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CONSENSUS STATEMENT

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

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Part I: Classification

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 etiology. 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 following headings:

Table 1. Classification.

C	for Clinical signs (Grade $_{0-6}$), supplemented by ($_A$) for asymptomatic and ($_S$) for symptomatic presentation
E	for Etiologic Classification (Congenital, Primary, Secondary)
A	for Anatomic Distribution (Superficial, Deep, or Perforator, alone or in combination)
P	for Pathophysiologic Dysfunction (Reflux or Obstruction, alone or in combination)

Clinical classification (C_{0-6})

The clinical classification is based on objective clinical signs of chronic venous disease (C_{0-6}) supplemented according to presentation (A) for 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 organised in terms of ascending

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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	Edema
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]

Etiologic classification (E_C, E_P or E_S)

This etiologic classification recognises three categories of venous dysfunction: congenital, primary, and secondary. Congenital problems may be apparent at birth or be recognised later. Primary problems are neither congenital nor do they have an identifiable cause. Secondary problems are those acquired conditions that have a known pathologic cause, such as thrombosis. These categories are mutually exclusive.

Table 3. Etiologic classification.

Congenital (E_C)
Primary (E_P) — with undetermined cause
Secondary (E_S) — with known cause
Post-thrombotic
Post-traumatic
Other

Anatomic classification ($A_{S,D,P}$)

This classification describes the anatomic 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 categorised using the anatomic segments listed in Table 4.

Table 4. Anatomic classification.

Segment #	Superficial veins (A_S):
1	Telangiectases/reticular veins
	Greater (long) saphenous (GSV)
2	Above-knee
3	Below-knee
4	Lesser (short) saphenous (LSV)
5	Non-saphenous
	Deep veins (A_D):
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
	Perforating veins (A_P):
17	Thigh
18	Calf

Pathophysiologic 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 pathophysiologic classification of a limb would be P_R, P_O, or $P_{R,O}$.

Table 5. Pathophysiologic classification.

Reflux (P_R)
Obstruction (P_O)
Reflux and Obstruction ($P_{R,O}$)

Because the severity of venous dysfunction is determined by the location and anatomic extent of reflux and/or obstruction[4,5] it may be desirable to report this in greater detail by using the anatomic 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 standardised using the well-recognised major sites of occlusion,[15] caval, iliac, femoral, popliteal and crural ($P_{O\text{-}Cav}$, $P_{O\text{-}I}$, $P_{O\text{-}F}$, $P_{O\text{-}P}$, $P_{O\text{-}C}$).

If obstruction is more extensive, this can also be reported using multiple subscripts (e.g. $P_{O\text{-}I,F,P}$). 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 anatomic segments affected (anatomic 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.

Anatomic score

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

Clinical score

This is the sum of the values assigned to the signs and symptoms listed below:

Table 6. Clinical score.

Pain	(0 = none; 1 = moderate, not requiring analgesics; 2 = severe, requiring analgesics)
Edema	(0 = none; 1 = mild/moderate; 2 = severe)
Venous Claudication	(0 = none; 1 = mild/moderate; 2 = severe)
Pigmentation	(0 = none; 1 = localised; 2 = extensive)
Lipodermatosclerosis	(0 = none; 1 = localised; 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)

Table 7. Disability score

0	Asymptomatic
1	Symptomatic, can function without support device
2	Can work 8-hour day only 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, the 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 maneuver 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 test.

If a patient presents with symptoms that are questionably related to venous disease such as mild edema 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,[17,19] air-plethysmography (APG)[20,21] or photoplethysmography (PPG)[22,23] may be used. Because the accuracy of PPG has been

challenged,[24–26] 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.[5,27,28] In addition, duplex scanning can determine the anatomy of veins in the popliteal fossa.[29–31] 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 anatomic obstruction, reflux or both. In addition, it will provide information about the anatomic extent. Measurements to quantify reflux in individual veins by duplex scanning have been recently developed such as valve closure time,[9] venous reflux index[32] and velocity at peak reflux[14] but experience with these is still limited.[33] Several other methods to quantify reflux are available. They include strain-gauge plethysmography,[17,19] foot volumetry[34,35] and the more recently developed air-plethysmography,[20,21] which measures global reflux in ml/s. Ascending and descending phlebography should be performed when deep venous valvular reconstruction is contemplated.[36,37]

A number of tests are available to determine the functional severity of chronic obsruction. They include the arm-foot pressure differential,[38] the outflow fraction using air-plethysmography[16,39] and femoral or popliteal pressure measurements during exercise.[40,41] Ascending phlebography should be performed if venous reconstruction (bypass) is being considered.

Ambulatory venous pressure is a test measuring global venous hypertension.[42,43] A high ambulatory venous pressure is associated with a high incidence of ulceration.[44]

In the presence of both obstruction and reflux, 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**: 172–181.

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**: 333–336.

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**: 50–59.

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**: 805–812.

6 Szendro G, Nicolaides AN, Zukowski AJ *et al.* Duplex scanning in the assessment of deep venous incompetence. *J Vasc Surg* 1986; **4**: 237–242.

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**: 425–431.

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**: 687–693.

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**: 913–920.

10 Masuda EM, Kistner RL. Prospective comparison of duplex scanning and descending venography in the assessment of venous insufficiency. *Am J Surg* 1992; **164**: 254–259.

11 Valentin LI, Valentin WH, Mercado S, Rosado CJ. Venous reflux localisation: Comparative study of venography and duplex scanning. *Phlebology* 1993; **8**: 124–127.

12 Lees TA, Lambert D. Patterns of venous reflux in limbs with skin changes associated with chronic venous insufficiency. *Br J Surg* 1993; **80**: 725–728.

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**: 20–26.

14 Vasdekis SN, Clarke GH, Nicolaides AN. Quantification of venous reflux by means of duplex scanning. *J Vasc Surg* 1989; **10**: 670–677.

15 May R, Nissl R. The post-thrombotic syndrome. In: May R, ed. *Surgery of the veins of the leg and pelvis.* Stuttgart: Georg Thieme, 1979.

16 Nicolaides AN, Sumner 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**: 207–211.

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**: 1304–1306.

19 Fernandes JF, Horner J, Needham T, Nicolaides A. Ambulatory calf volume plethysmography in the assessment of venous insufficiency. *Br J Surg* 1979; **66**: 327–330.

20 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**: 148–159.

21 Katz ML, Comerota AJ, Kerr R. Air Plethysmography (APG) A new technique to evaluate patients with chronic venous insufficiency. *J Vasc Technol* 1991; **15**: 23–27.

22 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**: 434–441.

23 Nicolaides AN, Miles C. Photoplethysmography in the assessment of venous insufficiency. *J Vasc Surg* 1987; **5**: 405–412.

24 VAN BEMMELEN PS, VAN RAMSHORST B, EIKELBOOM BC. Photoplethysmography reexamined: lack of correlation with duplex scanning. *Surgery* 1992; **112**: 544–548.
25 MASSER PA, DE FRANG RD, GENTILE A *et al.* Choice of tests for vascular laboratory evaluation of venous reflux. *J Vasc Tech* 1994 (in press).
26 BAYS RA, 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, February 23–25, 1994 Maui, Hawaii.
27 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.
28 MYERS KA, ZIEGENBEIN RW, ZENG GH, MATTHEWS PG. Patterns of medial calf perforator reflux shown by duplex ultrasound scanning in 1130 legs with chronic venous disease. Presented at the American Venous Forum, February 23–25, 1994, Maui, Hawaii.
29 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**: 169–174.
30 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**: 929–932.
31 ENGEL AF, DAVIES G, KEEMAN JN. Preoperative localisation of the saphenopopliteal junction with duplex scanning. *Eur J Vasc Surg* 1991; **5**: 507–509.
32 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**: 12–16.
33 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**: 742–748.
34 NORGREN L. Functional evaluation of chronic venous insufficiency by foot volumetry. *Acta Chir Scand Suppl* 1974; **444**: 1–48.
35 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**: 845–848.
36 RAJU S, FREDERICKS R. Venous obstruction: an analysis of one hundred thirty-seven cases with hemodynamic, venographic, and clinical correlations. *J Vasc Surg* 1991; **14**: 305–313.
37 KISTNER RL, FERRIS EB, RANDHAWA G, KAMIDA C. A method of performing descending venography. *J Vasc Surg* 1986; **4**: 464–468.
38 RAJU S. New approaches to the diagnosis and treatment of venous obstruction. *J Vasc Surg* 1986; **4**: 42–54.
39 NEGLEN P, RAJU S. Detection of outflow obstruction in chronic venous insufficiency. *J Vasc Surg* 1993; **17**: 583–589.
40 ALBRECHTSSON U, EINARSSON E, EKLOF B. Femoral vein pressure measurements for evaluation of venous function in patients with postthrombotic iliac veins. *Cardiovasc Intervent Radiol* 1981; **4**: 43–50.
41 PERRIN M. *Chronic venous insufficiency in the lower limbs.* Paris: McGraw Hill; 1990.
42 NICOLAIDES AN, ZUKOWSKI AJ. The value of dynamic venous pressure measurements. *World J Surg* 1986; **10**: 919–924.
43 WELKIE JF, COMEROTA AJ, KATZ ML, ALDRIDGE SC, KERR RP, WHITE JV. Hemodynamic deterioration in chronic venous disease. *J Vasc Surg* 1992; **16**: 733–740.
44 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**: 414–419.

Commentary

The aim of providing a classification with the purpose of putting order and comparability into communications on chronic venous disease is warmly applauded. The diversity of diagnostic tools and therapeutic options in recent years underscores the need. The Hawaii group have made a brave attempt.

It has to be said however that the medical literature is littered with classifications which have not received general endorsement. Usually this is because of excessive complexity. As the authors state it needs to be "simple enough to encourage universal acceptance". A coding or scoring system which requires either feats of memory or constant reference to keys will not be used. The classification needs to be practically orientated both in terms of patient identification and treatment options; enabling patients to be placed into mutually exclusive categories.

The Hawaii group have attempted to make their classification all-encompassing. This, from a practical clinical viewpoint is not necessary. Secondly, within some of the tables, attempts have been made to classify on more than one theme. For example it is not possible to have a rational classification which provides a gradation of severity and at the same time separately categorises a range of pathologies some of which are specific to venous disease (lipodermatosclerosis) and some of which are not (edema); some which are indices of severity of venous insufficiency (active ulcer) and some are not (telangiectasis). What is required is a working classification of chronic venous disease relating to valvular incompetence and/or obstruction. Congenital venous anomalies are, in our opinion, a separate clinical problem and better dealt with independently.

We would propose that nothing more is required than two basic classifications: the first an index of severity based on symptomatology. For this we suggest the following modification of Table 2:-

Table 2. Clinical presentation.

Grade 1	Asymptomatic
Grade 2	Cosmetic/trivial pain or swelling
Grade 3	Severe pain/swelling
Grade 4	Skin change (lipodermatosclerosis) without ulcer
Grade 5	Chronic ulceration

It is conceded that even within so simple a classification there may be difficulties. The point at which "skin change" is said to have occurred may be difficult to define. When does an ulcer become "chronic". Our

working definition in clinical trials is 4 weeks. These limitations, however, are arguments in favour of pragmatic simplicity rather than increasing complexity which compound the difficulties.

The second classification we propose is one of simple morphology which can be easily applied and related to treatment options:-

Morphology

1. Primary
   - 1.1 Superficial incompetence alone (Long/short saphenous + /– perforator incompetence)
   - 1.2 Deep incompetence + /– superficial incompetence (without evidence of post-phlebitic damage)
2. Secondary (post-phlebitic/traumatic)

The criteria for establishing the definition of incompetence are likely to vary. These would either have to be stated in individual communications or universally agreed. There may be uncertainty as to the presence or absence of post-phlebitic damage, a critical factor in outcome, but this is a problem for any system. We feel it better to combine "trauma" and "post-phlebitic" under a single category. The distinction is of academic interest only. The implications are the same.

A point worthy of further debate is whether there needs to be further refinement of patients with primary deep incompetence with or without co-existing superficial (saphenous) incompetence. Similarly the separation of perforator incompetence from deep or superficial incompetence, or both, would lead to added layers of complexity, best left for authors to enumerate at the time of communication.

Regarding the remainder of the classification, we do not see a practical role for the anatomical classification. Currently communications which present information on venous anatomy do so quite adequately utilising existing anatomical terms. If the purpose of the classification were to enable the researcher to construct a mathematical score of involved venous segments this classification would not help.

The scoring of venous dysfunction duplicates and further complicates Table 2. We are doubtful as to the precision or utility of the disability score. Diagnostic process is clearly of value and could be agreed more widely and then possibly integrated into the definitions of morphology.

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