

Consensus Statement

Classification and Grading of Chronic Venous Disease in the Lower Limbs: A Consensus Statement

This Consensus Statement was prepared by an ad hoc committee at the American Venous Forum 6th Annual Meeting, 22-25 February 1994, Maui, Hawaii (organized by the Straub Foundation).

Hugh G. Beebe, USA
 John J. Bergan*, USA
 David Bergqvist, Sweden
 Bo Eklof*, USA
 Invar Eriksson, Sweden
 Mitchel P. Goldman, USA
 Lazar J. Greenfield, USA
 Robert W. Hobson, II, USA
 Claude Juhan, France
 Robert L. Kistner*, USA

Nicos Labropoulos, UK
 G. Mark Malouf, Australia
 James O. Menzoian, USA
 Gregory L. Moneta*, USA
 Kenneth A. Myers, Australia
 Peter Neglen, United Arab Emirates
 Andrew N. Nicolaides**, UK
 Thomas F. O'Donnell, USA
 Hugo Partsch, Austria

Michel Perrin, France
 John M. Porter, USA
 Seshadri Raju, USA
 Norman M. Rich, USA
 Graeme Richardson, Australia
 Harry Schanzer, USA
 Philip Coleridge Smith, UK
 D. Eugene Strandness, USA
 David S. Sumner, USA

*Executive Committee, **Chairman

Part I: Classification

Introduction

Chronic venous disease is an important cause of discomfort and disability and is present in a significant percentage of the population worldwide. Methods to diagnose and measure severity have evolved rapidly so that accurate classification of venous disease is now possible. Standards for reporting venous disease have been based on a clinical classification developed in 1988 by a subcommittee of the Society for Vascular Surgery (SVS) and International Society for Cardiovascular Surgery (ISCVS) [1]. This classification has contributed to the uniform presentation of diagnosis and results of treatment. However, advances in the knowledge of chronic venous disease have created a need to expand

definitions to cover many aspects, including anatomy, pathophysiology and aetiology. The aim of this document is to present a more precise classification of chronic venous dysfunction which is simple enough to encourage its universal acceptance. Acceptance of a standard classification provides a basis for uniformity in reporting and assessing different modalities of diagnosis and treatment.

The classification has been developed under the headings listed in Table 1.

Table 1. Classification

C	For clinical signs (grades ₀₋₆), supplemented by (A) for asymptomatic and (S) for symptomatic presentation
E	For etiological classification (congenital, primary, secondary)
A	For anatomical distribution (superficial, deep or perforator, alone or in combination)
P	For pathophysiological dysfunction (reflux or obstruction, alone or in combination)

Correspondence and offprint requests to: Dr R. L. Kistner, Straub Foundation, 1100 Ward Avenue, Suite 1010, Honolulu, Hawaii 96814-1617.

Clinical Classification (C₀₋₆)

The clinical classification is based on objective clinical signs of chronic venous disease (C₀₋₆) (Table 2) supplemented according to presentation for (A) asymptomatic (e.g. C_{0-6,A}) or (S) for symptomatic limbs (e.g. C_{0-6,S}). Symptoms include aching, pain, congestion, skin irritation and muscle cramps as well as other complaints attributable to venous dysfunction. This clinical classification is organized in terms of ascending severity of disease [1]. Limbs in higher categories have more severe manifestations of chronic venous disease and may have some or all of the findings associated with less severe categories.

Table 2. Clinical classification

Class 0	No visible or palpable signs of venous disease
Class 1	Telangiectases or reticular veins
Class 2	Varicose veins
Class 3	Oedema
Class 4	Skin changes ascribed to venous disease (e.g. pigmentation, venous eczema, lipodermatosclerosis)
Class 5	Skin changes as defined above with healed ulceration
Class 6	Skin changes as defined above with active ulceration

Therapy may alter the clinical signs and symptoms and the limb should be reclassified after treatment.

Telangiectases are defined as dilated intradermal venules up to a diameter of approximately 1 mm and reticular veins are defined as dilated subdermal veins up to a size of about 4 mm which are not palpable. Varicose veins are palpable, dilated subcutaneous veins usually larger than 4 mm [2]. Telangiectases and reticular veins are separated from varicose veins in this classification as it is considered that the telangiectases do not lead to venous ulceration while the reticular veins may [2]. Both may be associated with patient symptoms [3].

Etiological Classification (E_C, E_P or E_S)

This etiological classification recognizes three categories of venous dysfunction: congenital, primary and secondary (Table 3). Congenital problems may be apparent at birth or be recognized later. Primary problems are neither congenital nor do they have an identifiable cause. Secondary problems are those acquired conditions that have a known pathological cause, such as thrombosis. These categories are mutually exclusive.

Table 3. Etiological classification

Congenital (E _C)	
Primary (E _P)	- with undetermined cause
Secondary (E _S)	- with known cause
	Post-thrombotic
	Post-traumatic
	Other

Anatomical Classification (A_{S,D,P})

This classification describes the anatomical extent of venous disease, whether in the superficial (A_S), deep (A_D) or perforating (A_P) veins. Disease may involve one, two or all three systems. For those reports for which greater detail is required, the site and extent of involvement of the superficial, deep and perforating veins may be categorized using the anatomical segments listed in Table 4.

Table 4. Anatomical classification

Segment no.	
	<i>Superficial veins (A_S)</i>
1	Telangiectases/reticular veins
	Greater (long) saphenous (GSV)
2	Above knee
3	Below knee
4	Lesser (short) saphenous (LSV)
5	Non-saphenous
	<i>Deep veins (A_D)</i>
6	Inferior vena cava
	Iliac
7	Common
8	Internal
9	External
10	Pelvic - gonadal, broad ligament, other
	Femoral
11	Common
12	Deep
13	Superficial
14	Popliteal
15	Crural - anterior tibial, posterior tibial, peroneal (all paired)
16	Muscular - gastrocnemial, soleal, other
	<i>Perforating veins (A_P)</i>
17	Thigh
18	Calf

Pathophysiological Classification (P_{R,O})

Clinical signs and symptoms of venous dysfunction may be the result of reflux (P_R), obstruction (P_O) or both (P_{R,O}). Therefore, the simplest pathophysiological classification of a limb would be P_R, P_O or P_{R,O}.

Because the severity of venous dysfunction is determined by the location and anatomical extent of reflux and/or obstruction [4,5], it may be desirable to report this in greater detail by using the anatomical segments listed in Table 4. The availability of duplex scanning allows this to be done non-invasively [6,13]. In addition, it may be appropriate to report duplex-derived severity and duration of reflux [8,9,14], as presented in part III.

Reporting of segmental obstruction can be simplified and standardized using the well-recognized major sites of occlusion [15]: caval, iliac, femoral, popliteal and crural (P_{0-Cav}, P_{0-I}, P_{0-F}, P_{0-P}, P_{0-C}). If obstruction is more extensive, this can also be reported using multiple subscripts (e.g. P_{0-I,F,P}). Functional obstruction is discussed in part III.

Part II: Scoring of Venous Dysfunction

A scoring system of chronic venous dysfunction provides a numerical base for scientific comparison of limb condition and evaluation of results of treatment. This is based on three elements: the number of anatomical segments affected (anatomical score); grading of symptoms and signs (clinical score); and disability (disability score). Although the grading of symptoms is subjective, the grading of signs is objective. The accuracy of this scoring system needs to be tested and may be modified in the future as experience accumulates.

Anatomical Score

This is the sum of the anatomical segments, each scored as 1 point (Table 4).

Clinical Score

This is the sum of the values assigned to the signs and symptoms listed in Table 5.

Table 5. Clinical score

Pain	0 = none; 1 = moderate, not requiring analgesics; 2 = severe, requiring analgesics
Oedema	0 = none; 1 = mild/moderate; 2 = severe
Venous claudication	0 = none; 1 = mild/moderate; 2 = severe
Pigmentation	0 = none; 1 = localized; 2 = extensive
Lipodermatosclerosis	0 = none; 1 = localized; 2 = extensive
Ulcer - Size (largest ulcer)	0 = none; 1 = <2 cm diameter; 2 = >2 cm diameter
Ulcer - duration	0 = none; 1 = <3 months; 2 = >3 months
Ulcer - recurrence	0 = none; 1 = once; 2 = more than once
Ulcer - number	0 = none; 1 = single; 2 = multiple

Disability Score

This is derived from the categories outlined in Table 6.

Table 6. Disability score

0	Asymptomatic
1	Symptomatic, can function without support device
2	Can work 8-hour day <i>only</i> with support device
3	Unable to work even with support device

Part III: The Diagnostic Process

The history and physical examination are the basis for the initial evaluation of patients with suspected chronic venous disease [16]. Since valvular incompetence or obstruction form the basis for most complications,

continuous-wave (CW) Doppler can be used at the time of the initial clinical evaluation to assist in the diagnosis [17,18]. Absence or diminution of a Doppler velocity signal despite an augmentation manoeuvre suggests obstruction. Reflux may be detected with a Valsalva manoeuvre or limb compression. Because CW Doppler provides subjective information, if positive, findings should be followed by objective tests.

If a patient presents with symptoms that are questionably related to venous disease, such as mild oedema or aching, a non-invasive test may be required. Duplex scanning is the method of choice used to confirm or exclude the presence of venous dysfunction [6-14]. In the absence of duplex scanning, strain-gauge plethysmography [19,20], air-plethysmography (APG) [21,22] or photoplethysmography (PPG) [23,24] may be used. Because the accuracy of PPG has been challenged [25-27], confirmation of the presence of chronic venous disease by another technique may be required if PPG is positive.

Duplex scanning has become the method of choice for testing individual veins of the superficial, deep and perforating systems [6-13]. If the problem is confined to superficial veins, duplex scanning will determine whether this involves the greater and/or lesser saphenous veins and their tributaries. It can also detect the presence of incompetent perforating veins [5,28,29]. In addition, duplex scanning can determine the anatomy of veins in the popliteal fossa [30-32]. Also, it will detect reflux at other sites such as vulval veins or lateral thigh incompetent perforating veins. In the presence of deep venous disease, duplex scanning will determine whether the problem is due to anatomical obstruction, reflux or both. In addition, it will provide information about the anatomical extent. Measurements to quantify reflux in individual veins by duplex scanning have been recently developed, e.g. valve closure time [9], venous reflux index [33] and velocity at peak reflux [14], but experience with these is still limited [34]. Several other methods to quantify reflux are available. They include strain-gauge plethysmography [19,20], foot volumetry [35,36] and the more recently developed air-plethysmography [21,22], which measures global reflux in ml/s. Ascending and descending phlebography should be performed when deep venous valvular reconstruction is contemplated [37,38].

Several tests are available to determine the functional severity of chronic obstruction. They include the arm-foot pressure differential [39], the outflow fraction using air-plethysmography [16,40] and femoral or popliteal pressure measurements during exercise [41,42]. Ascending phlebography should be performed if venous reconstruction (bypass) is being considered.

Ambulatory venous pressure is a test measuring global venous hypertension [43,44]. A high ambulatory venous pressure is associated with a high incidence of ulceration [45].

In the presence of both obstruction and reflux, the quantitative tests outlined above can be used to assess which is predominant.

References

1. Reporting standards in venous disease. Prepared by the Subcommittee on Reporting Standards in Venous Disease, Ad Hoc Committee on Reporting Standards, Society for Vascular Surgery/North American Chapter, International Society for Cardiovascular Surgery. *J Vasc Surg* 1988;8:172-81.
2. Goldman MP. Sclerotherapy: treatment of varicose and telangiectatic leg veins. St Louis: Mosby Year Book, 1991.
3. Weiss RA, Weiss MA. Resolution of pain associated with varicose telangiectatic leg veins after compression sclerotherapy. *J Dermatol Surg Oncol* 1990;16:333-6.
4. Gooley NA, Sumner DS. Relationship of venous reflux to the site of venous valvular incompetence: implications for venous reconstructive surgery. *J Vasc Surg* 1988;7:50-9.
5. Hanrahan LM, Araki CT, Rodriguez AA, Kechejian GJ, LaMorte WW, Menzoian JO. Distribution of valvular incompetence in patients with venous stasis ulceration. *J Vasc Surg* 1991;13:805-12.
6. Szendro G, Nicolaides AN, Zukowski AJ, et al. Duplex scanning in the assessment of deep venous incompetence. *J Vasc Surg* 1986;4:237-42.
7. van Bemmelen PS, Bedford G, Beach K, Strandness DE. Quantitative segmental evaluation of venous valvular reflux with duplex ultrasound scanning. *J Vasc Surg* 1989;10:425-31.
8. Neglen 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* 1992;16:687-93.
9. Welch HJ, Faliakou EC, McLaughlin RL, et al. Comparison of descending phlebography with quantitative photoplethysmography, air plethysmography, and duplex quantitative valve closure time in assessing deep venous reflux. *J Vasc Surg* 1992;16:913-20.
10. Masuda EM, Kistner RL. Prospective comparison of duplex scanning and descending venography in the assessment of venous insufficiency. *Am J Surg* 1992;164:254-9.
11. Valentin LI, Valentin WH, Mercado S, Rosado CJ. Venous reflux localisation: comparative study of venography and duplex scanning. *Phlebology* 1993;8:124-7.
12. Lees TA, Lambert D. Patterns of venous reflux in limbs with skin changes associated with chronic venous insufficiency. *Br J Surg* 1993;80:725-8.
13. Labropoulos N, Leon M, Nicolaides AN, et al. Venous reflux in patients with previous deep venous thrombosis: correlation with ulceration and other systems. *J Vasc Surg* 1994;20 (in press).
14. Vasdekis SN, Clarke GH, Nicolaides AN. Quantification of venous reflux by means of duplex scanning. *J Vasc Surg* 1989;10:670-7.
15. May R, Nissl R. The post-thrombotic syndrome. In: May R, editor. *Surgery of the veins of the leg and pelvis*. Stuttgart: Georg Thieme, 1979.
16. Nicolaides AN, Summer DS. Investigation of patients with deep vein thrombosis and chronic venous insufficiency. London: Med-Orion, 1991.
17. Barnes RW, Ross EA, Strandness DE Jr. Differentiation of primary from secondary varicose veins by Doppler ultrasound and strain gauge plethysmography. *Surg Gynecol Obstet* 1975;141:207-11.
18. Shull KC, Nicolaides AN, Fernandes e Fernandes J, et al. Significance of popliteal reflux in relation to ambulatory venous pressure and ulceration. *Arch Surg* 1979;114:1304-6.
19. Barnes RW, Ross, Standness DE Jr. Differentiation of primary from secondary varicose veins by Doppler ultrasound and strain gauge plethysmography. *Surg Gynecol Obstet* 1975;141:207-11.
20. Fernandes JF, Horner J, Needham T, Nicolaides A. Ambulatory calf volume plethysmography in the assessment of venous insufficiency. *Br J Surg* 1979;66:327-30.
21. Christopoulos DG, Nicolaides AN, Szendro G, Irvine AT, Bull ML, Eastcott HH. Air-plethysmography and the effect of elastic compression on venous hemodynamics of the leg. *J Vasc Surg* 1987;5:148-59.
22. Katz ML, Comerota AJ, Kerr R. Air plethysmography (APG). A new technique to evaluate patients with chronic venous insufficiency. *J Vasc Technol* 1991;15:23-7.
23. Abramowitz HB, Queral LA, Flinn WR, et al. The use of photoplethysmography in the assessment of venous insufficiency: a comparison to venous pressure measurements. *Surgery* 1979;86:434-41.
24. Nicolaides AN, Miles C. Photoplethysmography in the assessment of venous insufficiency. *J Vasc Surg* 1987;5:405-12.
25. van Bemmelen PS, van Ramshorst B, Eikelboom BC. Photoplethysmography re-examined: lack of correlation with duplex scanning. *Surgery* 1992;112:544-8.
26. Masser PA, De Frang RD, Gentile A, et al. Choice of tests for vascular laboratory evaluation of venous reflux. *J Vasc Technol* 1994 (accepted for publication).
27. Bays MA, Healy DA, Atnip RG, Thiele BL, Neumyer MM. Validation of air plethysmography, photoplethysmography and duplex ultrasound in the evaluation of severe venous stasis. Presented at the American Venous Forum, 23-25 February 1994, Maui, Hawaii.
28. Hanrahan LM, Araki CT, Fisher JB, et al. Evaluation of the perforating veins of the lower extremity using high resolution duplex imaging. *J Cardiovasc Surg* 1991;32:87-97.
29. Myers KA, Ziegenbein RW, Zeng GH, Matthews PG. Pattern of medial calf perforator reflux shown by duplex ultrasound scanning in 1130 legs with chronic venous disease. Presented at the American Venous Forum, 23-25 February, 1994, Maui, Hawaii.
30. Hobbs JT. Errors in the differential diagnosis of incompetence of the popliteal vein and short saphenous vein by Doppler ultrasound. *J Cardiovasc Surg* 1986;27:169-74.
31. Vasdekis SN, Clarke GH, Hobbs JT, Nicolaides AN. Evaluation of non-invasive and invasive methods in the assessment of short saphenous vein termination. *Br J Surg* 1989;76:929-32.
32. Engel AF, Davies G, Keeman JN. Preoperative localisation of the saphenopopliteal junction with duplex scanning. *Eur J Vasc Surg* 1991;5:507-9.
33. Beckwith TC, Richardson G, Sheldon M, Clarke GH. A correlation between blood flow volume and ultrasonic Doppler wave forms in the study of valve efficiency. *Phlebology* 1993;8:12-6.
34. Araki CT, Back TL, Padberg FT Jr, Thompson PN, Duran WN, Hobson RW. Refinements in the ultrasonic detections of popliteal vein reflux. *J Vasc Surg* 1993;18:742-8.
35. Norgren L. Functional evaluation of chronic venous insufficiency by foot volumetry. *Acta Chir Scand Suppl* 1974;444:1-48.
36. Bradbury AW, Stonebridge PA, Callam MJ, Ruckley CV, Allan PL. Foot volumetry and duplex ultrasonography after saphenous and subfascial perforating vein ligation for recurrent venous ulceration. *Br J Surg* 1993;80:845-8.
37. Raju S, Fredericks R. Venous obstruction: an analysis of 137 cases with hemodynamic, venographic, and clinical correlations. *J Vasc Surg* 1991;14:305-13.
38. Kistner RL, Ferris EB, Randhawa G, Kamida C. A method of performing descending venography. *J Vasc Surg* 1986;4:464-8.
39. Raju S. New approaches to the diagnosis and treatment of venous obstruction. *J Vasc Surg* 1986;4:42-54.
40. Neglen P, Raju S. Detection of outflow obstruction on chronic venous insufficiency. *J Vasc Surg* 1993;17:583-9.
41. Albrechtsson U, Einarsson E, Eklöf B. Femoral vein pressure measurements for evaluation of venous function in patients with postthrombotic iliac veins. *Cardiovasc Intervent Radiol* 1981;4:43-50.
42. Perrin M. *Chronic venous insufficiency in the lower limbs*. Paris: McGraw Hill, 1990.
43. Nicolaides AN, Zukowski AJ. The value of dynamic venous pressure measurements. *World J Surg* 1986;10:919-24.
44. Welkie JF, Comerota AJ, Katz ML, Aldridge SC, Kerr RP, White JV. Hemodynamic deterioration in chronic venous disease. *J Vasc Surg* 1992;16:733-40.
45. Nicolaides AN, Hussein MK, Szendro G, Christopoulos D, Vasdekis S, Clarke H. The relation of venous ulceration with ambulatory venous pressure measurements. *J Vasc Surg* 1993; 17:414-9.