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Presence of Lower-Extremity Venous Pulsatility Is Not Always the Result of Cardiac Dysfunction

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ABSTRACT *Introduction.*—**The presence of peripheral venous pulsatility is widely recognized and commonly attributed to cardiac dysfunction; however, the precise cause(s) have been poorly defined in the literature. We have noted multiple patterns of venous pulsatility, often in patients with no history of heart disease. In a normal, high compliance venous system, cardiac pulsatility is continually dampened with distance from the heart. We theorized that in a low-compliance system, the pulsatility would be transmitted throughout and its presence is a reflection of the hemodynamic state of the lower extremity venous system.**

Methods.—**We evaluated the right popliteal vein in 30 normal volunteers (average age 35) with no history of heart disease and minimal venous disease (C-Class range 0–2, average 0.4). The right popliteal vein was chosen for its distal anatomic location and to avoid any potential of left iliac vein compression. Three spectral waveforms were obtained in the following manner: (1) a left lateral supine position with the right popliteal vein at the level of the right atrium, (2) after standing for >3″ with no right leg weight bearing or muscle contraction, and (3) after 10 seconds of vigorous plantar/dorsiflexion.**

Results.— **In a supine position, all 30 limbs had normal respiratory phasicity with minor variation. In every patient, after quiet standing there was very low velocity forward flow with a discernable pulsatility. Immediately after dorsiflexion, flow became highly pulsatile.**

Conclusion.— **A total of 100% of patients demonstrated highly pulsatile flow when the lower extremity venous system was filled to resting hydrostatic pressure. Although cardiac dysfunction may result in venous hypertension, reducing system compliance, the presence of pulsatile venous flow in the lower extremity can be demonstrated in all persons and is therefore a function of venous hemodynamics.**

Introduction

Pulsatile flow in the lower-extremity venous system was first described by Kerr and Warren[1] in 1925 and attributed to congestive heart failure and tricuspid regurgitation. Since then, the presence of peripheral venous pulsatility, especially as demonstrated by ultrasound spectral analysis, has been widely recognized and commonly attributed to cardiac dysfunction, particularly congestive heart failure, tricuspid regurgitation, volume overload, or some form of right heart failure; however, the precise cause(s) have been poorly defined in the literature.[2] We perform a large volume of venous examinations for patients presenting with symptoms of chronic venous insufficiency (CVI). Venous reflux studies typically are performed with the patient in a standing position per guidelines from the American College of Phlebology[3] and the International Union of Phlebology.[4] Patients with CVI are evaluated to determine presence of deep or superficial venous thrombosis or other obstruction as well as determining the presence, severity, and location of any venous valvular incompetence. The spectral waveform in the lower-extremity venous system is also carefully evaluated for its characteristics and presence of cardiac pulsatility is noted. Although sometimes attributable to right heart dysfunction, we regularly encountered pulsatile flow patterns in patients with no clear evidence of right heart abnormality. Additionally, we have noted multiple patterns of venous pulsatility, all of which led us to theorize that the hemodynamic state of the lower-extremity venous system could in fact play a role in these pulsatile flow patterns. Therefore, the primary objective of this study was to explore the etiology of venous pulsatility, challenge the overly simplistic view, and to create a new thought paradigm.

Cardiac pulsatility is commonly identified and considered normal in the upper-extremity venous system, generally attributed to their location in proximity to the heart. As the distance from the heart increases, this pulsatility is dampened until flow in the lower extremities is typically described as spontaneous with respiratory phasicity. In a normally functioning venous system, typical venous pressure in the extremity is approximately 15 mm/Hg. When considering the hydrostatic pressure

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of the column of blood from the heart, the right atrium is considered the "reference," and although not completely accurate, is generally considered to be 0 mm/Hg. In actuality, right atrial pressure varies cyclically, and the mean pressure typically ranges between 2 and 8 mm/Hg, yielding a "working" pressure gradient of approximately 10 mm/Hg.[5] However, in an upright patient, the hydrostatic pressure of the column of blood comes into play. Hydrostatic pressure is the result of the weight of the blood column below the right atrium and is determined by the density of blood and the acceleration of gravity. Hydrostatic and gravitational pressures are expressed as a constant multiplier of the vertical distance in cm below the atrium. The pressure is greatest in the upright (sitting or standing) but motionless individual.[6] At 0.77 mmHg/cm (≈2 mm/Hg per inch of height), venous pressures in excess of 100 mm/Hg can occur in two scenarios. First, in a normal system, equilibrium is reached after several minutes of quiet standing and the venous pressure will approximate the hydrostatic column.[7] With activation of the calf muscle pump, pressure will rapidly decrease to approximately 15 mm/Hg. Second, when the venous valves fail as in CVI, the hydrostatic pressure will result in venous hypertension and quickly distends the veins and venules with a significant lowering of compliance and ultimately capacitance. In either of these two scenarios, cardiac pulsation is no longer dampened, and we would expect to see a pulsatile flow pattern similar to the upper extremity.

Methods

To test this hypothesis, we evaluated the right popliteal vein in 30 volunteers (average age 35 years, range 23–56) with no history of heart disease and minimal venous disease (C-Class average 0.4, range 0–2). Evaluation was performed with patient in two positions, supine and standing. Credentialed technologists (Registered Vascular Technologists and/or Registered Phlebology Sonographers) operating within a vascular laboratory accredited by the Intersocietal Commission for the Accreditation of Vascular Laboratories for peripheral venous studies performed the studies. The right popliteal vein was chosen for its ease of study, its distal anatomic location, and to avoid any potential of left iliac vein compression. Multiple spectral waveforms were obtained for each of the following conditions: (1) a left lateral supine position with the right popliteal vein at the level of the right atrium, (2) after standing for >3" with no right leg weight bearing or muscle contraction, and (3) after 10 seconds of vigorous plantar/dorsiflexion. Most studies suggest that the resting hydrostatic pressure is reached after approximately 1"; however, we arbitrarily chose 3" or standing to ensure complete filling of the hydrostatic column.[6] The subject was observed during the standing period to ensure no weight bearing or muscular contraction.

Results

When the patients were in a supine position, all 30 limbs had normal respiratory phasicity with minor variation (Figure 1). In every patient, after quiet standing there was very low velocity forward flow with a discernible pulsatility (Figure 2). Immediately after dorsiflexion, flow became highly pulsatile in 100% of patients when the lower-extremity venous system was filled to resting hydrostatic pressure (Figure 3).

We therefore conclude that although cardiac dysfunction may result in venous hypertension reducing system compliance, the presence of pulsatile venous flow in the lower extremity can be demonstrated in all persons and is therefore a function of venous hemodynamics.

Discussion

Normal Venous Flow Patterns

Normal venous flow is primarily affected by two factors: (1) initially by back-pulsations that result from cardiac movements and (2) the intra-thoracic and intra-abdominal pressure changes, which are respiratory phases. Venous waveforms can be described as comprising **S**, **v**, **D**, and **a** waves (Figure 4). **S** is the systolic wave resulting from negative intra-arterial pressure with movement of the atrioventricular septum toward the cardiac apex. The **v** wave is representative of a slowing of antegrade flow and is the result of positive intra-arterial pressure created by filling of the right atrium. **D** is the diastolic wave caused by decreased intra-arterial pressure during tricuspid valve opening. The **a** wave reflects the positive atrial pressures caused by atrial systole. The influence of the respiratory phase on each of these waveform components can vary significantly depending upon the vessel segment and particularly its anatomic location relative to the heart.[8,9]

The lower-extremity venous system normally exhibits a Doppler waveform commonly described as spontaneous with respiratory phasicity. There is a decrease in venous return from the lower extremities during inspiration resulting from a lowering of the diaphragm that increases intra-abdominal pressure. During expiration, there is an increase or augmentation of flow secondary to a lowering of intra-abdominal pressure. In contrast, significant cardiac pulsatility of the spectral waveform typically is encountered in the upper-extremity venous system that is generally attributed to the vessels' proximity to the heart. With normal venous capacitance and compliance, this pressure wave would tend to decrease as the distance from the heart increase so that only respiratory

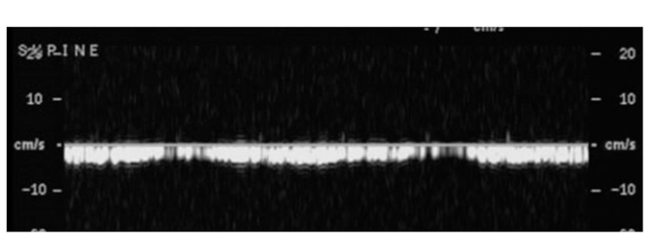

Figure 1

Normal respiratory phasicity in a resting supine position.

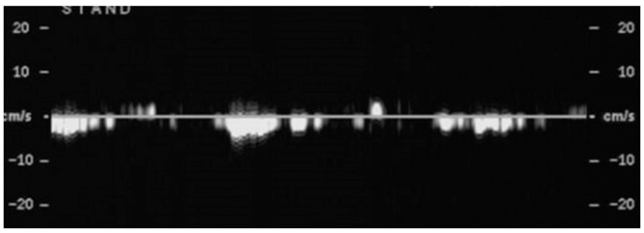

Figure 2

After 3" of motionless standing, spectral analysis revealed a low velocity flow with discernible pulsatility.

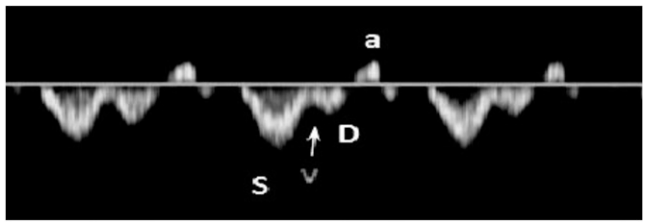

Figure 4

A normal upper extremity venous spectrum, which includes S, v, D, and a waves that represent back-pulsations caused by right atrial pressure changes during the cardiac cycle. This flow pattern is most evident in the veins closest to the heart.

phasicity would be present. Conventional thinking assumes than when the central venous pressures (CVPs) are increased, the pressure wave is presumably transmitted along a further distance through the venous circuit. Deep inspiration may still impact flow, but under normal resting respiration, peripheral cardiac pulsations would be present (A.M. Kupinski, personal communication, 2010).[10] This is an incomplete explanation because blood can generally be assumed to be an "incompressible" fluid and differences in the venous flow patterns are clearly impacted by vessel compliance and capacitance. The venous system, particularly of the lower extremities, normally has a very large capacitance and serves as the primary storage for total blood volume. In fact, more than two thirds of the total blood volume resides in the venous system in a normal patient. Capacitance is defined as a change in volume per change in time or $\Delta V/\Delta t$, where V = volume and t = time. Compliance is similar to capacitance but is also the ability to contain a change in volume but the change in volume is related to changing pressure. Compliance then is defined as a change in volume per change in pressure or $\Delta V/\Delta P$, where V = volume and P = pressure. A normal venous system is said to have both high capacitance and high compliance, i.e., it can accommodate a relatively large change in volume in a relatively short period of time with a small increase in pressure (Figure 5).[11]

It is this highly compliant system that results in a dampening of the pulsatility seen in the lower extremity veins.[5,12] In 2006, Hu et al.[13] used an in vivo cerebral blood flow model to show that a reduction in compliance did in fact increase venous flow pulsatility. Extrapolating that finding to the lower-extremity system, patients with significant venous insufficiency and resultant venous hypertension may demonstrate a similar reduction in compliance.

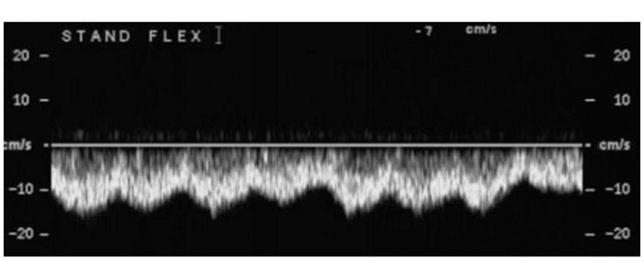

Figure 3

Notable pulsatility after 5–10 sec of rapid dorsiflexion/plantar flexion.

Pulsatile Venous Flow

The presence and characteristics of pulsatile Doppler flow signals has been proposed as a method to determine right atrial pressure. As far back as 1984, Krahenbuhl et al.[14] noted that venous flow became pulsatile and synchronous with cardiac systole when CVP exceeded 7 mm/Hg. In a study involving 46 patients, 13 were found to have a CVP greater than 7 mm/Hg. A total of 12 of 13 patients (sensitivity 92%) had a pulsatile peripheral venous flow whereas only 7 of the 13 (specificity 54%) had clinical signs of right heart failure. Four patients with a normal CVP also were found to have pulsatile peripheral venous flow; however, all suffered from significant valvular heart disease. They concluded the presence of pulsatile venous blood flow was an "effective early sign of right heart failure, more sensitive than clinical evaluation, and probably even more than CVP." Another study from 1996 by Abu-Yousef et al.[15] involved 51 of 343 patients with right atrial pressure measurements

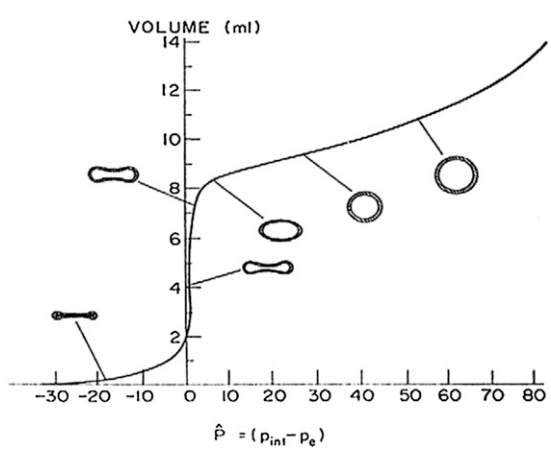

Figure 5

Graph from Katz et al.[11] depicting the pressure/volume relationship in the distensible venous lumen. With low pressure and volume, the vein is nearly collapsed. Note the large volume that can be accommodated without any significant increase in pressure. The vein lumen first becomes elliptical and finally circular as pressure increases.

available for comparison with peripheral venous Doppler waveforms performed within 4 weeks of the right atrium pressure measurement. A total of 17 of 51 (33%) had pulsatile Doppler waveforms whereas 33 (65%) had increased right atrium pressure. Correlation between the presence of these abnormal waveforms and increased right atrium pressure was statistically significant. The specificity of Doppler waveforms for detecting right-sided heart failure as determined by right atrium pressure measurement was very high at 94%; however, sensitivity was unacceptably low at 46%, whereas accuracy was 65%. They concluded that a pulsatile peripheral venous Doppler waveform correlated well with right-sided heart failure, however; because of low sensitivity, it was unacceptable as a screening tool. A follow-up study to this one found that when considered with evidence of "cardiac decompensation" on two consecutive chest radiographs, sensitivity increased to 79%.[16] In a similar study, Cozcolluela et al.[17] looked at 236 patients and demonstrated a "significant inverse relation…. between pulsatile flow and high atrium pressure." However, low sensitivity again precluded any recommendation as a screening tool for increased central venous pressure. They also noted that both cardiac and respiratory phasicity of the venous wave was observed. Tricuspid valve insufficiency also has been proposed as the reason for pulsatility in the lower-extremity venous system and this etiology has been widely reported.[18–20] However, there are also numerous reports of patients with pulsatility who have no evidence of significant tricuspid valve disease. Finally, Klein et al.[21] reported a case of unilateral femoral vein pulsatility in a patient with tricuspid insufficiency.

To determine other potential causes of peripheral venous pulsatility, Abu-Yousef, et al.[22] recruited 12 healthy volunteers (three males, nine females, age range, 21–50 years; mean, 29 years) who were evaluated by careful and extremely detailed spectral Doppler waveform analysis with simultaneous ECG and respiratory tracings. Two blinded observers independently analyzed results. They noted that lower-limb venous Doppler flow showed both cardiac and respiratory effects during quiet breathing. Although respiratory effects disappeared with deep inspiration, the waveform continued to be multiphasic and cardiac. They concluded that cardiac pulsatility, particularly the presence of minimal cyclic retrograde flow of 5 cm/sec or less, does not necessarily indicate cardiac disease and noted that respiratory phases can modulate this cardiac pattern. Given the volume of literature dealing with pulsatile flow in the peripheral veins, two salient facts emerge. First, it is apparent that the underlying etiology of lower-extremity venous pulsatility is complex and probably multifactorial. Second, it is also clear this phenomenon is not fully understood, even though this is a fairly common finding. Finally, any discussion about the position of the patient during examination was conspicuously lacking in all these studies.

In a previous study, we explored this concept from a different perspective. If patients exhibited pulsatile flow as the result of venous hypertension secondary to superficial venous insufficiency, we questioned whether truncal ablation allowed improvement in the venous hypertension and a return to a normal flow pattern.[23] We compared the presence and pattern of lower-extremity venous pulsatility in 293 consecutive patients suffering from CVI (mean CEAP 3.2). Of these, 242 limbs underwent endovenous thermal ablation of a refluxing truncal vein. Duplex ultrasound was used to evaluate these limbs approximately 3 days postprocedure, and the results of 101 limbs were available for comparison. Of the 101 limbs, 47 (46.5%) had evidence of pulsatile flow in the lower-extremity venous system preoperatively. Paired observations using the Wilcoxon method showed no difference in the venous pulsatility post ablation regardless of the presence of deep venous reflux. Although this study failed to confirm the premise that diminished distal capacitance and compliance of the venous system secondary to venous hypertension contributes to the presence or grade of venous pulsatility, there were several potential explanations. It is feasible that venous hypertension is not significantly reversed by ablation of a superficial truncal vein even in patients without evidence of deep venous reflux. Duplex ultrasound or our use of the technology may not be sensitive enough to detect all potential pressure pathways. We strongly considered that our initial follow-up period of 3 days was too short an interval to elicit significant changes in the hemodynamic state of the distal venous system.

Another potential explanation also exists. Several studies have looked at both arterial and venous compliance and capacitance in normotensive and hypertensive patients and found that there may in fact be altered vessel physiology that does not immediately revert after normalization of blood pressure.[24,25] Delaney et al.[26] showed that although venous compliance was similar, hypertensive adults have lower capacitance (volume). They surmised that hypertensive individuals might have altered venous smooth muscle tone compared with normotensive controls. In hypertension, normalization of cardiac output is in large part regulated by the reduction in both arterial and venous compliances. Reduced arterial compliance modulates systolic function of the heart whereas diastolic function is more likely regulated largely by reduced venous compliance. It is therefore quite possible that the decrease in compliance and capacitance is not simply the mechanical consequence of the increased blood pressure but also reflects intrinsic alterations of the vascular wall that may in fact be permanent. If this is so, early and possibly more aggressive treatment of venous hypertension may be prudent. This hypothesis is borne out by a recent study by Lattimer et al.[27] In this observational study, the authors investigated the prevalence and degree of spontaneous pulsation within the great saphenous vein (GSV) in volunteers and patients studied in a standing position by using color duplex and compared this with the presence of reflux and disease severity. They detected pulsatile flow in only 2 of 44 (4.5%) legs with $C_{0–1}$, 9 of 17 (52.9%) legs with $C_{2–3}$, and 16 of 17 (94.1%) legs with $C_{4–6}$ ($p < 0.05$). They also found the median GSV diameter of refluxing GSVs were larger in patients with pulsatile flow than those

without (7 mm [range, 4–9.4] versus 5.1 mm [range, 2.7–8.1], respectively (p = 0.003). Although the presence of cardiac disease was not noted, it seems unlikely there would be a distribution with 94% of C4-6 patients having cardiac disease as compared with only 4% of those with no or minimal disease.

Conclusion

It is clear that the underlying etiology of lower-extremity venous pulsatility is complex and probably multifactorial. The fact that all patients may have pulsatile flow in the absence of any cardiac disease demonstrates that its presence is reflective of the venous system hemodynamics. This must create a paradigm shift in our thinking about the flow patterns commonly identified in the lower extremity venous system. Admittedly, although cardiac dysfunction may be the most frequent cause of clinically significant venous hypertension, a blanket statement that pulsatile flow in the lower-extremity venous system is the result of cardiac abnormality is no longer acceptable. Additional studies that explore both the etiology and natural history of this phenomenon continue to be warranted.

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