Saphenous pulsation on duplex may be a marker of severe chronic superficial venous insufficiency

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Background: Pulsatile flow in deep, perforating veins and varicose veins (VVs) has been described previously to support a hypothesis of arteriovenous (AV) fistulae in the pathogenesis of VVs. Its presence has also been suggested as a cause of failure of VV treatments. However, AV communications have never been adequately visualized and direct pressure tracings within leg veins have been inconclusive. The present study was observational aiming to investigate the prevalence and rate of spontaneous pulsation within the great saphenous vein (GSV) in volunteers and patients using color duplex and compare this to reflux and markers of disease severity.

Methods: Twenty-seven consecutive patients (32 legs, median Venous Clinical Severity Score (VCSS) = 5 (0-11)) attending the VV clinic and 23 consecutive ambulatory normal volunteers (46 legs) had their GSV assessed at midtigh using color duplex. Subjects were examined standing with the hips resting against an adjustable couch, bearing weight on the contralateral leg, with the test leg touching the ground. The presence of flow and reflux were initially determined using manual calf compression. Saphenous pulsation (SP) was defined as a cyclical change in velocity. The GSV diameter and SP rate were then recorded after 2 minutes of dependency. The number of pulsations was counted from video recordings.

Results: The resting SP, if present, was discrete, monophasic, of variable amplitude, antegrade, and irregular, irrespective of respiration. Pulsation was detected in 2/44 (4.5%) legs with C0-1 (C part of CEAP), 9/17 (52.9%) legs with C2-3, and 16/17 (94.1%) legs with C4-6 (P < .05, z test of column proportions). Reflux occurred in 8/32 (25%) legs without SP (C0 = 2, C1 = 1, C2 = 3, C3 = 2). The median GSV diameter was significantly elevated in the presence of SP (no pulse: 3.5 [range, 1.5-8.1] mm; pulse: 7 [range, 4-9.4] mm; P < .0005). The median refluxing GSV diameter in GSV pulsators compared with nonpulsators was 7 (range, 4-9.4) mm; vs 5.1 (range, 2.7-8.1) mm, respectively (P = .003). The median SP rate in refluxing GSVs was 52 (range, 22.95) beats per minute.

Conclusions: The high prevalence of pulsatile antegrade saphenous flow is a novel observation in patients with severe superficial chronic venous insufficiency. It is detectable in 75% of patients with GSV reflux and significantly increases with clinical severity and saphenous diameter. It may be a marker of advanced venous disease and, as it is easy to record, it could supplement duplex evaluations of reflux. Further work is needed to establish the clinical relevance of the SP in terms of disease progression, recurrence after treatment, and as a hemodynamic marker of severity. (J Vasc Surg 2012;56: 1338-43.)

Pulsatile flow in varicose veins (VVs) has been described previously to support a causal hypothesis of arteriovenous (AV) fistulae.1 However, AV communications have never been adequately visualized and direct pressure tracings within leg veins have been inconclusive.2 Nevertheless, the evidence suggests that great saphenous vein (GSV) pulsation does occur3 but its prevalence, etiology, and associations remain unclear. This research reintroduces pulsation in the GSV.

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In 2009, Hingorani et al recorded the venous flow patterns in deep and perforating veins in patients (44 legs) undergoing radiofrequency stylet ablation for incompetent perforating veins.4 The authors concluded that pulsation was a significant predictor of failure of the procedure.

Pulsation is defined as a cyclical change in velocity that can be regular or irregular. Pulsatile flow occurs when there is a predominant component in one direction. Palpability, or the detection of a pulse by touch, is not necessary to determine the presence of pulsation. The hypothesis was that impalpable, pulsatile antegrade flow is a common duplex finding in the GSV in patients with VVs. The aim of this study was to determine the prevalence and clinical impact of spontaneous saphenous pulsation (SP) in patients and control subjects by investigating its association with GSV reflux, GSV diameter, and clinical severity.

METHODS

Study design. This was a prospective observational study at a single public hospital to investigate SP. Twenty-three healthy ambulatory volunteers (46 legs) and 27 patients (32 legs) attending the VV clinic were recruited over
3 months by four physicians. These were consecutive subjects meeting the inclusion criteria. The study group was precisely defined to reduce the number of confounding variables. Inclusion criteria included the GSV or a straight continuation of it as the only refluxing vein in the thigh, if reflux was present. Exclusion criteria were known deep vein thrombosis, evidence of a past deep vein thrombosis, deep venous reflux, or concurrent reflux from other sources on duplex. These patients were excluded to confine this study to patients with exclusive superficial venous insufficiency (SVI) rather than study a heterogeneous group of patients with differing pathologies and sites of reflux. Reflux >0.5 seconds following a manual calf compression and release maneuver was considered a significant cut-off point. Patients were stratified into refluxers and non-refluxers on the basis of this test. Spontaneous reflux duration at rest was recorded, if present. Measurements were also taken of the GSV diameter in the midthigh since this is a known marker of clinical severity. Assessments included the C part of CEAP and the Venous Clinical Severity Score (VCSS).

This was a study to establish whether saphenous pulsation was a common observation in patients with VVs, which formed the basis of our hypothesis. Since the prevalence of an SP is not known, there were no data in the literature from which a power calculation could be based. The contralateral "normal" leg was not used as a control because SVI is often bilateral with many patients having significant reflux in the absence of symptoms.

Recording the saphenous pulse with duplex. This was performed by an experienced vascular ultrasonographer (M.A.) with volunteers and patients in the standing position. Subjects were assessed with their hips resting against an adjustable couch with most of their body weight on the contralateral leg. The foot of the test leg was touching the ground and positioned in a forward direction to minimize weight bearing. This was to ensure that the venous reservoir was full and able to transmit the arterial impulse because hydraulic conductivity is diminished in partially filled collapsible tubes. Previous work using the VFT90 (time to fill 90% of the venous volume) of air-plethysmography (APG; ACI Medical LLC, San Marcos, Calif) has shown that it can take over a minute to "fill" the venous reservoir in a leg with SVI and over 2 minutes for healthy controls.

The GSV was insonated transversely at midthigh using a linear 7 MHz transducer attached to an ultrasound machine (Philips iU22; Philips, Bothell, Wash). The midthigh was chosen because it was remote from the pulsating femoral arteries. The presence of an SP was recorded and the rate was counted from video records. Color duplex, not the waveform analysis, was used to count the saphenous pulse rate. A cycle was defined from the start of antegrade flow until the start of the next episode of antegrade flow, irrespective of whether there was intervening cessation of flow, reflux, or both.

Statistical analysis. Data were collected onto spreadsheets throughout the duration of the study and transferred into the IBM SPSS statistics package v. 19 (IBM Corporation, Armonk, NY) at completion. Results are given as median (range) unless otherwise indicated. A Mann-Whitney U test was used to compare differences between groups. The χ² test was used to compare frequencies between groups unless the expected frequency was less than 5 when the Fisher exact test was used for correction. The z test was used to compare column proportions (>2) using the Bonferroni correction. The Spearman ρ test was used to correlate individual column proportions against ordinal data from the C of CEAP. A P value of <.05 was considered significant.

RESULTS

Characteristics of volunteers and patients. The characteristics of the study group are illustrated in Table I where subjects were stratified based on the presence or absence of GSV reflux >0.5 second. Unsurprisingly, reflux was rare in C0-1 and the absence of reflux was rare in the remaining C classes, confirming its usefulness as a cut-off point of disease. Spontaneous GSV reflux at rest did not occur in any of the volunteers. Respiratory movements occasionally induced reflux in the study group between 100 and 200 ms in duration as seen in Fig 1.

Pulse description and waveform. As shown in Fig 1, the SP when present was discrete, monophasic, of variable amplitude, antegrade, and irregular. The duration of antegrade flow ranged from a flicker (10-20 ms) to an impulse lasting 500 ms. Occasionally two impulses would follow in close succession with a flicker of stasis discriminating the end of the first pulse from the beginning of the second. The blue antegrade impulse usually filled the entire cross-sectional area of the GSV but when the pulse was very brief, it was not unusual for this to be detected in only its central portion. Respiratory phasicity was observed as occasional episodic reflux which “interfered” with the recordings. In one patient, the SP was biphasic: antegrade flow followed immediately by reflux.

Table I. Characteristics of the legs in volunteers (n = 23) and patients (n = 27) stratified by the presence or absence of GSV reflux (>0.5 seconds or <0.5 seconds)

<table>
<thead>
<tr>
<th></th>
<th>GSV reflux</th>
<th>No GSV reflux</th>
<th>P valuea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of legs</td>
<td>32</td>
<td>46</td>
<td>—</td>
</tr>
<tr>
<td>Age (years)</td>
<td>46 (30-80)</td>
<td>46 (22-69)</td>
<td>.784</td>
</tr>
<tr>
<td>Female (%)</td>
<td>62.5</td>
<td>71.7</td>
<td>.389b</td>
</tr>
<tr>
<td>C0-1</td>
<td>3</td>
<td>41</td>
<td>—</td>
</tr>
<tr>
<td>C2-3</td>
<td>14</td>
<td>3</td>
<td>—</td>
</tr>
<tr>
<td>C4-6</td>
<td>15</td>
<td>2</td>
<td>—</td>
</tr>
<tr>
<td>GSV diameter (mm)</td>
<td>6.9 (2.7-9.4)</td>
<td>3.5 (1.5-7.2)</td>
<td>&lt;.0005</td>
</tr>
<tr>
<td>Detectable SP</td>
<td>24/32 (75%)</td>
<td>3/46 (6.5%)</td>
<td>&lt;.05c</td>
</tr>
<tr>
<td>SP rate/min</td>
<td>52 (22-95)</td>
<td>18 (16-75)</td>
<td>.231</td>
</tr>
</tbody>
</table>

GSV, Great saphenous vein; SP, saphenous pulse.
Results are given as median (range) unless otherwise indicated.

aMann-Whitney U test.
bχ² test.
cFisher exact test.
The median SP rate in refluxing GSVs was 52 (range, 22-95) beats per min. There was no significant difference between these rates compared with the only three subjects that had no reflux yet demonstrated an SP. Therefore, it appears that reflux is not an essential prerequisite for pulsation to occur.

A Video of the SP is provided in the electronic version of this article on the Journal’s Web site.

**Pulsation and GSV diameter.** The GSV diameter was significantly larger in legs with GSV reflux than those without reflux (Table I). The median GSV diameter was significantly elevated in the presence of an SP (no pulse: 3.5 [range, 1.5-8.1] mm; pulse: 7 [range, 4-9.4] mm; \( P < .0005 \), Mann-Whitney U test). When the subgroup of 32 legs with GSV reflux was examined, an SP was present in 24/32 (75%), 23/24 of which had a GSV diameter \( \geq 5 \) mm. Of the non-SP legs with GSV reflux, 7/8 had a GSV diameter \( \leq 6 \) mm. The presence of an SP, therefore, appears to be related to increases in GSV diameter.

**Pulsation, reflux, and clinical severity.** The relationship between clinical severity vs the proportion of legs with GSV reflux and an SP is illustrated in Fig 2. There was no statistical difference in the proportion of legs with GSV reflux in the early \( (C_{2.3}) \) and late \( (C_{4.6}) \) stages of disease \( (P = 1.0) \) as shown in Fig 2, A. However, the proportion of legs with an SP was significantly higher in late disease, occurring in 94.1% of legs with \( C_{4.6} \) but in only 52.9% of legs with \( C_{2.3} \) \( (P = .017) \) as shown in Fig 2, B.

The relationship between reflux and the presence of an SP is further illustrated in Fig 3 when both are directly compared. Reflux occurred in 8/32 (25%) legs without an SP and these were all in the earlier stages of clinical severity \( (C_0 = 2, C_1 = 1, C_2 = 3, C_3 = 2) \). This indicates that reflux is unlikely to be associated with an SP at stages \( C_{0.3} \). It would appear, therefore, that reflux may be of greater value in defining the presence or absence of venous disease and an SP may be of greater value as a marker of severe disease.

**DISCUSSION**

There are several early reports of pulsatile blood flow within VVs, which were used historically to support the concept of AV connections in their etiology. Pratt introduced the term “arterial varices,” reported a prevalence of 24% in 272 patients with advanced VVs and speculated their predisposition to recurrence. Gius supported the hypothesis of AV anastomoses in VVs, with an observational study using the operating microscope. When the AV connections were divided, pulsating spurs were observed. This concept was expanded by Haimovici using serial arteriography and Doppler ultrasonography. He concluded that AV shunting was present in 80% of VVs and at an advanced stage, the saphenous trunks may also become affected. Studies measuring oxygen partial pressure and venous oxygen content in legs with VVs demonstrated significant increases consistent with functioning AV shunts.

More recently, a detailed investigation in 39 patients with VVs and 10 control subjects revealed opposing evidence. They used direct VV cannulation to measure the venous oxygen content and pressure and compared this with the GSV cannulation in the controls. The mean venous \( pO_2 \) in varicosities was 4.5 kPa (SD, 1.0) in the supine position reducing to 3.9 kPa (SD, 0.9) on standing, which were not significantly different to samples from controls. Furthermore, pulsatile pressure tracings were not observed. The authors concluded that AV shunting is unlikely to be a causative factor in primary VVs. Radiolabeled studies on 19 legs with VVs and 26 control legs using albumen microparticles of various sizes (5-50 \( \mu \)m in diameter) injected into the femoral artery failed to confirm the increased caliber AV communications.

Pulsation in the GSV and varicose tributaries is rarely reported. When present this is usually attributed to tricuspid regurgitation as evidenced by several case reports. Table II provides a brief overview of the classification of SP.

To our knowledge, this is the first study to examine the relationship between SP against clinical severity, GSV diameter, and reflux. This study has shown that an SP occurs in 94.1% of patients in \( C_{4.6} \) stage of clinical severity. There was also a significant stepwise increase in the proportion of legs with an SP \( (P < .05, z \text{ test}) \) with increasing clinical severity, and a significant correlation between the percentage of legs with an SP for each stage of C in CEAP \( (n = 8, r = .976, P < .0005, \text{Spearman}) \). Furthermore, larger diameter refluxing GSVs \( (>6 \text{ mm}) \) were more likely to have an SP than small diameter \( (<6 \text{ mm}) \) refluxing GSVs \( (P = .003, \text{Mann-Whitney} U \text{test}) \). However, an SP was detected in a 4-mm-diameter vein but not in an 8-mm vein. The ease at which an SP could be detected therefore did not seem to be related to diameter. Although our study suggests that the presence of an SP parallels increasing GSV diameter, this does not confirm a causal relationship.

When the SP was compared with GSV reflux \( (>0.5 \text{ seconds}) \), only 75% of legs with reflux had an SP but
88.9% of legs with an SP had reflux. In contrast to SP, there was no significant difference in the presence of reflux between the early and late stages of clinical severity ($P = .017$ vs $P = 1.0$, respectively, Fisher exact). Furthermore, 25% of subjects with reflux did not have an SP, all of which were in stage C2-3. These results suggest that reflux may be the marker of the presence of SVI but SP may be a better marker of clinical severity. No attempts were made to quantify the SP or reflux. The presence or absence alone was assessed. The SP was assessed at rest but reflux assessments followed an unphysiological manual calf compression and release maneuver. Reflux values quantified in this way have minimal, if any, discriminatory usefulness in relation to clinical severity.

This study has also shown that patients with mild clinical disease (C2-3) can be stratified into two roughly equal groups using the presence or absence of an SP. However, comparisons between clinical evaluation and hemodynamic parameters are usually poor. The C part of CEAP, for example, has a poor interobserver correlation, and the assumption that the disease progresses orderly from C1 to C6 is not always the case. An easily performed hemodynamic assessment of severity could, therefore, complement clinical evaluations. Further work is needed to validate the above statements and confirm the extent at which the changes at the tissue level are represented by the SP.

### Table II. Classification of SP and the modes by which SP can usually be detected

<table>
<thead>
<tr>
<th></th>
<th>Visible</th>
<th>Palpable</th>
<th>Duplex</th>
</tr>
</thead>
<tbody>
<tr>
<td>AV malformations</td>
<td>No</td>
<td>Yes/No</td>
<td>Yes</td>
</tr>
<tr>
<td>AV fistula</td>
<td>No</td>
<td>Yes/No</td>
<td>Yes</td>
</tr>
<tr>
<td>Iatrogenic (catheterization)</td>
<td>No</td>
<td>Yes/No</td>
<td>Yes</td>
</tr>
<tr>
<td>Traumatic</td>
<td>No</td>
<td>Yes/No</td>
<td>Yes</td>
</tr>
<tr>
<td>IV drug abuse</td>
<td>No</td>
<td>Yes/No</td>
<td>Yes</td>
</tr>
<tr>
<td>Tricuspid valve</td>
<td>No</td>
<td>Yes/No</td>
<td>Yes</td>
</tr>
<tr>
<td>Regurgitation</td>
<td>Yes</td>
<td>Yes/No</td>
<td>Yes</td>
</tr>
<tr>
<td>Right heart failure</td>
<td>Yes</td>
<td>Yes/No</td>
<td>Yes</td>
</tr>
<tr>
<td>VVs</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>

AV, Arteriovenous; IV, intravenous; SP, saphenous pulse; VVs, varicose veins. Table II. Classification of SP and the modes by which SP can usually be detected. The SP associated with VVs can only be detected using duplex.

aPartial leg elevation is often required for its detection (like head elevation and the detection of the jugular venous pulse).
The explanation of hydraulic conductivity can be likened to a U-tube manometer with two equal columns of blood, arterial and venous, connected at the U-bend by the microcirculation, as shown in Fig 4. In situations where the resistance of the microcirculation is reduced, arterial pulses could then be transmitted to the venous side. This has been termed “microcirculatory failure” for the purposes of this discussion. These situations include inflammation and the later stages of CVI (C4-C6). Increased flow patterns have been demonstrated in these cases with experiments using venous duplex scanning and laser Doppler. Measurements of SP correlated against changes to the resistance of the microcirculation are likely to provide more information on the relationship between the SP and the microcirculation. Experiments utilizing the venoarteriolar response and the Bayliss effect are future research projects that may unravel the degree SP is related to failure of microcirculatory resistance.

A tricuspid valve (TV) etiology for the SP, although very unlikely, was eliminated as a confounding variable for five reasons. First, TV incompetence would promote pulsatile reflux and not antegrade flow. Second, the expectation from TV transmission would be a regular pulsation like the cardiac cycle, not irregular. Third, it is very unlikely that all the patients with an SP had TV insufficiency. Fourth, SP can occur in the absence of GSV reflux. Last, in reports of pulsation in incompetent perforating veins and VVs, GSV pulsation in the thigh was rare, suggesting an etiology remote from the TV.

The typical SP differed from the cardiac cycle in that it was irregular, with unequal amplitude. The explanation of this discrepancy may relate to an unproven assumption that the SP wave is the summation of a cardiac impulse arriving at different times depending on differing levels of resistance at different locations within the leg. Our hypothesis is that as the resistance fails the pulse becomes more prominent, regular, and discrete. However, several other mechanisms may also contribute to the wide variation in duration of antegrade flow from 10 to 20 ms to over 500 ms. These include the usual forces hampering and augmenting antegrade flow like changes in intrathoracic pressure from respiration and muscular adjustments to maintain posture.

The mechanisms of venous return from the leg have been extensively documented and include the calf-muscle pump, the negative intrathoracic pressure during the respiratory cycle, and the unidirectional blood flow from venous valves. When these mechanisms fail, then arterial drive-through from hydraulic conduction may start to play a significant role as the final step in forcing antegrade venous flow. This can be confirmed experimentally using color duplex in a patient with isolated GSV incompetence. When the patient stands up from a period of leg elevation, the first observation from the mid GSV is reflux (red) for about 10 to 60 seconds. This is followed by a few seconds of equilibration with no detectable flow (black). Then, the SP appears with irregular impulses of antegrade flow (blue) and occasional reflux (red) on a background of no flow (black). Tip-toe movements can temporarily arrest the SP until the venous reservoir refills. The SP may mark the point when all other antigravitational mechanisms of the leg cease to function. The mechanism of antegrade flow from the SP is unlikely to occur in normal individuals because the GSV behaves like a collapsed tube with minimal resting flow, the vast majority of the venous return being delivered through the popliteal-femoral venous axis.

Limitations of study. The number of volunteers and patients in this study is small. While there are sufficient numbers to identify the presence of an SP, it is recommended that this work is considered as a pilot study. Any conclusions regarding the extent of the relationship as a marker of severe SVI, although statistically significant, should be confirmed by independent vascular units. The ease of detection should make this possible for many phlebology centers that use duplex as a routine in their investigation of venous disease. Currently, measurement of SP is more of academic interest than of clinical severity because an SP by itself is not a sufficiently discriminating marker to determine whether or what course of treatment to pursue.

None of the subjects had echocardiography to establish the competence of the TV. Simultaneous recordings of the
SP and heart rate were not performed and the SP was not assessed in relation to symptoms.

CONCLUSIONS

The presence of a duplex detectable SP is a common observation in patients with chronic SVI. It is easy to record and could complement assessments of reflux. The SP appears to parallel clinical severity and is related to increases of the GSV diameter. Its etiology remains unknown. Hydraulic transmission from the arterial circulation may be a plausible hypothesis in pathologic states of microcirculatory failure (diminished capillary resistance). Future work will be required to define the relationship between microcirculatory failure and the appearance of an SP.

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AUTHOR CONTRIBUTIONS

Conception and design: CL
Analysis and interpretation: CL, EK, GG
Data collection: CL, MA, EK, GM
Writing the article: CL, EK
Critical revision of the article: MA, EK, GM, GG
Final approval of the article: CL, MA, EK, GM, GG
Statistical analysis: CL
Obtained funding: CL, GG
Overall responsibility: GG

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