

Editors' Choice

The 2020 update of the CEAP classification system and reporting standards



Fedor Lurie, MD, PhD,^{a,b} Marc Passman, MD,^c Mark Meisner, MD,^d Michael Dalsing, MD,^e Elna Masuda, MD,^f Harold Welch, MD,^g Ruth L. Bush, MD,^h John Blebea, MD,ⁱ Patrick H. Carpentier, MD,^j Marianne De Maeseneer, MD,^k Anthony Gasparis, MD,^l Nicos Labropoulos, MD,^l William A. Marston, MD,^m Joseph Rafetto, MD,ⁿ Fabricio Santiago, MD,^o Cynthia Shortell, MD,^p Jean Francois Uhl, MD,^q Tomasz Urbanek, MD,^r André van Rij, MD,^s Bo Eklof, MD,^f Peter Gloviczki, MD,^t Robert Kistner, MD,^f Peter Lawrence, MD,^u Gregory Moneta, MD,^v Frank Padberg, MD,^w Michel Perrin, MD,^x and Thomas Wakefield, MD,^b *Toledo, Ohio; Ann Arbor and Saginaw, Mich; Birmingham, Ala; Seattle, Wash; Indianapolis, Ind; Honolulu, Hi; Burlington and Boston, Mass; Houston, Tex; Grenoble, Paris, Lyon and Décines, France; Rotterdam, The Netherlands; Stony Brook, NY; Chapel Hill and Durham, NC; Goiania, Brazil; Katowice, Poland; Otago, New Zealand; Rochester, Minn; Los Angeles, Calif; Portland, Ore; Newark, NJ*

ABSTRACT

The CEAP (Clinical-Etiology-Anatomy-Pathophysiology) classification is an internationally accepted standard for describing patients with chronic venous disorders and it has been used for reporting clinical research findings in scientific journals. Developed in 1993, updated in 1996, and revised in 2004, CEAP is a classification system based on clinical manifestations of chronic venous disorders, on current understanding of the etiology, the involved anatomy, and the underlying venous pathology. As the evidence related to these aspects of venous disorders, and specifically of chronic venous diseases (CVD, C2-C6) continue to develop, the CEAP classification needs periodic analysis and revisions. In May of 2017, the American Venous Forum created a CEAP Task Force and charged it to critically analyze the current classification system and recommend revisions, where needed. Guided by four basic principles (preservation of the reproducibility of CEAP, compatibility with prior versions, evidence-based, and practical for clinical use), the Task Force has adopted the revised Delphi process and made several changes. These changes include adding Corona phlebectatica as the C4c clinical subclass, introducing the modifier "r" for recurrent varicose veins and recurrent venous ulcers, and replacing numeric descriptions of the venous segments by their common abbreviations. This report describes all these revisions and the rationale for making these changes. (*J Vasc Surg: Venous and Lym Dis* 2020;8:342-52.)

Keywords: Chronic venous disease; Veins; Varicose veins; Post-thrombotic syndrome; Disease classification

The development of a standardized clinical classification system of chronic venous disease (CVD) is critical to our understanding of the natural history of the disease, as well as comparing methods of diagnosis and treatment. The clinical manifestations of CVD can vary substantially between individual patients with similar pathology, making clinical scientific communications and practice guidelines difficult to implement without

CVD reporting standards. To address the complexity of the clinical manifestations of CVD, a standardized classification system (CEAP), based on our current understanding of venous pathology and the clinical manifestations of the disease, as well as its natural history, was introduced in 1996 and last revised in 2004, to provide a reliable and reproducible classification of the many manifestations of CVD.^{1,2} However, recent advances in

From the Jobst Vascular Institute, Toledo^a; the Division of Vascular Surgery, University of Michigan, Ann Arbor^b; the Department of Surgery, University of Alabama at Birmingham, Birmingham^c; the Department of Surgery, University of Washington School of Medicine, Seattle^d; the Department of Surgery, Indiana University School of Medicine, Indianapolis^e; the Department of Surgery, University of Hawaii, Honolulu^f; the Vascular Surgery, Lahey Hospital and Medical Center, Burlington^g; the University of Houston College of Medicine, Houston^h; the Department of Surgery, Central Michigan University College of Medicine, Saginawⁱ; the Grenoble University Hospital, Grenoble^j; the Erasmus MC, Rotterdam^k; the Center for Vein Care, Stony Brook University, Stony Brook^l; the Department of Surgery, University of North Carolina School of Medicine, Chapel Hill^m; the Harvard Medical School, Uniformed Services University of the Health Sciences, VA Boston HCS, Brigham and Women's Hospital, Bostonⁿ; the Venous and Lymphatics Disease Institute, Goiania^o; the Department of Surgery, Duke University Medical Center, Durham^p; the Paris Descartes University, Paris^q; the

Medical University of Silesia, Katowice^r; the Dunedin School of Medicine, University of Otago, Otago^s; the Vascular Surgery, Mayo Clinic, Rochester^t; the University of California, Los Angeles^u; the Oregon Health & Science University, Portland^v; the Rutgers New Jersey Medical School, Newark^w; and the Service de Chirurgie Vasculaire, Clinique du Grand Large.^x

Author conflict of interest: none.

Correspondence: Fedor Lurie, MD, PhD, Jobst Vascular Center, 2109 Hughes Dr, Ste 400, Toledo, OH 43606 (e-mail: fedor.lurie@promedica.org).

The editors and reviewers of this article have no relevant financial relationships to disclose per the Journal policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

2213-333X

Copyright © 2020 by the Society for Vascular Surgery. Published by Elsevier Inc.

<https://doi.org/10.1016/j.jvsv.2019.12.075>

scientific knowledge regarding CVD, as well as the adoption of CEAP by a wide spectrum of venous disease stakeholders, some of whom have had difficulty interpreting and using the current system, has mandated an update of CEAP to align it with our current understanding of CVD.

HISTORY OF CEAP

The first widely adopted venous classification, developed by Widmer³ in 1978, was based on the natural history of CVD and defined three stages of CVD. Although it was a major advance, the lack of specificity between stages I and II significantly limited the reproducibility and clinical usefulness of the Widmer system. The lack of clinical and physiologic integration in the original classification was addressed by Partsch⁴ in 1980, who added additional functional assessments of subclasses of CVD, based on anatomic involvement of superficial, perforator, and deep veins.⁵ Objective measurements proposed to assist in implementing this classification were foot volumetry and ambulatory venous pressure, which were not widely used clinically at that time.⁵ Sytchev⁶ proposed in 1985 that these tests be replaced with duplex ultrasound examination, and this modified system proved to be a more practical classification system. In the same year, Pierchalla and Tronier⁷ refined the pathologic definitions which had been advocated by Olivier and Merlen,⁸ and also proposed differentiating between primary and secondary (post-thrombotic) disease.

Despite the contributions of many investigators in the evolution of a venous classification system, there remained a need for a more precise and effective reporting standard for venous disease. Consequently, an ad hoc committee of The Society for Vascular Surgery and the North American chapter of the International Society for Cardiovascular Surgery was charged with this responsibility in 1988.⁹ The new classification system added two additional components: etiology of the CVD and the anatomic distribution, and noninvasive imaging and physiologic tests were required to accurately classify the CVD.

After the fifth Annual Meeting of the American Venous Forum (AVF), Kistner and Eklof formed a working group that included Comerota, Nicolaidis, Raju, Richardson, and Strandness. They convened the First Pacific Vascular Symposium in June of 1993 and proposed an additional pathophysiologic component in the classification of CVD. They also proposed using a standardized ultrasound assessment to determine each patient's CEAP classification. A consensus conference at the sixth annual meeting of the AVF in 1994, chaired by Nicolaidis, with representatives from Australia, Europe, and the United States, developed the first CEAP consensus document, which was published in 1996.¹ This classification system was based on clinical manifestations (C), etiologic factors (E), anatomic distribution (A), and the underlying pathophysiology (P), and was abbreviated CEAP. It was endorsed by the Joint

Council of the Society for Vascular Surgery and the North American chapter of the International Society for Cardiovascular Surgery and incorporated into CVD reporting standards. The dissemination and promotion of CEAP was assisted by the publication of the consensus statement in 26 scientific journals and textbooks in nine languages. CEAP rapidly became a requirement for classifying and reporting the severity of CVD in scientific publications.

Progress in the diagnosis and treatment of CVD increased rapidly during the 1990s, resulting in the need for an update in the CEAP classification system. In addition, although the original CEAP classification showed good intraobserver reproducibility ($\kappa = 0.54-0.86$), it had a more limited interobserver reproducibility ($\kappa = 0.39-0.55$).^{10,11} Reproducibility was also better for advanced venous disease (C₄-C₆) than for less severe C₀-C₂ disease, an observation believed to be partially related to a lack of clarity in the less severe CVD definitions. In April 2002, AVF appointed an ad hoc committee to revise and improve CEAP by 2004, 10 years after its introduction. An international ad hoc committee was again established to ensure continued universal use. The revisions of CEAP included refinements of several definitions used in describing CVD and of the C classes of CEAP. In addition, the descriptor *n*, meaning no venous abnormality identified, adding the date of classification, and the anatomic level of clinical investigation were incorporated into the document, as well as offering a simpler alternative to the full (advanced) CEAP classification.²

Since the 2004 revision of CEAP, it has become apparent that the revised classification is still limited in some areas, and there is a need for more precise nomenclature,^{12,13} better description of the properties of diagnostic tests,¹⁴ and better definitions of the underlying pathology.¹⁵

As with any reporting standard or clinical classification, CEAP has limitations. It was designed as a descriptive classification and does not attempt to measure disease severity or outcomes of therapy. The importance to some clinicians of assessing severity and outcomes sometimes leads to misuse of CEAP and has resulted in criticism of its inability to address these issues. However, the value of CEAP in clinical practice relates to its simplicity and practicality, recognizing that limited data result in some loss of specificity. Capturing more detailed information has been resisted by each CEAP revision committee because, as a detailed classification system, it requires more data entry time and expertise.

In May 2017, the AVF created a CEAP Task Force and charged it with critically analyzing the current CEAP classification system and recommending revisions, where needed.

REVISION PROCESS

To maintain the continuity and uniformity of the CEAP revision process through multiple versions, the following guiding principles were adopted by the task force.

Preservation of the reproducibility of CEAP. As a descriptive classification, the sole purpose of CEAP is to describe a patient with CVD at a single point in time. Reproducibility is the most important property of CEAP, meaning that two different clinicians assessing the same patient at the same time point should agree on the patient's CEAP classification. However, repeatability (assessing the same patient at a different time point when the disease may have progressed) and responsiveness (assessing change in CEAP after treatment) are not the focus of CEAP. Therefore, only studies that address the reproducibility and other important properties of a descriptive classification, such as specificity and precision, were considered in the analysis of evidence and revision process.

Compatibility with prior versions. Although revisions reflecting an evolution in our understanding of CVD are necessary, a substantial body of scientific literature has been accumulated during the 25 years of CEAP use worldwide. Extensive revisions of the current CEAP classification may make newer studies incompatible with the existing literature and limit opportunities for future meta-analyses and comparative evidence synthesis. Therefore, revisions should be conducted with a goal of allowing comparisons between the newest and previous versions of CEAP.

Evidence based. Revisions should only be made when they are supported by an appropriate level of evidence. A structured expert consensus, using a modified Delphi technique, was defined as a minimal standard of appropriate evidence.

Practicality. As in previous versions of CEAP, a balance is needed between a highly specific and detailed description of a patient with CVD and a classification that allows for practical use in routine clinical settings. Simplicity and practicality should remain as important principles of any CEAP revisions.

PROCESS OF REVISION

A modified Delphi process was implemented in four phases for development of a consensus regarding CEAP revisions. During the first phase, four working groups representing each category of CEAP (Table I) were formed.

Each working group collected and analyzed publications that either directly addressed the CEAP classification or provided examples of CEAP use that suggested CEAP revision was necessary. Each group made a list of suggested revisions with a detailed rationale for each suggestion. During the second phase, suggestions from all groups were shared and discussed by the entire task force as well as the advisory committee, which included representatives from prior versions of CEAP, with the intent to maintain compatibility of the current revisions with earlier versions (Table II).

Suggested revisions that resulted in disagreement among the members of the task force were returned to the corresponding working group for clarification and additional justification. This phase was concluded during a face-to-face meeting of the task force during the 30th AVF Annual Meeting in February 2018, and disputed suggestions for revision were discussed. Voting for each proposal was concluded when at least 90% of task force cast a vote, with a 75% majority necessary for accepting a revision. The voting documents reviewed by all voters included suggested revisions, description of the reasons for a revision, and supporting evidence. The voting options included undecided, with a request for discussion. The details of the process are depicted in the Fig. During the final phase, the results of the two prior rounds of voting were reviewed, to establish a consensus on all proposed revisions, reconcile a lack of a sufficient number of votes for adopting any revision, and discuss revisions that required additional consensus. The third phase was completed at the face-to-face meeting during the 31st AVF Annual Meeting in February 2019. The fourth and final phase was writing and revising the manuscript and reaching agreement by each task force member.

LIMITATIONS OF THE 2004 CEAP VERSION AND THE RATIONALE FOR REVISION

Despite the proven usefulness of CEAP, potential limitations have been identified, particularly with respect to the clinical (C) classification, the most widely used component of CEAP.¹⁶ There are fundamentally two types of instruments for the measurement of health status: *discriminative* instruments are designed to measure

Table I. Working groups of the American Venous Forum (AVF) CEAP Task Force

Co-chairs: Fedor Lurie and Marc Passman		
Group	Group leader	Group members
C	Mark Meissner	William Marston, Cynthia Shortell, Tomasz Urbanek, Fabricio Santiago
E	Elna Masuda	Michael Dalsing, John Blebea, Patrick Carpentier
A	Harold Welch	Anthony Gasparis, André van Rij, Marianne De Maeseneer
P	Ruth L. Bush	Nicos Labropoulos, Joseph Rafetto, Jean Francois Uhl

Table II. Advisory Committee of the American Venous Forum (AVF) CEAP Task Force

Bo Eklof
Robert Kistner
Peter Gloviczki
Peter Lawrence
Gregory Moneta
Frank Padberg
Thomas Wakefield
Michel Perrin

cross-sectional differences between individuals at a single point in time, whereas *evaluative* instruments are designed to measure longitudinal changes within people over time.^{16,17} Statistically, these instruments are very different, and although both depend on a high ratio of signal to noise (measurement error), the signal in the case of discriminative instruments indicates differences between patients, whereas for evaluative instruments it denotes longitudinal changes within patients that reflect changes in health status.¹⁷ Discriminative instruments should include key components of the disease that are

stable, at least over short periods of time; have a limited number of options and clear definitions that enable uniform interpretation; and have large and stable between-subject variation.¹⁸ Notably, responsiveness to change is not a relevant consideration for discriminative instruments. From a simplistic standpoint, discriminative instruments place patients into homogenous bins with similar clinical features, natural histories, and response to treatment, whereas evaluative instruments measure improvement or deterioration in response to treatment or the natural history of the disease. In the specific case of venous disease, CEAP was designed to be a purely discriminative instrument; the Venous Clinical Severity Score (VCSS) is its evaluative complement.^{19,20}

At least some of the criticisms of CEAP stem from misconceptions that CEAP is an evaluative instrument that quantitatively measures severity and change over time or in response to treatment, rather than being a descriptive instrument designed to categorize patients. Although the C classification is arranged such that more severe manifestations of venous disease are assigned a higher category, CEAP is purely a categorical instrument, not a linear ascending score. Thus, in describing patient populations, absolute numbers and percentages of each category should be presented, rather than a mean score. Although few would argue that disease severity is substantially worse in a patient with a venous ulcer than in a patient with uncomplicated varicose veins, CEAP is not a quantitative severity scale or scoring system and is not designed to reflect changes over time. Other evaluative instruments, such as clinical severity scores (eg, the VCSS) and patient-reported outcome measures (Aberdeen Varicose Vein Questionnaire, Chronic Venous Disease Quality of Life Questionnaire, VEnous INsufficiency Epidemiological and Economic Study—Quality of Life/Symptoms) are designed specifically for this purpose.²¹

Despite these misconceptions regarding CEAP, a number of potential shortcomings that were identified in the literature and reviewed by the task force include the following.

1. The C₀ category, designating no visible or palpable signs of venous disease and including both asymptomatic (A) and symptomatic (S) patients, is perhaps the most overlooked category in CEAP. However, it has been noted that symptomatic patients (C_{0s}) actually comprise two groups of patients: those with venous symptoms, no signs of venous disease, with reflux or obstruction identified on routine investigation; and a second group with venous symptoms, no venous signs, and no pathologic findings. The former would be designated as C_{0s} E_p or s A_{s, d} and/or p P_r or o and the latter as C_{0s} E_n A_n P_n. It has accordingly been suggested that C_{0s} be subdivided into two subclasses based on the presence or absence of reflux or

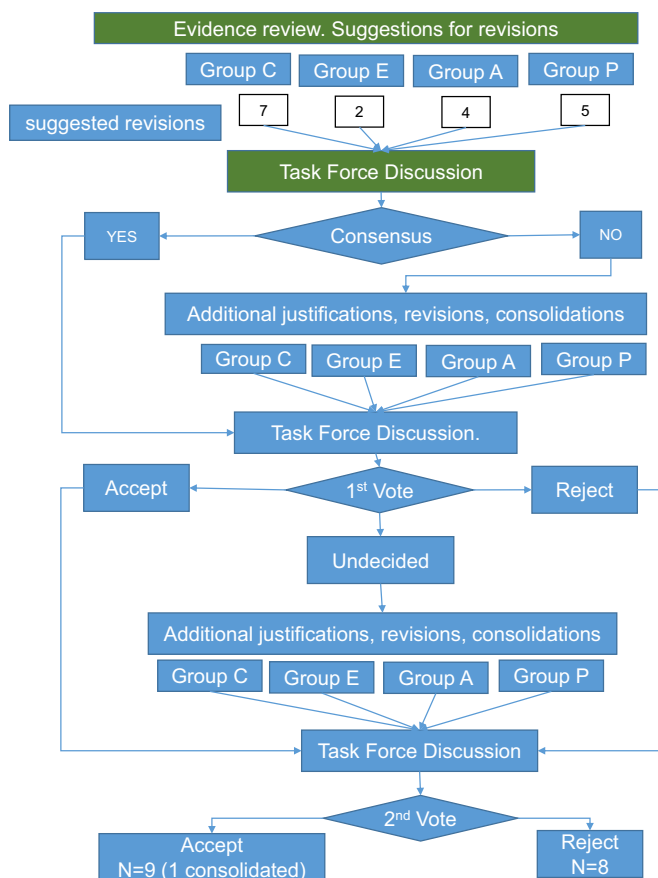


Fig. Evidence review: suggestions for revisions.

obstruction on routine investigative studies. However, because these subclasses are well-defined using the E, A, and P classifications, subdividing C₀ would unnecessarily complicate the instrument.

2. The C₂ clinical class includes varicosities of variable origin—saphenous, accessory saphenous, and nonsaphenous—which may have different implications with respect to natural history and treatment.¹⁵ Although this may be true, even basic CEAP is designed such that the C classification is only one component of a patient or population's description. The C designation is specifically designed to be used in conjunction with the appropriate E, A, and P descriptors, which should account for differences in anatomy.
3. The diameter of the vein may influence both symptoms and treatment and is not included in CEAP.^{10,16}
4. The C₃ category is overly broad and does not include potentially important subcategories of edema. The C₃ class does not quantify the degree or extent of edema or recognize other causes of leg edema.¹⁶ For example, transitory ankle edema at the end of the day likely has a different pathophysiology and natural history than severe permanent edema, which may also have different implications for treatment. In addition, bilateral edema is more likely due to a systemic disease than unilateral edema which invariably had a regional or local component. The current C₃ category also fails to recognize the degree of associated induration (firm vs soft)²⁰ and does not specifically recognize phlebolymphe²⁰demia.
5. Corona phlebectatica is not recognized as a more advanced sign of CVD. This criticism is perhaps the most common of CEAP, because many investigators consider corona phlebectatica to be an early marker of progression from uncomplicated to advanced CVD.^{10,22}
6. CEAP does not recognize recurrent varicose veins after intervention.²²
7. CEAP does not differentiate between an initial and recurrent ulcer.
8. There is a lack of strict additivity of C categories; that is, not all patients with advanced venous disease, specifically venous ulcers, demonstrate all manifestations of C₁ through C₄ disease.²³ As discussed, as a descriptive instrument, CEAP is not intended to have a strictly linear progression from one C class to the next. Because discriminative instruments optimally demonstrate cumulative scaling, this is potentially a valid criticism. However, CEAP is consistent with our understanding of the pathophysiology of venous disease and it is recognized that cumulative scaling is not possible for this instrument.
9. Despite being designed for the classification of lower extremity venous disease, CEAP does not adequately account for lower extremity manifestations of pelvic and abdominal venous disorders, including venous claudication and pelvic origin lower extremity varices. Efforts are currently in progress to develop a classification scheme for pelvic and abdominal venous disease and are beyond the scope of this revision of CEAP.

However, given the frequent inter-relationship between abdominal, pelvic, and lower extremity venous disorders, it is clear that any such classification should be both consistent with and complementary to CEAP.

10. The secondary CVD category of CEAP includes only intravenous causes of venous disease (venous wall and/or valve damage resulting from conditions that include DVT, traumatic arteriovenous fistulas, primary intravenous sarcoma, or other). The extravenous causes, in which case there is no obligatory venous wall or valve damage, yet symptoms are present owing to a condition affecting venous hemodynamics either locally or systemically, are not included in the classification. Such conditions include central venous hypertension (obesity,²⁴⁻²⁸ congestive heart failure,²⁹⁻³¹ nutcracker syndrome, and pelvic and venous congestion), extrinsic compression, or muscle pump dysfunction owing to motor disorders (paraplegia, arthritis, chronic immobility, frozen ankle, severe sedentary state³²⁻⁴¹).
11. Using numbers for venous segments under A of CEAP is not practical. In clinical practice as well as in the literature, standard abbreviations for venous segments are used almost exclusively.
12. Addressing all four components of the CEAP classification provides much more complete description of an individual patient and patient population than using just a clinical class C. The majority of publications to date do not directly state the E, A, and P components of the classification, although frequently provide description of patients included in the studies as having primary disease (E_p), and describing anatomic distribution of reflux, obstructed venous segments (A and P), and so on. Such indirect use of CEAP classification is partially due to perceived complexity of this instrument, and to an inconvenience of replacing routine set of terminology with a new system.

REVISIONS TO THE CLINICAL (C) CLASSIFICATION

Although incorporating many observations made since the last revision, the fundamental tenets of Clinical (C) CEAP remain unchanged in this revision. The clinical definitions included in the 2004 revision of CEAP for each classification have been preserved. The new revised C designation is presented in [Table III](#). As in the 2004 revision, basic CEAP should report the single highest C classification in a limb, and advanced CEAP should report all C classes present in the limb. Each clinical class should be further characterized by a subscript indicating the presence (symptomatic, *s*) or absence (asymptomatic, *a*) of symptoms. Approved revisions to the C designation include the following.

Subscript for C₂ and C₆ classes recurrent (*r*) disease.

Venous diseases, particularly varicose veins (C₂) and venous ulcers (C₆), tend to recur. Recurrent disease is a

Table III. The 2020 revision of CEAP: Summary of clinical (C) classifications

C class	Description
C ₀	No visible or palpable signs of venous disease
C ₁	Telangiectasias or reticular veins
C ₂	Varicose veins
C _{2r}	Recurrent varicose veins
C ₃	Edema
C ₄	Changes in skin and subcutaneous tissue secondary to CVD
C _{4a}	Pigmentation or eczema
C _{4b}	Lipodermatosclerosis or atrophie blanche
C _{4c}	Corona phlebectatica
C ₅	Healed
C ₆	Active venous ulcer
C _{6r}	Recurrent active venous ulcer

CVD, Chronic venous disease.
Each clinical class subcharacterized by a subscript indicating the presence (symptomatic, s) or absence (asymptomatic, a) of symptoms attributable to venous disease.

clinical definition including true recurrences, residual veins, and varicose veins occurring as a consequence of disease progression after prior treatment.^{42,43} In some cases of recurrent varicose veins and venous ulcers, recurrent disease may have a different natural history and may require different treatment strategies. Rather than subcategorizing the C₂ and C₆ categories, the subscript (r) for recurrence has been added to the class designation (ie, C_{2r} or C_{6r}).

Addition of corona phlebectatica to the C₄ class and subdivision into 3 categories (C_{4a}, C_{4b}, and C_{4c}). According to the 2004 revision of CEAP, corona phlebectatica is defined as a fan-shaped pattern of numerous small intradermal veins on the medial or lateral aspects of the ankle and foot. Synonyms include malleolar flare and ankle flare. Although such lesions would likely be classified as telangiectasias (C₁) in the 2004 revision of CEAP, many venous authorities consider corona phlebectatica to be an early sign of advanced venous disease and to warrant inclusion in more advanced C categories. Clinical data from a series of 872 patients evaluated by 49 venous specialists demonstrated a statistical association between corona phlebectatica and more advanced C clinical class.²³ A pattern of blue telangiectasias, consistent with corona phlebectatica, has also been found to have good sensitivity (91%) but marginal specificity (52%) for advanced venous disease (C₄-C₆).⁴⁴ Perhaps most importantly, patients with corona phlebectatica have been demonstrated to be 5.3 times more likely to develop an ulcer, a risk of similar magnitude to other C₄ skin changes.⁴⁵ To account for this association of corona phlebectatica with more advanced venous disease, designation of corona phlebectatica as C_{4c} was made, leaving hyperpigmentation or eczema as C_{4a} and lipodermatosclerosis or atrophie blanche as C_{4b} unchanged.

Although CEAP is a descriptive classification, some elements of this classification are listed in the order that may be seen as an increased severity of the signs. For example, varicose veins are classified as C₂ and venous ulcer as C₆. Keeping this order of listing in the revised classification should not be interpreted as a reference to clinical severity. An appropriate instrument, such as the VCSS, should be used to assess severity of the disease.

REVISIONS TO THE ETIOLOGIC (E) CLASSIFICATION

Documenting etiology of venous disease is critically important, as it determines prognosis, guides treatment choices, and affects outcomes. The 2004 CEAP stratified E classification into congenital, primary, and secondary categories. Although etiologies have not changed in the revisions, more information on the subgroups has been recommended, resulting in a clearer description of each E subclass of the CEAP classification. The revised E designation is presented in Table IV. Approved revisions to the E designation include the following.

Refinement of the definition of E_p. The primary etiologic subclass is one of the most common causes of venous insufficiency, yet description of primary disease is

Table IV. The 2020 revision of CEAP: Summary of etiologic (E) classification

E class	Description
E _p	Primary
E _s	Secondary
E _{si}	Secondary – intravenous
E _{se}	Secondary – extravenous
E _c	Congenital
E _n	No cause identified

often under-recognized or poorly defined. In the 1996 CEAP, primary etiologic problems were described as “neither congenital nor having any identifiable cause.” In the 2004 CEAP, there was no change in the description but E_n was added to subcategorize those with “no venous cause identified.” A more refined definition of primary etiology is that primary disease is a degenerative process of the venous valve and/or venous wall, leading to valve and/or vein wall weakness and dilatation that results in pathologic reflux, demonstrated by imaging. These findings are devoid of scarring or vein wall thickening typical for post-thrombotic syndrome. In the revised CEAP classification, there should be no other factors that would classify the process as congenital or secondary.

Subcategorization of the E_s classification. The limitation of the 2004 CEAP E_s classification is that there is no clear-cut description of the two different etiologies for secondary venous disease. There are many examples of intravenous pathology and extravenous pathology, both of which can lead to similar clinical venous signs and symptoms. Owing to an increased understanding of the impact of these secondary causes and the necessity for treatment options addressing different components, the need to subcategorize E_s is now more important. E_s is subcategorized to recognize intravenous secondary causes of venous disease (E_{si}), which is defined as any intravenous condition causing venous wall and/or valve damage, resulting from conditions such as DVT, traumatic arteriovenous fistulas, primary intravenous sarcoma, or other luminal change internal to the vein; and extravenous secondary causes (E_{se}), in which case there is no venous wall or valve damage, yet symptoms are present owing to a condition affecting venous hemodynamics either locally or systemically, such as central venous hypertension (eg, obesity, congestive heart failure, nutcracker syndrome, and pelvic and venous congestion), extrinsic compression due to mass effect (eg, extravenous tumor and local fibrosis, such as retroperitoneal fibrosis), or muscle pump dysfunction owing to motor disorders (eg, paraplegia, arthritis, chronic immobility, frozen ankle, or severe sedentary state).^{46,47}

Combinations of etiologic states can coexist. At times, both primary and secondary and intravenous and extravenous etiologic states can be present. When there are combinations of etiologic states, multiple subscript notations may be necessary. For example, primary varicose veins (superficial reflux without prior acute venous thrombosis) can be present with prior DVT. Because the varicose veins are primary and the DVT is secondary, the description for this etiology would be E_{psi} to describe the clinical picture from an etiologic perspective. Another example would be nonthrombotic iliac vein compression (May-Thurner syndrome), where the etiology may be associated with pure extrinsic compression (nonthrombotic iliac vein

lesions), but may also be associated with intraluminal obstructive findings secondary to extrinsic compression; the description for this etiology would be E_{sie} .

Refinement of the definition of E_c . Based on the 1996 and 2004 CEAP versions, the E_c , or congenital category, currently refers to the congenital abnormality that may be apparent at birth or can be recognized later. This is a limited definition and is made more complete by expanding to include the following: the congenital category now includes conditions present at birth, but not limited to venous agenesis, venous malformation (such as Klippel-Trenaunay syndrome), and arteriovenous malformation that can result in signs and symptoms of venous disease. Each of these presentations may or may not be present at birth, but may also manifest later in life.

Refinement of the definition of E_n . The descriptor E_n was added to CEAP in 2004. It refers to no venous abnormality identified. This designation can be confusing, because the definition overlaps with the E_p subclass for primary or idiopathic etiology, which also included an undetermined cause. To clarify, the E_n descriptor should be present when no other venous etiology (E_p , E_{si} , or E_{se} , E_c) is found, yet there are clinical signs and symptoms that can be consistent with those typically associated with venous disease. Basically, this is a category of exclusion.^{48,49}

REVISIONS TO THE ANATOMIC (A) CLASSIFICATION

As with the 2004 CEAP, the anatomic site(s) of the venous disease should be described as superficial (A_s), deep (A_D), or perforating (A_p) vein(s). One, two, or three systems may be involved in any combination. The limb being reported with CEAP should be identified (right[_R] and left[_L]). For reports requiring greater detail, the specific anatomic involvement of the superficial, deep, and perforating veins should be localized by use of the anatomic segments but documented under the pathophysiologic P class corresponding with that vein segment as per 1996 and 2004 CEAP. The revised specific A designations are presented in Table V. Approved revisions to the A designation include the following.

Use of anatomic abbreviations instead of numbers. The numbering classification for vein segments in advanced CEAP anatomic was believed to be too difficult to effectively use; they are difficult to recall, having no systematic rationale or other association, so using standard abbreviations derived from anatomic terms are easier to interpret and remember.⁵⁰ Additional abbreviations also allow the expansion of anatomic locations not previously specified. To maintain compatibility with prior CEAP documents, the new abbreviations should be linked electronically to the previous system of segment numbers. Although the detailed elaboration of venous disease in this form may seem unnecessarily

Table V. The 2020 revision of CEAP: Summary of anatomic (A) classification

A class		Description	
A _s	Superficial		
	<i>Old</i>	<i>New^a</i>	<i>Description</i>
	1.	Tel	Telangiectasia
	1.	Ret	Reticular veins
	2.	GSVa	Great saphenous vein above knee
	3.	GSVb	Great saphenous vein below knee
	4.	SSV	Small saphenous vein
		AASV	Anterior accessory saphenous vein
	5.	NSV	Nonsaphenous vein
A _d	Deep		
	<i>Old</i>	<i>New^a</i>	<i>Description</i>
	6.	IVC	Inferior vena cava
	7.	CIV	Common iliac vein
	8.	IIV	Internal iliac vein
	9.	EIV	External iliac vein
	10.	PELV	Pelvic veins
	11.	CFV	Common femoral vein
	12.	DFV	Deep femoral vein
	13.	FV	Femoral vein
	14.	POPV	Popliteal vein
	15.	TIBV	Crural (tibial) vein
	15.	PRV	Peroneal vein
	15.	ATV	Anterior tibial vein
	15.	PTV	Posterior tibial vein
	16.	MUSV	Muscular veins
	16.	GAV	Gastrocnemius vein
16.	SOV	Soleal vein	
A _p	Perforator		
	<i>Old</i>	<i>New^a</i>	<i>Description</i>
	17.	TPV	Thigh perforator vein
	18.	CPV	Calf perforator vein
A _n	No venous anatomic location identified		

^aNew specific anatomic location(s) to be reported under each P (pathophysiologic) class to identify anatomic location(s) corresponding to P class.

complex, it provides universally understandable descriptions, which may be essential to investigators in the field. For standardized reporting in scientific journals, a more precise anatomic grouping of those with the same types of disease allows better comparative analysis and enables the outcomes of treatments to be assessed more accurately. Furthermore, reports that use more precise anatomic CEAP can be compared with each another with much greater certainty.

REVISIONS TO THE PATHOPHYSIOLOGIC (P) CLASSIFICATION

The current 2004 CEAP P pathophysiologic class has basic and advanced designations. The basic designation includes: r (reflux), o (obstruction), r,o

(reflux and obstruction), and n (no venous pathophysiology). The advanced CEAP is the same with the addition of any (one or more) of named specific A anatomic venous segments. The newly revised specific P designation are presented in Table VI. Approved revisions to the P designation include the following.

Continue basic P classifications and use new A abbreviations for advanced anatomic segments. Although it is recognized that use of the anatomic notations for each P category increases the complexity of CEAP, the recommendation of the task force was to continue current format outlined in 2004 CEAP. With the revision made in abbreviations for anatomic locations, those using advanced CEAP should replace prior numerical subscripts with new anatomic abbreviations.

Table VI. The 2020 revision of CEAP: Summary of patho-physiologic (P) classification

P class	Description
P _r	Reflux
P _o	Obstruction
P _{r,o}	Reflux and obstruction
P _n	No pathophysiology identified
**Advanced New abbreviations for specific A anatomic location(s) to be reported under each P Pathophysiologic class to identify anatomic location(s) corresponding to P class.	

Continue the P_n classification for no venous pathophysiology identified. In some patients, there may be no underlying venous pathology such as reflux and/or obstruction, but stigmata of CVD exist nonetheless. Hemodynamic changes can take place in the venous system with or without the presence of valvular incompetence or reflux, leading to skin changes and ulceration. The task force recommended continuation of the P_n classification.

OTHER PROPOSED REVISIONS

The four working groups have proposed several revisions that have not been approved by the task force at this time. The main reason they did not gain approval is the lack of evidence supporting these revisions and the concern that these revisions will increase variability in reporting or complicate the clinical use of the CEAP classification.

The following revisions were proposed by the C working group, but were not approved by the task force: subcategorization of the C₀ class to include those with venous symptoms, no signs, and no reflux or obstruction and those with venous symptoms, no signs, and the presence of reflux or obstruction; subcategorization of the C₁ class to separately designate telangiectasias and reticular veins; creation of a single class (C₅) for healed, active, or recurrent ulcers, effectively collapsing categories C₅ and C₆, and eliminating the C₆ class; and changing numerical subscripts to alphabetical subscripts (eg, C_{4a} would now be C_{4.1}).

Although these proposed changes reflected some of the prior criticisms of clinical C class, the task force felt that these changes would unnecessarily increase the complexity of the C classification, would be too disruptive to current familiarity, and would significantly affect compatibility with evidence-based prior versions of CEAP.

Other proposed revisions proposed by the E working group included other veins not previously accounted for anatomically in the 2004 CEAP document. These anatomic locations included the renal vein, ovarian vein, uterine vein, lumbar vein, intersaphenous vein, gluteal vein, and pudendal vein. These additions would

allow for the inclusion of parts of the venous system not previously addressed in our understanding of the evolution of lower limb venous disorders and sites of treatment, but that are now part of modern venous management in lower extremities. However, this proposal was not approved by the task force owing to a lack of evidence supporting the connection of these anatomic locations and lower extremity venous disorders.

Recognizing that there is no allowance or designation in the 2004 CEAP document for other contributing patient-level factors which may augment or worsen venous disease severity, the P working group proposed including designations for morbid obesity (body mass index >30 kg/m²), symptomatic conditions leading to right heart failure, and conditions leading to impaired calf muscle pump. These contributing factors may stand alone as pathophysiologic mechanisms or in conjunction with valvular incompetence or reflux. However, at this time the task force did not identify enough evidence to clearly support adding these factors to P classification of CEAP, and from a practicality perspective, these would increase the complexity of P.

CONCLUSIONS

Since its initial development, the CEAP classification has been and continues to be an important contributor to progress in the field of CVD. It has become a universally accepted standard in research and reporting. Although the stability of classification is essential for maintaining scientific and clinical advancement, continuously accumulated evidence and knowledge require revisiting the classification and its definitions and revising them when necessary. This 2020 CEAP revision is a result of a rigorous process of evidence analysis. Although several proposed changes were not included in the final version, proponents of these and other future potential revisions are encouraged to develop and publish supporting evidence. When such evidence is available, the AVF Task Force will revisit the CEAP classification system, making revisions as part of a continual process and maintaining the integrity of CEAP as the universally accepted classification system and reporting standard for CVD.

AUTHOR CONTRIBUTIONS

Conception and design: FL, MaP

Analysis and interpretation: FL, MaP, MM, MD, EM, HW, RB, JB, PC, MDM, AG, NL, WM, JR, FS, CS, JU, TU, AvR, BE, PG, RK, PL, GM, FP, MiP, TW

Data collection: Not applicable

Writing the article: Not applicable

Critical revision of the article: FL, MaP, MM, MD, EM, HW, RB, JB, PC, MDM, AG, NL, WM, JR, FS, CS, JU, TU, AvR, BE, PG, RK, PL, GM, FP, MiP, TW

Final approval of the article: FL, MaP, MM, MD, EM, HW, RB, JB, PC, MDM, AG, NL, WM, JR, FS, CS, JU, TU, AvR, BE, PG, RK, PL, GM, FP, MiP, TW
Statistical analysis: Not applicable
Obtained funding: Not applicable
Overall responsibility: FL

REFERENCES

1. Beebe HG, Bergan JJ, Bergqvist D, Eklöf B, Eriksson I, Goldman MP, et al. Classification and grading of chronic venous disease in the lower limbs: a consensus statement. *Vasc Surg* 1996;30:5-11.
2. Eklöf B, Rutherford RB, Bergan JJ, Carpentier PH, Gloviczki P, Kistner RL, et al. American Venous Forum International Ad Hoc Committee for Revision of the CEAP Classification. Revision of the CEAP classification for chronic venous disorders: consensus statement. *J Vasc Surg* 2004;40:1248-52.
3. Widmer LK. Classification of venous disorders. In: Basle, editor. *Peripheral venous disorders*. Bern, Switzerland: Hans Huber Publishers; 1978.
4. Partsch H. "Betterable" and "nonbetterable" chronic venous insufficiency. A proposal for a practice-oriented classification. *Vasa* 1980;9:165-7.
5. Hach W. Neue Aspekte zum Spontanverlauf einer Stammvarikose derv. Saphena magna. *Phlebol Proktol* 1988;17:79-82.
6. Sytchev GG. Classification of chronic venous disorders of lower extremities and pelvis. *Int Angiol* 1985;4:203-6.
7. Pierchalla P, Tronnier H. Diagnosis and classification of venous insufficiency of the leg. *Dtsch Med Wochenschr* 1985;110:1700-2.
8. Olivier C, Merlen JF. *Précis des maladies des vaisseaux*. Paris: Masson Editions; 1983.
9. Porter J, Rutherford R, Clagett GP, Cranley J, O'Donnell T, Raju S, et al. Reporting standards in venous disease. *J Vasc Surg* 1988;8:172-81.
10. Antignani PL. Classification of chronic venous insufficiency: a review. *Angiology* 2001;52(Suppl 1):S17-26.
11. Allegra C, Antignani PL, Bergan JJ, Carpentier PH, Coleridge-Smith P, Cornu-Thénard A, et al. The "C" of CEAP: suggested definitions and refinements: an International Union of Phlebology conference of experts. *J Vasc Surg* 2003;37:129-31.
12. Eklöf B, Perrin M, Delis KT, Rutherford RB, Gloviczki P. American Venous Forum; European Venous Forum; International Union of Phlebology; American College of Phlebology; International Union of Angiology. Updated terminology of chronic venous disorders: the VEIN-TERM transatlantic interdisciplinary consensus document. *J Vasc Surg* 2009;49:498-501.
13. Perrin M, Eklöf B, Maleti O. The vein glossary. *J Vasc Surg Venous Lymphat Disord* 2018;6:e11-217.
14. Lurie F, Comerota A, Eklöf B, Kistner RL, Labropoulos N, Lohr J, et al. Multicenter assessment of venous reflux by duplex ultrasound. *J Vasc Surg* 2012;55:437-45.
15. Lee BB, Nicolaidis AN, Myers K, Meissner M, Kalodiki E, Allegra C, et al. Venous hemodynamic changes in lower limb venous disease: the UIP consensus according to scientific evidence. *Int Angiol* 2016;35:236-352.
16. Rabe E, Pannier F. Clinical, aetiological, anatomical, and pathological classification (CEAP): gold standard and limits. *Phlebology* 2012;27(Suppl 1):114-8.
17. Guyatt GH, Kirshner B, Jaeschke R. Measuring health status: what are the necessary measurement properties? *J Clin Epidemiol* 1992;45:1341-5.
18. Kirshner B, Guyatt G. A methodological framework for assessing health indices. *J Chron Dis* 1985;38:27-36.
19. Rutherford RB, Padberg FT, Comerota AJ, Kistner RL, Meissner MH, Moneta GL. Venous severity scoring: an adjunct to venous outcome assessment. *J Vasc Surg* 2000;31:1307-12.
20. Vasquez MA, Rabe E, McLafferty RB, Shortell C, Marston WA, Gillespie D, et al. Revision of the venous clinical severity score: venous outcomes consensus statement: special communication of the American Venous Forum Ad Hoc Outcomes Working Group. *J Vasc Surg* 2010;52:1387-96.
21. Catarinella FS, Nieman FH, Whittens CH. An overview of the most commonly used venous quality of life and clinical outcome measures. *J Vasc Surg Venous Lymph* 2015;3:333-40.
22. Cornu-Thenard A, Uhl JF, Carpentier PH. Do we need a better classification than CEAP? *Acta Chir Belg* 2004;104:276-82.
23. Carpentier PH, Cornu-Thenard A, Uhl JF, Partsch H, Antignani PL, Societe Francaise de Medecine V, et al. Appraisal of the information content of the C classes of CEAP clinical classification of chronic venous disorders: a multicenter evaluation of 872 patients. *J Vasc Surg* 2003;37:827-33.
24. Davies HO, Popplewell M, Singhal R, Smith N, Bradbury AW. Obesity and lower limb venous disease - the epidemic of phlebeity. *Phlebology* 2017;32:227-33.
25. Musil D, Kaletova M, Herman J. Age, body mass index and severity of primary chronic venous disease. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub* 2011;155:367-71.
26. van Rij AM, De Alwis CS, Jiang P, Christie RA, Hill GB, Dutton SJ, et al. Obesity and impaired venous function. *Eur J Vasc Endovasc Surg* 2008;35:739-44.
27. Vlajinac HD, Marinkovic JM, Maksimovic MZ, Matic PA, Radak DJ. Body mass index and primary chronic venous disease—a cross-sectional study. *Eur J Vasc Endovasc Surg* 2013;45:293-8.
28. Willenberg T, Schumacher A, Anamm-Vesti B, Jacomella V, Thalhammer C, Diehm N, et al. Impact of obesity on venous hemodynamics of the lower limbs. *J Vasc Surg* 2010;52:664-8.
29. Abbas M, Hamilton M, Yahya M, Mwipatayi P, Sieunarine K. Pulsating varicose veins!! The diagnosis lies in the heart. *ANZ J Surg* 2006;76:264-6.
30. Brickner PW, Scudder WT, Weinrib M. Pulsating varicose veins in functional tricuspid insufficiency. Case report and venous pressure tracing. *Circulation* 1962;25:126-9.
31. Krahenbuhl B, Restellini A, Frangos A. Peripheral venous pulsatility detected by Doppler method for diagnosis of right heart failure. *Cardiology* 1984;71:173-6.
32. Back TL, Padberg FT Jr, Araki CT, Thompson PN, Hobson RW 2nd. Limited range of motion is a significant factor in venous ulceration. *J Vasc Surg* 1995;22:519-23.
33. Williams KJ, Ayekoloye O, Moore HM, Davies AH. The calf muscle pump revisited. *J Vasc Surg Venous Lymphat Disord* 2014;2:329-34.
34. Uhl JF, Chahim M, Allaert FA. Static foot disorders: a major risk factor for chronic venous disease? *Phlebology* 2012;27:13-8.
35. Yang D, Vandongen YK, Stacey MC. Changes in calf muscle function in chronic venous disease. *Cardiovasc Surg* 1999;7:451-6.
36. Yang D, Vandongen YK, Stacey MC. Effect of exercise on calf muscle pump function in patients with chronic venous disease. *Br J Surg* 1999;86:338-41.
37. Crisostomo RS, Candeias MS, Armada-da-Siva PA. The use of ultrasound in the evaluation of the efficacy of calf muscle pump function in primary chronic venous disease. *Phlebology* 2014;29:247-56.
38. Fukuoka M, Sugimoto T, Okita Y. Prospective evaluation of chronic venous insufficiency based on foot venous pressure measurements and air plethysmography findings. *J Vasc Surg* 2003;38:804-11.

39. Christopoulos D. Air-plethysmography in the quantification of the clinical severity of chronic venous disease. *Int Angiol* 2008;27:86.
40. Uhl JF, Gillot C. Anatomy of the veno-muscular pump of the lower limb. *Phlebology* 2015;30:180-93.
41. Saggini R, Bellomo RG, Iodice P, Lessiani G. Venous insufficiency and foot dysmorphism: effectiveness of visco-elastic rehabilitation systems on veno-muscle system of the foot and of the calf. *Int J Immunopathol Pharmacol* 2009;22:1-8.
42. Perrin MR, Guex JJ, Ruckley CV, dePalma RG, Royle JP, Eklof B, et al. Recurrent varices after surgery (REVAS), a consensus document. REVAS group. *Cardiovasc Surg* 2000;8:233-45.
43. Perrin MR, Labropoulos N, Leon LR Jr. Presentation of the patient with recurrent varices after surgery (REVAS). *J Vasc Surg* 2006;43:327-34; discussion: 334.
44. Uhl JF, Cornu-Thenard A, Satger B, Carpentier PH. Clinical analysis of the corona phlebectatica. *J Vasc Surg* 2012;55:150-3.
45. Robertson L, Lee AJ, Gallagher K, Carmichael SJ, Evans CJ, McKinstry BH, et al. Risk factors for chronic ulceration in patients with varicose veins: a case control study. *J Vasc Surg* 2009;49:1490-8.
46. Raju S, Neglen P. High prevalence of nonthrombotic iliac vein lesions in chronic venous disease: a permissive role in pathogeniticty. *J Vasc Surg* 2006;44:136-43.
47. Neglen P, Berry MA, Raju S. Endovascular surgery in the treatment of chronic primary and post- thrombotic iliac vein obstruction. *Eur J Vasc Endovasc Surg* 2000;20:560-71.
48. Kataoka H. Clinical characteristics of lower-extremity edema in stage A cardiovascular disease status defined by the ACC/AHA 2001 Chronic Heart Failure Guidelines. *Clin Cardiol* 2013;36:555-9.
49. Ely JW, Osheroff JA, Chambliss ML, Ebell MH. Approach to leg edema of unclear etiology. *J Am Board Fam Med* 2006;19:148-60.
50. Cavezzi A, Labropoulos N, Partsch H, Ricci S, Caggiati A, Myers K, et al. Duplex ultrasound investigation of the veins in chronic venous disease of the lower limbs—UIP consensus document. Part II. Anatomy. *Eur J Vasc Endovasc Surg* 2006;31:288-99.

Submitted Oct 10, 2019; accepted Dec 22, 2019.