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Ambulatory Venous Hypertension: Component Analysis in 373 Limbs

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ABSTRACT

Purpose: We studied factors contributing to ambulatory venous hypertension in patients with symptoms of chronic venous insufficiency in order to delineate the contribution of each to global venous hypertension.

Methods: A total of 373 consecutive limbs with ambulatory venous hypertension were studied. Simultaneous ambulatory venous pressure and air plethysmography measurements allowed compliance calculations. With reactive hyperemia, maximal arterial inflow was measured. Air plethysmography provided calf venous pump capacitance and ejection fraction data. Reflux was quantified by a point system based on Duplex, venous filling index on airplethysmography (VFI-90), Valsalva foot venous pressure, and ambulatory venous pressure recovery time (VFT). Multiple regression analysis was used to model ambulatory venous pressure in terms of these variables.

Results: Six major causes of ambulatory venous hypertension were identified and quantified: 1. reflux, 2. increased arterial inflow, 3. reduced venous capacitance, 4. poor ejection fraction, 5. poor compliance of the calf venous pump, and 6. a combination of factors. Of the total, 91% of the limbs had at least two of these factors, 57% had three factors, and 24% had four factors contributing to ambulatory venous hypertension. Reflux was present in 97% of limbs but was significant (VFT <15 seconds) in only 57%, suggesting that other factors were dominant in the remainder. *(continued on next page)*

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(Abstract continued)

Conclusion: Ambulatory venous hypertension is a multifactorial entity. Many of the contributing factors are interrelated in a complex fashion. Regression analysis indicates that even though reflux followed by arterial inflow and capacitance were important as single contributing factors, a combination of factors is more important than any single individual factor in the genesis of ambulatory venous hypertension. VFT was found to be superior to other measurements in quantifying global reflux.

Introduction

Ambulatory venous pressure measurement is a global index of venous function in the lower limb. In a pioneering report, Pollack and Wood[1] noted that ambulatory venous pressure is dependent upon venous reflux, arterial inflow, and calf venous pump volume. Compliance and ejection fraction of the calf venous pump[2] should be added to this list. Valvular reflux as related to ambulatory venous hypertension has received such particular emphasis in the literature, however, that other relevant causative factors tend to be ignored. To our knowledge there has been no previous publication dealing with the prevalence of the different components causative of ambulatory venous hypertension in a given patient population. Such prevalence data for our practice are presented in this report.

Ambulatory venous pressure is traditionally reported in terms of two parameters: the pressure reduction with exercise (percent drop) and the recovery time (VFT). Though interrelated, they are best considered independent variables owing to the non-linearity of the pressure volume relationship of the calf venous pump and the variability of the shape of the non-linear curve itself; VFT is dependent on the latter. In this report we also provide a differential analysis of these two parameters as related to venous stasis ulceration, which may provide clues as to which of the two is more important: the absolute low pressure level reached with calf exercise or the *duration* of the low pressure environment in the genesis of symptoms.

Methods

A total of 411 patients with suspected chronic venous insufficiency were tested in the venous laboratory. From this pool, 373 consecutive limbs with ambulatory venous hypertension in 238 patients (47% male, 53% female) were identified over a period of 22 months. The distribution of these limbs according to CEAP classification[3] is shown in Table I.

Ambulatory venous hypertension was defined as a percentage drop of less than 50% with calf exercise and/or venous filling time (VFT) of less

Table I

Distribution of Case Material According to CEAP Classification

Clinical Severity	Etiology	Anatomy	Pathology
Class 1 15%	Primary 30%	Superficial System 9%	Reflux 7%
Class 2 11%	Secondary 70%	Deep System 17%	Obstruction 20%
Class 3 29%		Combination 74%	Reflux & Obstruction 73%
Class 4 14%			
Class 5 15%			
Class 6 16%			

than 20 seconds. *Normal* values for ambulatory venous pressure and other test parameters described below were obtained in 45 healthy volunteers.

Test Protocol

A complete history was obtained and a relevant physical examination was performed. Clinical severity of chronic venous insufficiency was classified according to current reporting standards.[4]

Ambulatory venous pressure with calf exercise[5,6] was monitored through a needle in the dorsal foot vein and recorded on a polygraph. Postexercise pressure was the lowest point on the pressure curve. Percentage drop was calculated from this value and resting pressure. Venous filling time (VFT) was noted.

The major contributors to ambulatory venous hypertension, namely arterial inflow, compliance of the calf venous pump, ejection fraction, venous capacitance and reflux, were determined by the following tests: Simultaneous with venous pressure measurement, calf volume changes during outflow following cuff release were monitored with an air plethysmograph (ACI Medical) so as to determine compliance.[5] Compliance data were plotted against a nomogram developed from normal volunteers and categorized as normal, borderline, or abnormal (Figure 1). In addition, traditional parameters of the calf venous pump function,[7] such as venous volume (VV), ejection fraction (EF), resting volume fraction (RVF), and venous filling indexes (VFI-90), were also obtained. These measurements provided data on two other listed components of ambulatory venous hypertension, namely capacitance and ejection fraction. Valsalva foot venous pressure[8] ("Valsalva venous pressure") and arm/foot venous pressure differential[9] with reactive hyperemia in the supine

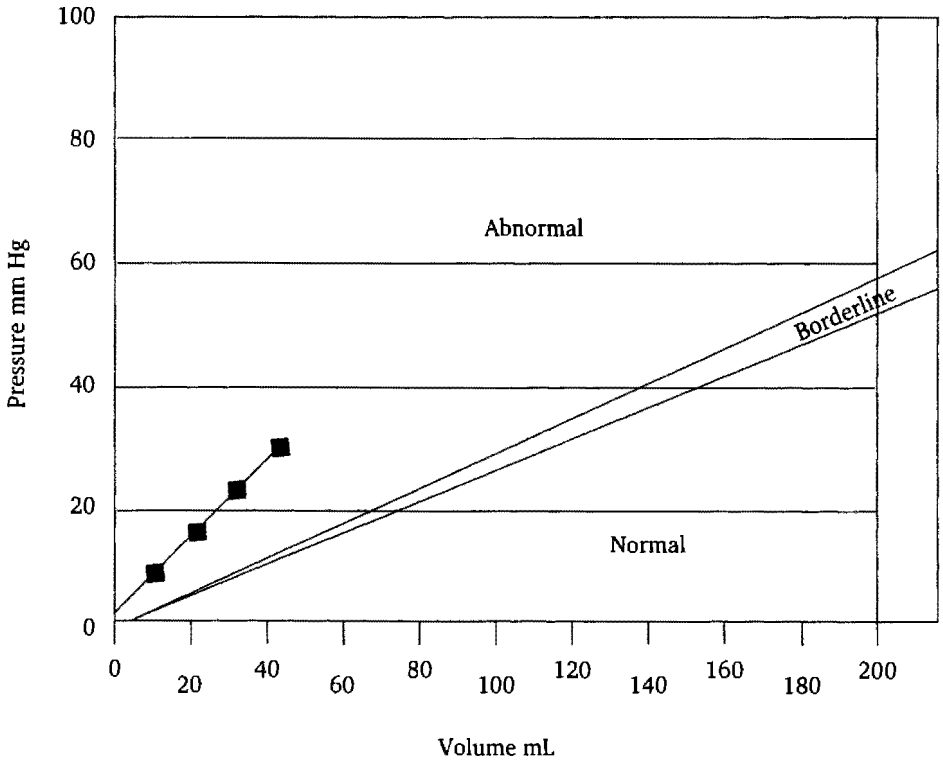

Figure 1. A calf venous compliance curve plotted from simultaneous pressure and plethysmographic recordings. In this instance, the compliance was abnormal compared with a nomogram derived from normal limbs.

position were also measured during the same test session. Reactive hyperemia was induced with a 3-minute ischemic thigh cuff occlusion at >250 mm Hg. Ischemic occlusion was monitored by a photoplethysmographic probe (PPG) applied to the toe. When the thigh cuff was released, pressure was not taken down to zero but was maintained at a venous occlusion setting of 60 mm Hg, allowing the air plethysmograph in place around the calf to record the maximal arterial inflow, another listed component of ambulatory venous pressure. When the inflow curve reached a plateau, the thigh cuff was completely deflated to record the post-reactive hyperemia foot venous pressure.

For determination of reflux, a major component of ambulatory venous pressure, color Duplex examination in supine and erect positions was performed with compression maneuvers. Compression maneuvers were carried out with standardized quick inflation-deflation cuffs.[11] Extent of reflux present in a limb was characterized by a point scoring system[11] as previously described; one point was assigned to each venous segment that was refluxive (saphenous, superficial femoral, deep femoral and popliteal, respectively) and a total score ("Duplex score") obtained. Limbs with clinical severity Class 4 and higher, exhibiting stasis skin changes, presented with a mean Duplex score of 2.8 ±1.3 SD in the previous study. This method of grading the extent and severity of reflux appears to be more reliable than a grading system based on the distal extent of reflux.[11] Several practical measures, such as local spray anesthesia for venipunctures, simultaneous performance of different tests that required the same maneuvers (e.g., toe stands, reactive hyperemia) and adequate rest periods between tests aided clinical implementation of the test protocol with a high degree of patient acceptance. A more detailed methodology of the techniques described above can be found elsewhere.[4-9]

Statistical Analysis

Multiple regression analysis was used to model ambulatory venous pressure in terms of reflux, ejection fraction, arterial inflow, capacitance and compliance. The Duplex score, VFT, VFI-90 and Valsalva venous pressure were considered as measures of reflux. The contribution of individual variables was assessed by regression analysis. A standard correlation analysis of the variables using Pearson Correlation Coefficients was also carried out.

Results

Normal Limbs. Normal (mean ±SD) values for the various test parameters in 85 limbs among 45 healthy volunteers were as follows: (1) Post-exercise pressure 37.6 ±11.7 mm Hg, ambulatory venous pressure percent drop 57 ±7 (n=67), and VFT (sec) 43 ±22 (n=37); (2) Arm/foot venous pressure differential and reactive hyperemia test: Grade 0 for all limbs; (3) Air plethysmography: VV (mL) 120 ±28 (n=28), EF (%) 61 ±11 (n=28), RVF (%) 19 ±11 (n=28) and maximal arterial inflow (data from nonvolunteer *normal* limbs) (mL/sec) 3.8 ±2.2 (n=13). Mean variation from the opposite limb for all listed values was <20%, with the exception of RVF for which the mean variation was 35% ±20.

Limbs with Ambulatory Hypertension. Ambulatory venous pressure parameters (mean ±SD) in this group of limbs was as follows: postexercise pressure 54 ±17 mm Hg, percent drop 44 ±16%, and VFT 17 ±17 seconds. Twenty percent of patients had significant obstruction (Grades III and IV) as determined by the arm/foot venous pressure differential technique.

The relationship between varying grades of outflow obstruction and ambulatory venous hypertension has been reported in detail elsewhere.[6] Outflow obstruction causes ambulatory venous hypertension by a change in one or more of the following parameters: compliance, capacitance, ejection fraction and residual volume fraction. These components are individually covered in the current analysis and include limbs with outflow obstruction.

Although 50% for percent drop and 20 seconds for VFT were used as criteria to differentiate normal from abnormal, a wide range of values for these parameters was in fact found in this group of patients (Tables I and II). The incidence of Class 4 and higher clinical severity is also shown for each category of values presented (Table III).

The incidence of clinical Class 4 or higher was 49% in limbs with percent drop of >50% in ambulatory venous pressure. This represents 30 limbs among 290 (Table III) for an overall incidence of 10%, confirming our previous finding[8] that some patients with venous stasis ulceration have *normal* postexercise pressure.

Table II

Categorization of Ambulatory Venous Hypertension in 373 Limbs

Category Prevalence Clinical	% Drop Abnormal VFT Normal n=97 (26%)	% Drop Normal VFT Abnormal n=92 (25%)	% Drop Abnormal VFT Abnormal n=184 (49%)
Class 0 to 2	42% n=41	21% n=19	20% n=37
Class 3	41% n=40	29% n=27	23% n=43
Class ≥ 4	17%* n=16	50% n=46	57% n=104

*Significantly less ($p<.001$) compared with Class ≥ 4 incidence in the other two groups.

Table III

Ambulatory Venous Hypertension Analyzed According to Different Ranges for VFT and % Drop (n=290). Corresponding Incidence of Clinical Class 4 or Higher Is Also Shown.

Severity of Ambulatory Venous Hypertension	VFT Range Seconds	Prevalence (%)	Clinical Class ≥4	(%) Drop Range	Prevalence (%)	Clinical Class ≥4
Severe	1–10	36% n=103	63% n=65	0 to 10%	0% n=0	0% n=0
Moderate	10–15	21% n=61	48% n=29	10 to 30%	18% n=52	56% n=29
Mild	15–20	15% n=44	48% n=21	30 to 50%	61% n=177	0% n=0
Normal	≥ 20 sec	28% n=82	17% n=14	≥ 50%	21% n=61	49% n=30

Six major causes of ambulatory venous hypertension were identified in the test population: (1) reflux, (2) poor compliance of the calf venous pump, (3) reduced calf venous capacitance (<20% vs. opposite limb and/or <60 mL), (4) poor ejection fraction (<40%), or higher residual volume fraction (>50%), (5) increased arterial inflow (>20% opposite limb), and (6) a combination of factors. These components are described in greater detail below.

Reflux involving one or more venous segments was identified by color Duplex examination in 97% of the limbs. Such reflux was confined to the superficial system alone in 9%, deep system alone in 17%, and a combination in 74%. Current methodology with Duplex examination does not allow detection of reflux through unnamed non-axial veins. The presence of qualitative reflux through one or more venous segments on Duplex does not signify that quantitatively significant reflux producing ambulatory venous hypertension is present. We utilized several other methods (see below) in an attempt to quantify the severity of reflux. The distribution of patients in this study according to the Duplex score method[11] of grading severity of reflux is shown in Table IV. Based on this method, 46% of patients in the test population had severe reflux (reflux score = >3). VFT, VFI-90 and Valsalva foot venous pressure[8] also measure reflux. A

moderate or severe reduction in VFT (<15 seconds) was present in 57% of the limbs (Table III). VFI-90 (which measures passive reflux without valve closure) was abnormal (>2.0 mL/sec) in 52% and Valsalva foot venous pressure was abnormal (>4 mm Hg) in 29% of patients in this study. Though reflux was universally present in these patients by Duplex, quantifiable reflux was identified by these global methods (i.e., Duplex scoring method, VFI-90 and Valsalva foot venous pressure) in only about one-half of the patients. Ambulatory venous hypertension could be present even when only one or two venous segments were refluxive (Table IV). Factors other than reflux causative of ambulatory venous hypertension were (likely) present in these patients.

Table IV

Distribution of Reflux in Hypertensive Limbs (n=331) According to the Duplex Score System (See Text)

Duplex Reflux Score		Prevalence (%)
0	Normal	7
1	Mild	21
2	Moderate	26
3	Severe	23
4	Pan-refluxive	23

Compliance was borderline in 12%, grossly abnormal in 44%, and normal in the remaining 44%.

Venous capacitance was normal in 48%, decreased in 29%, and increased in 23%. Seventy-two percent of limbs with reduced venous capacitance (n=110) had associated obstruction.

Ejection fraction was reduced in 44% of limbs. Residual volume fraction was abnormal in 32%.

Maximal arterial inflow was increased in 32% of limbs.

Combination. Even though reflux was commonly present (97%), 91% of the limbs had at least two factors, 57% had three factors, 24% had four factors, and 2% had five factors that could affect ambulatory venous pressure independently of reflux.

Many of the above variables were found to be interdependent from an analysis of Pearson Correlation Coefficients:

1. Ejection fraction correlated inversely to compliance ($p<.005$), VFI-90 ($p<.05$), and capacitance ($p<.0001$). There was no significant correlation to arterial inflow.
2. Capacitance was correlated directly to compliance ($p<.0001$), arterial inflow ($p<.003$), and to VFI-90 ($p<.001$).
3. Arterial inflow correlated directly to capacitance ($p<.005$).
4. VFI-90 was related directly to compliance ($p<.003$) and capacitance ($p<.0001$) and inversely to ejection fraction ($p<.04$).
5. Compliance was related directly to the above three variables ($p<.003$) and inversely to ejection fraction ($p<.004$).
6. VFT was correlated directly to ejection fraction ($p<.0003$).

Though statistically derived, the above interrelationships are intuitively understandable. VFI-90 measures passive reflux/refilling into the leg when the subject assumes an erect position from recumbency and valve closure is not obtained, hence the correlation to capacitance and compliance. Correlation analysis of ambulatory venous pressure (postexercise pressure) and the four quantitative measures of reflux (VFI-90, Duplex score, Valsalva venous pressure and VFT) showed that VFI-90 did not correlate to ambulatory venous pressure whereas the other three parameters did. It is noteworthy that all of the latter three parameters involve active valve closure (by Valsalva, active or mechanical calf compression) in the measurement of reflux. Not surprisingly, all of these three measures of active reflux were found to be interrelated by significant Pearson Correlation Coefficients. Although the Duplex score, Valsalva venous pressure and VFT were all significantly correlated to ambulatory venous pressure, the best fit for the data was obtained when VFT was used to measure reflux (R^2= 15.7%, Mallow's C_p=6.9). Using a similar technique to analyze reflux and non-reflux variables that may influence ambulatory venous pressure, the following overall rank (in decreasing order of

importance to the regression) was obtained: (1) VFT (partial R^2 of 12%), (2) arterial inflow (partial R^2 of 2.9%) and (3) capacitance (partial R^2 of 2.8%). The relatively small partial R^2 of even VFT, the most important single component, suggests that a combination of factors rather than any single component per se is more important as the basis of ambulatory venous hypertension.

Discussion

Ambulatory venous pressure measurement has been widely accepted as the "gold standard" in assessing venous function of the lower limb. This arises from an intuitive belief that abnormalities of ambulatory venous pressure must be fundamentally related to the pathophysiologic manifestations of chronic venous insufficiency even though the precise relationship is not clear.[8] The process of ambulatory venous pressure measurement involves emptying of the calf venous pump by a series of calf muscle contractions. As the calf volume is reduced by emptying, pressure falls and then increases with refilling. The shape of the volume pressure curve is non-linear and is variably related to the compliance of the calf venous pump. In the absence of reflux, volume and pressure recovery are dependent on arterial inflow into the pump, normally around 2 mL per second. At this rate of refilling into the calf venous pump of normal capacitance and compliance, a VFT of 20 seconds or more is expected. With this somewhat simplified framework in mind, factors influencing ambulatory venous pressure can be explained on a logical basis. Increased arterial inflow results in faster refilling of the tank and therefore a shortened VFT. Since a lowered peripheral resistance is associated with increased arterial inflow, higher than normal post-capillary pressures may be present contributing to the increased postexercise pressure. Abnormal compliance will influence both the postexercise pressure and the VFT.[10] Reduced ejection fraction, higher residual volume, and reduced capacitance will each shorten VFT as the volume to be refilled is reduced. Many of the foregoing theoretical relationships were confirmed by the current study.

In measuring ambulatory venous pressure, two parameters, percent drop and VFT, are traditionally monitored. In the present analysis we point out that either one or both of the parameters may be abnormal. This has not been emphasized previously. Limbs with a normal percent drop but reduced VFT are still considered "hypertensive" as the *duration* of reduced pressure after calf exercise is truncated even though the absolute low point in pressure reached after calf exercise is *normal*. Such patients will likely experience a higher time-averaged mean pressure during the course of daily activity compared to patients with a normal VFT. Current analysis confirms a strong relationship between VFT and post-exercise pressure. Furthermore, VFT is closely related to reflux and may be the best among the currently available methods to measure it.

A venous filling time of >20 seconds is accepted as *normal*. In our own laboratory, however, a wide range of *normal* values for this parameter was found in 37 healthy volunteers (mean: 43 ±22 seconds; range: 20 to 89 seconds). In patients with unilateral hypertension, the opposite "*normal*" limb also had a very wide range for VFT (mean: 21 seconds; range: 1–144 seconds). In 26% of hypertensive limbs, VFT was *normal*, i.e., >20 seconds, suggesting that the dividing line between normal and pathologic with regard to this parameter may have an individual basis, rather than conforming to a universal standard. The wide range of values for both normal and pathologic in this important physiologic parameter, central to the function of calf venous pump, is surprising.

In this study both percent drop and VFT were abnormal in 49%. In the remainder only one of the two parameters (percent drop, VFT) was abnormal (Table II), with roughly equal incidence for each. Of the two, VFT appears to be relatively more important in the genesis of stasis skin changes (Table II). The strong association of VFT with reflux is probably relevant in this context. There have been previous attempts to construct an index incorporating both VFT and percent drop.[12,13]

Another difficulty with the clinical application of ambulatory venous pressure measurement is that it is an extreme measurement obtained with maximal calf exercise. Volume pressure curves in collapsible tubes such as the calf venous pump are non-linear (Figure 2).[2] The calf muscles are infrequently exercised to maximal venous pressure reduction during the course of daily activity. This fact, combined with the non-linearity of the pressure volume relationship, may cause misleading conclusions to be drawn from ambulatory venous pressure measurement (Figure 2).

Many laboratories interpret the presence of ambulatory venous hypertension as synonymous

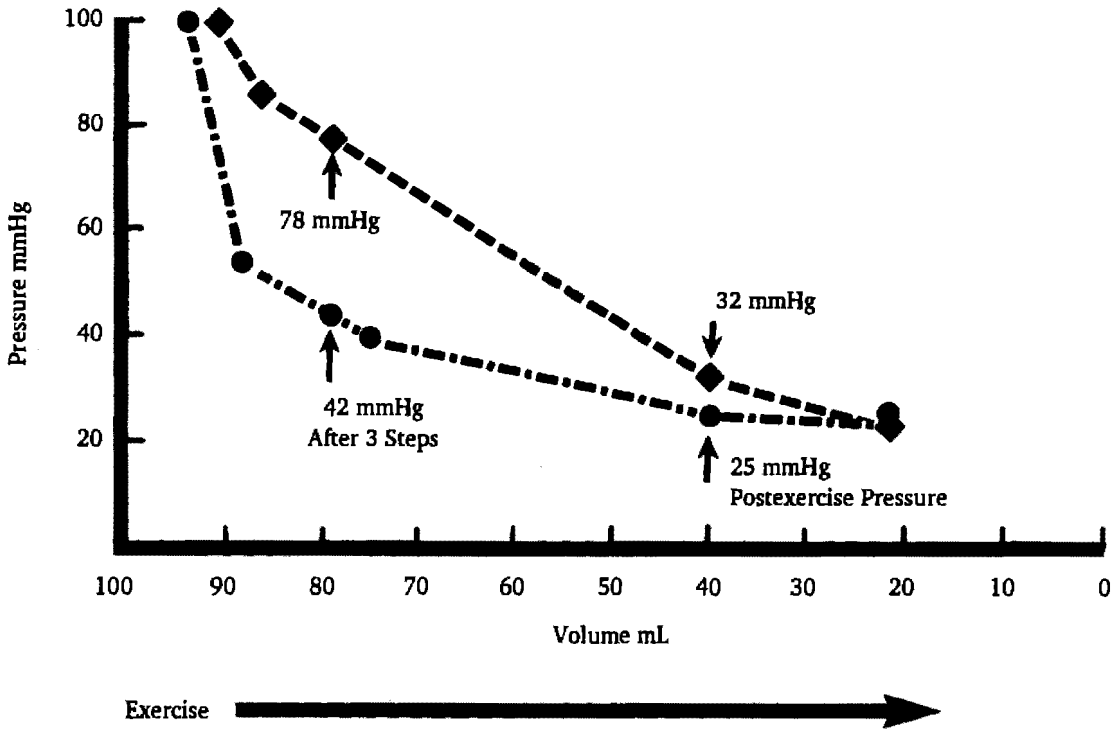

Figure 2. Compliance differences between two limbs may result in similar or even identical pressures with near maximal or maximal exercise. Yet vastly different pressures may prevail with minimal or moderate exercise, such as taking three easy steps typical of normal daily activity. Higher prevailing pressures with low or intermediate levels of calf exercise typical of normal daily activity may be more relevant to the genesis of symptoms than the maximal pressure reduction with maximal calf exercise utilized in the protocol for ambulatory venous pressure measurement.

with the presence of reflux. Reflux was indeed ubiquitous in this case material, but moderate or severe reflux per VFT was present in only about half the limbs with ambulatory hypertension. Current analysis indicates that multiple other factors are often involved. These nonreflux factors are interrelated and influence ambulatory venous pressure in a complex fashion. A corollary inference that may be drawn from this is that surgical correction of reflux alone may not result in normalization of ambulatory venous pressure when multiple causative factors contribute to the genesis of ambulatory venous hypertension. Clinical conditions producing ambulatory venous hypertension, whether postthrombotic or primary in etiology, often involve reflux and nonreflux factors. For example, postthrombotic syndrome, a common cause of ambulatory venous hypertension, can result in capacitance reduction (clot retention, fibrosis, luminal contraction), valve reflux, and poor ejection fraction due to reduced ankle movement from scarring[14] or hypertension-induced neuromyopathy.[15] Poor compliance due to postthrombotic-calf venous pump wall changes is frequently present as well.[5,6] Schalin[16] has demonstrated increased arterialization of chronic varicosities, a clinical condition causative of ambulatory venous hypertension due to primary etiology. Increased arterial inflow is probably present in many of these cases.[17] The importance of considering arterial inflow in venous testing has been emphasized by Pierce and colleagues.[18] The presence of multifactorial ambulatory hypertension in many patients provides an explanation as

to why ambulatory venous hypertension is not always normalized by reflux corrective surgery such as valve reconstruction. In a patient with significant obstruction, ambulatory venous hypertension may persist even after successful veno-venous bypass procedures because of persistent adverse compliance or capacitance changes in the calf venous pump.

Component analysis of ambulatory venous hypertension may provide a prognostic view for reflux corrective surgery in a given patient. At present, surgical correction of only one of the several listed factors causative of ambulatory hypertension, namely reflux, is possible. Poor ejection fraction can perhaps be improved by active physiotherapy to increase ankle joint mobility[14] and strengthen calf muscles. Marked improvement and even normalization in ambulatory venous pressure can be expected if severe reflux (see under Reflux in the Results section) is the only factor identified. VFT is probably the best available global gauge of reflux even though VFT itself is influenced by other non-reflux factors (capacitance, ejection fraction) that are interrelated in a complex fashion and often occur in association with reflux in many clinical conditions. It is not known if correction of reflux would eventually result in correction of associated factors. When other significant non-correctable factors are found and reflux is not severe per VFT, the outlook for relief of ambulatory venous hypertension remains guarded even if associated reflux is corrected. For example, maximal arterial inflow of 21 mL/second (normal: 5 to 6 mL/second) was encountered in a patient with Klippel-Trenaunay syndrome with associated venous reflux. High arterial inflow was likely due to A-V malformation. Correction of reflux pathology in this patient produced only a minor improvement in ambulatory venous hypertension.

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References

1. Pollack AA, Wood EH: Venous pressures in the saphenous vein at the ankle in man during exercise and changes in posture. Am J Physiol 1:649-662, 1949.
2. Raju S, Fredericks R, Lishman P, et al: Observations on the calf venous pump mechanism: Determinants of postexercise pressure. Phlebology 5:21-23, 1993.
3. Beebe HG, Bergan JJ, Bergqvist D, et al: Classification and grading of chronic venous disease in the lower limbs: A consensus statement. Phlebology 10:42-45, 1995.
4. Porter JM, Moneta GL, Beebe HG, et al: Reporting standards in venous disease: An update. J Vasc Surg 21:635-645, 1995.
5. Neglén PN, Raju S: Compliance of the normal and postthrombotic calf. J Cardiovasc Surg 36:225-231, 1995.
6. Raju S, Fredericks R: Venous obstruction: An analysis of 137 cases with hemodynamic, venographic, and clinical correlations. J Vasc Surg 14:305-313, 1991.
7. Christopoulos D, Nicolaides AN, Szendro G, et al: Air plethysmography and the effect of elastic compression on venous hemodynamics of the leg. J Vasc Surg 5:148-159, 1987.
8. Raju S, Fredericks R: Hemodynamic basis of stasis ulceration—a hypothesis. J Vasc Surg 13:491-495, 1991.
9. Neglén P, Raju S: Detection of outflow obstruction in chronic venous insufficiency. J Vasc Surg 17:583-589, 1993.
10. Raju S, Greene AB, Fredericks RK, et al: Tube collapse and valve closure in foot venous pressure regulation: Studies with a mechanical model. J Endovasc Surg 5:42-51, 1998.
11. Neglén P, Raju S: A comparison between descending phlebography and Duplex Doppler investigation in the evaluation of reflux in chronic venous insufficiency: A challenge to phlebography as the "gold standard." J Vasc Surg 16:687-693, 1992.
12. Pierce EC II, Premus G, Martinelli G, et al: Foot blood flow measurements improve venous plethysmographic studies. Surgery 101:422-429, 1987.
13. Kriessmann A: Ambulatory venous pressure measurements. In: Nicolaides AN, Yao JST (eds): Investigation of Vascular Disorders. New York: Churchill Livingstone, 1981, pp 461-477.
14. Back TL, Padberg FT Jr, Araki CT, Thompson PN, et al: Limited range of motion is a significant factor in venous ulceration. J Vasc Surg 22:519-523, 1995.
15. Taheri SA, Heffner R, Pendergast D, et al: Myopathy and venous insufficiency. Phlebology 2:7-12, 1987.
16. Schalin L: Role of arteriovenous shunting in the development of varicose veins. In: Eklöf B, Gjöres JE,

Thulesius O, Bergqvist D (eds): Controversies in the Management of Venous Disorders. London: Butterworth & Co, 1989, pp 182-194.

17. Haimovici H: Clinical review: Role of precapillary arteriovenous shunting in the pathogenesis of varicose veins and its therapeutic implications. Surgery 101:515-522, 1987.

18. Pierce EC, Chiano K, Schanzer H: Volume tests for chronic venous insufficiency: An appraisal. Surgery 109:567-574, 1991.

DISCUSSION

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A taxonomy is often a first step in scientific comprehension. Classification is then followed by understanding of components contributing to what might appear, on superficial examination, to be a singular phenomenon. The final step would be quantification of each component with an aim of prediction or control. The authors studied 373 consecutive limbs with ambulatory venous hypertension. They used many tests: simultaneous ambulatory venous pressure and air plethysmography measurements for compliance calculations, reactive hyperemia to delineate arterial inflow, and measurement of calf venous pump capacity and ejection fraction. Reflux severity was examined using several techniques including Duplex scanning, air plethysmography, and Valsalva foot venous pressure. As might be expected, and has been pointed out before, sometimes these measurements were divergent. Multiple contributing causes of ambulatory venous hypertension were identified and quantified using multiple regression analysis. These were, in order of importance: reflux, increased arterial inflow, reduced venous capacitance, poor ejection fraction, poor compliance of the calf venous pump, and a combination of factors. Almost all the limbs exhibited at least two factors, and half had three factors contributing to venous hypertension.

The fact that reflux was present in most patients but severe in only about half the limbs suggests that a well-chosen study might identify patients who could benefit from reflux corrective surgery. This intervention is one of our most effective tools. However, the authors could only speculate about this possibility. The idea that ambulatory venous hypertension is a nonlinear phenomenon and related to multiple underlying factors is a sound one.[1] Their retrospective study confirms the notion that absolute levels of venous filling time or hypertension cannot be dogmatically applied to the complications of CVI. For example, they found and documented a wide range of normal values for venous filling time in healthy volunteers, and then suggest that the dividing line between normal and pathologic might have an individual basis. The variability might relate to the rate of change of rate of pressure increase with dependency as well as individual factors related to activated leukocytes.[2] Outcomes incited by differing mechanisms contributing to rate changes and magnitude of venous hypertension could vary.

The authors had hoped that their complex component analysis would provide a prognostic view for reflux corrective surgery; this hope was not realized. But what they did show was that most patients *have* reflux. Such complex investigation will probably be beyond the capabilities of most vascular laboratories. Inasmuch as venous hypertension contributes to cutaneous complications of CVI, it is important to try to understand its genesis. Clearly, examinations are needed before proposing a particular operation, but limits and clinical judgment are also needed. Would anyone really need all this testing? It will be critical to delineate the fine dividing line between occlusion versus compensated occlusion and dominant reflux in terms of interposition of competent valves. The authors' findings emphasize the potential futility of randomizing for trial any single procedure for the treatment of chronic venous insufficiency with hypertension severe enough to produce Class 4–6 changes. The cohorts demanded by the CEAP classification would be daunting.

Ultimately a simpler goal might be to interrupt the transmission of the venous hypertension to the affected areas. More than that, we might change the rate of change of rate of transmission