resistance. Pulsatile pressure and pulsatile flow introduce the concept of impedance. Practical measurements of these variables are discussed with potential applications.

Introduction

Arteries of the lower extremity transport blood to tissues such as muscles, skin, and bones and nerves of the thigh, knee, calf, and foot. This transport equates to movement of blood. The variables that describe blood movement encompass the field of hemodynamics. In the vascular laboratory, hemodynamic evaluation has concentrated on observations of pressure, pulse, or velocity waveforms and to measurements of systolic pressure and maximum peak systolic velocity. Although these measurements imply pulsatility of the peripheral arterial system, most concepts are limited to a basic model relating mean pressure to mean volumetric flow rate and resistance. This model is the most taught and learned, despite the fact that none of its variables is commonly measured in most vascular laboratories. In this review, these concepts are expanded to a discussion of pulsatile pressure, pulsatile flow rate, and pulsatile resistance or impedance. These topics are associated with vascular laboratory measurements and with the understanding of vascular pathologic conditions and treatment.

The heart through a diverse network of arteries, capillaries, and venous conduits propels blood. Three major problems can alter effective blood distribution: arterial blockage, capillary ineffectiveness, and venous blockage. The objective of this article is to focus on the arteries. Abnormalities in the capillaries and veins, however, also affect arterial performance and will be briefly discussed.

Several hemodynamic variables are well known in

the vascular laboratory. Traditionally, the examiner's fingers have been used as the detector of arterial pulses. Sensitive electromechanical transducers have been designed to improve and normalize pulse recording. This pulse has been related to blood pressure and to blood volume. Piezoelectric transducers permit detection of blood movement with time, that is, the measurement of blood velocity. Doppler velocity detectors have dominated lower extremity vascular testing since the 1960s.¹ The relationship between pulse, or pulse pressure, and velocity, or flow velocity, provides the intuitive concept of peripheral resistance. Good pulse and high flow give the impression of low resistance. A low flow state reflects high resistance. One of the causes of high resistance is arterial blockage. This blockage causes the distal pulse to decrease. Therefore, peripheral arterial occlusive disease has been linked to decreases in pressure and flow. The following sections formalize some of these observations and intuitive concepts.

Peripheral Circulation

The heart feeds blood to the aortic arch, which in turn provides flow to the head and arms. The arch continues as the thoracic and abdominal aorta. From the abdominal aorta blood flow is diverged into hepatic, splenic, mesenteric, renal, and lumbar vessels. The abdominal aorta bifurcates, and the pelvis and lower extremities are fed by way of the iliac and femoral arteries. These major channels reach the thigh as the common femoral artery. The common femoral artery bifurcates, with the deep femoral artery feeding primarily the thigh, whereas the superficial femoral artery continues in the direction of the calf. Above the knee, the superficial femoral artery becomes the popliteal artery. Below the knee, the popliteal artery tri-

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furcates and feeds the individual compartments of the leg. The anterior tibial artery branches first and then a short trunk is subdivided into the posterior tibial and peroneal arteries. The anterior and the posterior tibial arteries reach the foot directly as the dorsal pedal and plantar arteries, respectively. All these major arteries yield branches that create a network capable of collateral flow that may be needed during exercise or in the presence of occlusive disease.

The arterial network delivers blood to the capillaries in the tissue. Most of us think of capillaries as another tubular network of vessels connecting arteries and veins named arterioles and venules. Some of these tubes have valves that open and close, allowing for intermittent feeding, and others are arteriovenous shunts that bypass the tissue. An expanded concept does not classify capillaries as having arterial or venous sites. Capillaries are an integral part of tissue that open and close to the arterial side to receive blood. The blood is processed, with the tissue taking out needed nutrients and then dumping waste. Finally, the capillaries open and close to the venous side to expel blood. This blood returns to the circulation by way of the veins to be cleaned by multiple organs and returns to the periphery recirculating again by means of the arteries.

The circulation requires energy that is primarily created by the heart. A driving pressure produces the flow according to the peripheral resistance to flow. These concepts are detailed later. A note of caution, however, is needed. Although the peripheral circulation maintains tissue viability primarily, other functions such as temperature control by means of skin flow also take place. These multiple tasks confuse the understanding and interpretation of hemodynamic changes, particularly of blood flow.

Blood Pressure

Systolic blood pressures are measured at the posterior and anterior tibial arteries at the ankle. These pressures are compared with the brachial systolic pressure and an index, referred to as the ankle/brachial index (ABI), is calculated. If systolic ankle pressure is less than systolic arm pressure (ABI less than one), arterial obstruction is suspected. If systolic ankle pressure is less than half of the systolic brachial pressure (ABI <0.5), severe arterial blockage is diagnosed, and often, an operative procedure is indicated. Curiosity raises the following questions:

- (1) Why do we measure only systolic pressure at the ankle when classical measurement of arm pressure includes systolic and diastolic pressures?
- (2) Is there any value in the measurement of mean, diastolic, or pulse pressures?
- (3) Why do we measure systolic pressure during deflation of the cuff? Is there any reason not to make this measurement while inflating the cuff?
- (4) Traditionally, the higher of the posterior or anterior tibial pressure has been used to represent the

status of the distal circulation. Why? Why not the lower or both?

Several Doppler techniques were tested to measure diastolic pressure without success, and because of technical deficiencies we were forced to measure systolic pressure only. We measure systolic pressure with a Doppler velocity detector primarily. Strain-gauge, air, or photo plethysmographs provide similar information.² The ease of these techniques is detection of the onset of flow.

In the 1980's an oscillometric technique was successfully developed to monitor systolic, mean, and diastolic pressures.³ This instrument has invaded virtually all areas of hospitals and clinics. Can this technique be used to measure ankle pressures? Yes, but with one major caveat: pulse must be present. The problem is not necessarily low pressure, it is low *pulse* pressure. This technique detects pulse pressures as the cuff pressure is varied from suprasystolic to infradiastolic pressures. Transmitted pulses may be present in the cuff even if flow is occluded. Maximum pulse is detected if mean pressure is applied to the cuff. Pulse amplitude is stable for infradiastolic cuff pressures. Algorithms take these premises into consideration to extrapolate the values of systolic, mean, and diastolic pressures. Some of our work with this technique, mostly unpublished, has addressed the following items:

- (1) Are systolic ankle pressures measured with oscillometry similar to Doppler pressures?
- (2) Can oscillometry be used to measure pressures in patients with incompressible or partially compressible ankle arteries?
- (3) What is the value inherent in mean and diastolic pressure determinations?

Our findings revealed that ankle systolic pressures by Doppler were statistically significantly higher than by oscillometry, with a mean difference of 2 mmHg. Most measurements were either equal or the Doppler was consistently higher by 5 mmHg. Although paired *t*-test showed a statistical significance, there was no real clinical implication. Therefore, the oscillometric technique is a viable method to measure ankle pressures in patients with detectable pulses. A major advantage of the oscillometric technique is that it eliminates operator variability, sometimes an important factor in large laboratories with several technologists, new personnel in training, and/or personnel changes.

We also determined that pressures could be estimated with oscillometry in patients with total arterial incompressibility. A possible explanation is that pulse amplitudes still vary with varying applied cuff pressure. Therefore, mean pressure can be determined, and systolic pressure may be estimated by extrapolation of the pulse amplitude envelope.

Yes, we can measure mean and diastolic ankle pressures in many patients. So, what is the added value? Apparently none. Systolic pressure is more sensitive to the presence of arterial blockage than mean or diastolic pressure. Systolic ABI had to decrease to about

0.7 before a significant decrease in mean or diastolic ABI was noted. In the presence of disease, the systolic ABI was the lowest ABI compared with the mean or diastolic pressure ABI. Pulse pressure is altered in disease to maintain mean pressure at normal levels. Only significant obstruction results in decreased mean pressures. In some patients with unsuspected partial arterial incompressibility, however, the measurement of mean and/or diastolic ankle pressures may be of value. Total incompressibility results in flow that is detectable despite cuff inflation to high pressures. Partial incompressibility is suspected if the measured ABI is greater than 1.3 or 1.5. An ABI of 0.7, for example, may be falsely elevated. We suspect false systolic ABI determinations if the ABI based on mean pressures is lower than the systolic ABI.

Further discussion on the potential usefulness of pulse pressure determinations is presented later under "Peripheral Impedance". We still have to address two of the four original questions.

Doppler pressures are measured on deflation as a legacy from the auscultatory method of obtaining brachial pressures. The deflation method is preceded by a period of rapid cuff inflation, which can potentially displace the artery. In reality, measuring during inflation may be more appropriate, particularly in the presence of small, diseased arteries at the ankle level. Gradual increments of pressure during cuff inflation minimize error, and the arterial signal is never lost until true systole. Problem: *during inflation*, fluctuations of systolic pressure with respiration, for example, may confuse selection. Obtaining pressures during deflation is the classic "ostrich, head-in-the-sand" excuse that ignores more complete information in favor of "not having to make a decision at all."

One measurement has a variability of about 10% and does not represent the mean systolic pressure. The average of two determinations decreases the variability error from 10 to 7%. Why not make two determinations: one on inflation, one on deflation?

The highest ankle pressure has been used to describe the status of the peripheral arterial network. Such practice may not be based on solid data but on the assumption that it is the highest pressure that drives flow distally. Flow rate data collected at Saint Joseph's Hospital in Burbank, however, indicated that distal flow was compatible with the highest pressure in 30%, with both pressures in 20%, and with the lowest pressure in 50% of the cases with different ankle pressures. If only one value has to be selected rather than both, our data suggest that the lowest ABI is more compatible with distal flow.

Blood Flow

We have measured volumetric blood flow rate in the leg with electromagnetic, magnetic resonance, and ultrasonographic techniques.⁴⁻³⁰ The most practical technique nowadays is an ultrasonographic technique that detects flow velocity waveforms and diameter, calculates average velocity and vessel area, and multiplies both to estimate volumetric blood flow rate.³⁰

Our learning experience with thousands of measurements is summarized in the following.

Most in the vascular laboratory equate blood flow to velocity measurements. This tendency is a result of training to detect increased velocities at carotid or bypass graft stenoses. The peripheral arterial system, however, was designed to provide a certain amount of blood in a certain period of time. The most important variable measured during peripheral arterial studies should be volumetric blood flow rate. Measuring velocities, or pressures, is a weak substitute that is equivalent to paying the water bill in units of pressure or speed. This is clear absurdity. Why don't we measure volumetric blood flow rates consistently? Some mention lack of measurement reliability. This factor is true, but only to a certain degree. Pressure measurements have errors of about 5%, depending on instrument resolution, visual extrapolation of the markers, and inflation/deflation rates. Flow rate measurement errors are similar. Physiologic variability, however, hinders analysis of flow rate data more so than pressure data. Flow is highly sensitive to changes in peripheral conditions. Although the body tries to maintain constant blood pressure, flow is the variable that adapts. We have demonstrated significant increase in flow rate to the lower extremities after balloon dilatation of arterial stenosis in patients with either unchanged or normal ABIs at rest.^{31,32} Unfortunately the analysis of resting flow data has poor specificity. Maneuvers to change flow and increase specificity must be used. A change in pressure of approximately 10% or more can be considered significant for arterial obstruction, whereas flow should change by more than 20% or even 30%.

To improve analysis of flow rate data, flow should be decomposed into pulsatile and diastolic flow components. The diastolic component represents mostly capillary demand. Diastolic flow increases with peripheral inflammation and infection. Pulsatile flow, in contrast, is primarily a reflection of the arterial conditions (i.e., of the large arteries [1–2 mm in diameter or larger]). If the arteries are the focus of the noninvasive evaluation, the examination should concentrate on pulsatile flow rate, primarily because this is the flow rate that must feed the periphery in the worst-case conditions of vasoconstriction.

We have related pulsatile flow to ABI. A linear relationship was found between mean pulsatile flow and ABI in vascular patients. A flow rate of 60 ml/min at the upper calf level correlated with an ABI equal to 1, whereas a flow rate of 30 ml/min correlated with an ABI of 0.5. A claudicator with a decreased ABI has decreased resting flow!³³ If such correlation was seen in most patients, flow rates would not need to be measured. However, about 20% of the patients had flow rates significantly above expectation, and about 20% of the patients had flow rates significantly below expectation. Therefore, flow rate measurement may add novel information in only about 40% of the cases. The linear correlation between leg flow and ABI was unexpected. Classical teaching indicates that flow remains constant until a stenosis greater than 50% in

diameter results in a significant decrease in flow rates. These data, however, are primarily based on in vitro studies, and it was not confirmed in our noninvasive, in vivo data collection.

Most expect flow rate to be proportional to leg volume. Reporting flow rate per volume in ml/min/ml is traditional. This assumption is not clearly observed in practice. Published data rarely show correlation better than 60% between flow rate and leg volume. The explanation is simple. Flow rates to muscle, skin, and bone are quite distinct. Legs have different components of muscle, skin, and bone volume. Flow rate per volume could make sense only if decomposed in muscle, skin, and bone flow rates and volumes. This is an impractical task.

We have measured 60 ml/min as normal infrapopliteal flow in the elderly. Other studies have documented flows of about 100 ml/min in normal popliteal arteries and in bypass grafts extended to the popliteal artery. At present, we use 50 ml/min as a threshold for low popliteal flow rate. However, variations exist. On average, women had 10% less flow than men belonging to a population referred to a vascular laboratory. Cold feet have minimal flow rate. This finding brings another assumption into question. Many believe in autoregulation with constant flow rates. This is perhaps a concept applicable to other organs but not to the peripheral circulation. Cold extremities are not necessarily the result of arterial blockage or abnormal neural tone. It may be a sign of extremely effective use of blood supply. Very little blood may be necessary to maintain a viable extremity. Our assumptions must then include another factor yet to be evaluated in vascular laboratories: the effective transmission of substances between blood trapped in the capillaries and tissue. This subject may not be an issue for the surgically-oriented vascular laboratory with a desire to detect blockage and support procedures in diseased arteries. It may, however, become fundamental for a vascular medicine-oriented laboratory that focuses primarily in true tissue perfusion. Lower extremity flow autoregulation, therefore, does not mean constant flow but probably means ability to have flow on demand. It is the author's impression that the lower extremity arterial system was over dimensioned. There are more arteries supplying more flow rates than actually needed under usual conditions. With all these alternate conditions, I recommend a two-tier analysis: (1) low flow rate, and (2) a significant decrease in flow rate greater than 20% as indicators for further evaluation.

Resistance

The classical concept of hemodynamic resistance indicates that flow rate (Q) from one point to another depends on the pressure differential and the resistance to flow between the two points ($P_1 - P_2 = Q \times R$). The resistance increases with increased blood viscosity and increased length between the two points but decreases significantly with increased diameter. This concept is useful for basic understanding but fails to

include fundamental characteristics of the human arterial system: pulsatility and viscoelasticity. Later, we present some additional concepts to broaden our perspective from resistance to impedance.

As mentioned before, a linear relation was obtained between average flow rate and ABIs. The meaning of such a finding is unclear. Perhaps the distal arterial network likes to maintain a constant resistance. More so, perhaps these vessels preferentially maintain a constant diameter. To expand on these observations, the phrase "ischemia causes a decrease in resistance" is erroneous. Even the statement "ischemia causes vasodilation" may not be strictly correct. An observation constantly used to support these statements is the reactive hyperemia that occurs after revascularization.

If one assumes that ischemia is caused by arterial obstruction, the following events may occur. The blockage causes a decrease in ankle pressure. This decrease in arterial pressure causes the diameter to decrease. A decrease in diameter causes the peripheral resistance to increase markedly. In fact, total occlusion is synonymous with infinite resistance. The biochemistry of the arterial wall, however, reacts to a decrease in diameter by releasing vasodilatory substances. Arteries may dilate to their original size, and resistance may remain constant. Once revascularization occurs, pressure is reinstated, diameters increase because of this vasodilatable state, and peripheral resistance decreases temporarily. The arterial wall then stops secreting vasodilators for the diameter and resistance to return to their normal mode. This may be the average behavior, but many patients dilate more or less than average.

Pulsatile Pressure

The pulsatile pressure waveform may be detected, albeit distorted, with air plethysmographs. Its usefulness to evaluate the patients with advanced disease examined in vascular laboratories is qualitative. In practice, it is more appropriate to describe such recordings as pulse volume rather than pressure waveforms. The relationship between pressure and diameter waveforms determines the arterial viscoelasticity. Future research may relate the viscoelastic properties of large arteries with the onset and/or progression of atherosclerosis. Pulsatile pressure, however, is fundamental for the understanding of pulsatile impedance described later.

Pulsatile Flow Rate

Pulsatile flow rate is also fundamental for the understanding of pulsatile impedance. Two concepts, however, deserve mention for their practical applications. Pulsatile flow has been associated with the condition of the large arteries, whereas diastolic flow may represent primarily the state of the capillaries. If the objective is to diagnose the status of the large conduits, only pulsatile flow should be evaluated. The other point is the evaluation of worst-case conditions for bypass grafts or natural arterial supply. The worst

case is represented by maximum vasoconstriction, a condition that may eliminate diastolic flow. Therefore, evaluation of pulsatile flow only may mimic worst-case ischemic conditions.

Pulsatile Impedance

Only great mathematicians are able to analyze and dissect the concept of pulsatile impedance. One must start with the pressure and flow rate waveforms and perform a fast Fourier transformation (FFT) to decompose these waveforms in their frequency components. Each pressure component is divided by the corresponding flow rate component at the same frequency. The result is a graphical representation of the resistance at zero frequency and a multitude of peripheral impedances at the fundamental frequency and its harmonics. In research, it has been shown, for example, that the impedance plot is altered in the presence of arterial obstruction. Perfect recordings of pressure and flow rate waveforms plus FFT analysis are necessary for reliable data collection. It is difficult to envision this method as a practical and successful vascular laboratory evaluation.³⁴

We have developed a simplified formula for investigation of pulsatile impedance. We simply divide pulse pressure by pulse flow rate. Pulse pressure is measured with oscillometric technique as systolic minus diastolic pressure, and pulse flow is calculated in a similar manner from the flow rate waveform (CVI-Q technology). Research is needed to evaluate this simple calculation in cases of arterial obstruction and vasoconstriction or vasodilation.

Summary

Vascular laboratories may consider the use of oscillometry for automatic, operator-independent ankle pressure measurements. Mean and/or diastolic blood pressures may affect the results of a few tests performed in patients with arterial wall calcification. Both anterior and posterior tibial Doppler ankle pressures should affect interpretation of the status of the distal arterial network. These measurements should be obtained during cuff inflation and deflation and then averaged. Degree of stenosis may be assessed with velocity measurements, but flow rate is the variable of importance to determine the status of the peripheral circulation. Despite high sensitivity, our present knowledge renders poor specificity to flow rate determinations. Flow rate changes in a specific individual are more practical observations than absolute value comparisons.

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