Reporting standards in venous disease: An update

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At the request of the Ad Hoc Committee on Reporting Standards of the Joint Council of the Society for Vascular Surgery and the North American Chapter of the International Society for Cardiovascular Surgery, this report updates and modifies "Reporting standards in venous disease" (J VASC SURG 1988;8:172-81). As in the initial document, reporting standards for publications dealing with (1) acute lower extremity venous thrombosis, (2) chronic lower extremity venous insufficiency, (3) upper extremity venous thrombosis, and (4) pulmonary embolism are presented. Numeric grading schemes for disease severity, risk factors, and outcome criteria present in the original document have been updated to reflect increased knowledge of venous disease and advances in diagnostic techniques. Certain recommendations of necessity remain arbitrary. These standards are offered as guidelines whose observance will in our opinion improve the clarity and precision of communications in the field of venous disorders. (J VASC SURG 1995;21: 635-45.)

ACUTE LOWER EXTREMITY VENOUS THROMBOSIS

Risk factors and predisposing conditions

The following is an attempt to quantify risk for acute lower extremity deep vein thrombosis (DVT). This quantification scheme, although unproven, may allow more precise patient characterization in reports evaluating DVT prophylaxis and treatment.

Prior history of DVT. A prior episode of lower extremity DVT is the greatest risk factor for a subsequent episode of DVT.[1] A history of an abnormal phlebography or objective vascular laboratory examination result (phleborheography, impedance plethysmography, B-mode ultrasound imaging, or ultrasonic duplex scanning) is sufficient to establish a prior episode of proximal lower extremity DVT. A history of isolated calf vein thrombosis requires a previous abnormal phlebogram or a definitive duplex ultrasound examination. A patient with clinical post-thrombotic syndrome and no history of prior DVT should be classified as grade 1 (suspected).

Assign grade

0 = none
1 = suspected
2 = proven
3 = multiple

Immobilization. Immobilization from illness or injury is a DVT risk factor.[2] Duration and cause of the immobilization influence the risk of DVT.

Assign grade

0 = none
1 = 1 to 3 days
2 = >3 days
3 = immobilization caused by acute paraplegia[3]

Postoperative state. Duration of operation and type of anesthesia appear related to development of postoperative DVT.[4] Patients undergoing pelvic, hip, and lower extremity orthopedic procedures and intercranial neurosurgical operations are at particular risk.

Assign grade

0 = local anesthesia
1 = <45 minutes, regional or general
2 = >45 minutes, regional or general
3 = extensive major (>3 hours) and/or pelvic operation[5]

Age. Increasing age increases the risk of DVT.[6]

Assign grade

0 = <40 years

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†See appendix.

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1 = 40 to 70 years
2 = >70 years

Malignancy. The presence of a malignancy and its extent may influence the development of DVT.[7]
Assign grade
0 = none
1 = nonrecurrence or local recurrence only
2 = extensive regional tumor
3 = metastatic

Tissue type of malignancy. Tissue type of associated malignancy may also influence development of DVT.
Assign grade
0 = other than adenocarcinoma
1 = adenocarcinoma (especially mucinous adenocarcinoma), malignant glioma of the brain

Cardiac disease. Severity of cardiac disability appears to increase the risk for development of DVT.[8]
Assign grade
0 = New York Heart Association (NYHA) class 1
1 = NYHA class 2
2 = NYHA class 3
3 = NYHA class 4

Limb trauma. Bony and extensive soft tissue, lower extremity, or pelvic injury increases the likelihood of lower extremity DVT.[9]
Assign grade
0 = none
1 = soft tissue injury including bruise, contusion, and sprain
2 = fracture of tibia and/or fibula
3 = fracture of femur
4 = fracture of hip or pelvis

Prethrombotic state. Several coagulation abnormalities predispose to *abnormal clotting,* including venous thrombosis. These include but are not limited to antithrombin III deficiency; protein C or protein S resistance or deficiency; myeloproliferative disorders, especially thrombocytosis; plasma hyperviscosity states; and lupus anticoagulant and/or anticardiolipin antibodies.[10-14]
Assign grade
0 = none suspected
1 = suspected
2 = proven, treated
3 = proven, untreated

Hormonal therapy. Prolonged exogenous *ethinyl estradiol in a dosage in excess of 50 μg daily* is associated with an increased risk of venous thrombosis.[15,16] Neither low-dose estrogen therapy nor any other hormonal therapy has such a clear relationship to DVT, although others are suspected.
Assign grade
0 = no
1 = yes

Pregnancy and postpartum state. Venous thrombosis is five times more likely in a pregnant or postpuerperal woman compared with a nonpregnant, nonpuerperal woman not taking oral contraceptives.[17]
Assign grade
0 = absent
1 = present

Obesity. Extreme obesity may be a mild independent risk factor in DVT.[18]
Assign grade
0 = normal to 175% ideal body weight
1 = >175% ideal body weight

Summary. The factors listed represent our impression of the risk factors relevant to the development of DVT. With the relative risk assessment weighting scheme described (maximum possible score = 28), future studies of DVT prophylaxis and therapy may be stratified by the DVT risk factor score to ensure equality of comparison populations.

Clinical presentation

Extent of thrombus and site involvement. The anatomic sites of involvement must be detailed for clinical reports of DVT. Involvement of the following deep venous segments should be specified: tibial-soleal veins, popliteal vein, common femoral or superficial femoral vein, iliac vein, and vena cava. Specified superficial sites include greater saphenous vein and its branches; lesser saphenous vein and its branches; and unnamed cutaneous veins, in which case the location of the veins should be specified. Each of the six deep and two superficial venous segments should be graded as follows and tabulated individually.
Assign grade
0 = patent
1 = subsegmental, nonocclusive thrombus
2 = subsegmental, occlusive thrombus
3 = occlusive thrombus throughout length of segment

The maximal thrombotic score for a single limb is 24. Either phlebography or duplex scanning may be used to calculate the thrombotic score.

Clinical description

Acute venous thrombosis should be described as superficial, deep, or combined.

Superficial venous thrombosis. Site and extent of involvement should be specified. Tenderness, erythema, induration, or suppuration should be noted. It is essential that the patency of the deep veins be documented phlebographically or by duplex scanning in reports dealing with superficial venous thrombosis because the coexistence of DVT may confuse the clinical presentation and response to therapy of superficial venous thrombosis. In particular, superficial venous thrombosis involving the proximal greater saphenous vein is often associated with extension into the common femoral vein.[19]

DVT. The site and extent of involvement should be specified. The presence and location of pain and the extent of swelling should be noted in case reports.

Definitions

The following terms are used often enough to require uniform definition.

Phlegmasia alba dolens. This term indicates marked swelling of the lower extremity without associated cyanosis. It refers to a characteristic clinical picture and does not denote the existence of DVT at a precise site.

Phlegmasia cerulea dolens. This term indicates massive limb swelling and cyanosis. Venous thrombosis is more extensive than in phlegmasia alba dolens, and the extremity is painful. It may be associated with arterial insufficiency, compartmental compression syndrome, or venous gangrene.[20]

Venous gangrene. This refers to full-thickness skin necrosis resulting from DVT. Blebs or blisters represent partial thickness necrosis and as such may be regarded as early venous gangrene. Compartment syndrome(s) may coexist.

Diagnostic testing

Diagnostic tests for acute DVT are classified as physiologic, anatomic, or combined.

Physiologic tests. Recognized physiologic tests for the diagnosis of DVT include (1) continuous-wave Doppler examination with the use of flow interruption or augmentation maneuvers and (2) plethysmography—impedance/air/volume and strain-gauge. These tests are valid only when performed by experienced examiners and usually only under the following conditions: (1) absence of prior history of deep vein thrombosis of the same extremity and (2) absence of extrinsic venous compression as may occur with pregnancy or hematoma.[21]

Anatomic tests. Contrast phlebography and ultrasound B-mode imaging combined with color-flow imaging are acceptable techniques for determining the anatomic extent of acute DVT. Uptake of iodine 125–labeled fibrinogen, because of poor specificity, is not.[22]

When phlebography is used the technique should be described in detail, specifically the use of tourniquets, head or foot table elevation, and volume and type of contrast media. Positive phlebography results must be described as directly or indirectly positive. A directly positive test result is one in which thrombus is outlined by contrast media. Nonfilling of deep vein segments without visualization of thrombus is an indirect sign of DVT.

B-mode image positivity requires visualization of the thrombus or absence of normal venous wall coaptation with external pressure. Color-flow scanning should be used, when available, to confirm absence of venous flow or the presence of nonoccluding thrombis.

Combined physiologic and anatomic evaluation. Duplex scanning combines ultrasound image (anatomic) and Doppler flow (physiologic) data. The criteria for positivity of this test must be specified by the author. Pulsed-wave Doppler flow data are considered positive for DVT in the absence of a normal intraluminal venous flow signal and image evidence of thrombosis in the insonated segment as detailed above.

Treatment

The choices of treatment of DVT include nonoperative supportive therapy, drug treatment, and thrombectomy.

Nonoperative supportive treatment. Reports should specify use and duration of bed rest, leg elevation, and compressive therapy. If the latter is used, the type should be specified: such as bandages, inelastic devices, external pumps, elastic hosiery. When describing elastic hosiery the length of the garment (below-knee, above-knee, or waist-high), whether the garment is of gradient construction, and the manufacturer's stated amount of compression measured in torr should be specified.

Drug therapy. Drug therapy for DVT includes anticoagulants, thrombolytics, and other less recognized medications.

Anticoagulation. If heparin sodium is used, the rate of administration, dosage, and duration of therapy should be specified. The type and frequency of monitoring to determine anticoagulant effect must be reported. The usual tests for monitoring heparin anticoagulant activity include partial thromboplastin time (PTT) and activated clotting time (ACT). Whatever test is used, it should be related to the PTT

to allow readers a normative reference. If the route of drug administration is other than by continuous intravenous administration, the timing of blood sampling for monitoring tests in relation to prior doses of heparin must be specified. If low molecular weight heparin is used, the molecular weight and source of the heparin must be specified, as well as the dosing schedule.

When a patient is switched from heparin to warfarin, the timing and duration of the drug overlap should be described. The dose, frequency, type of monitoring, and duration of therapy for warfarin anticoagulation must be specified. The intensity of warfarin anticoagulation ideally is expressed in terms of the international normalized ratio (INR).[23] When this is not possible the intensity should be expressed as a prothrombin ratio (e.g., 1½ to 2 × control). When the intensity of warfarin anticoagulation is expressed as a prothrombin time ratio, the test reagent used in performing the prothrombin time test should be specified.

Thrombolytic therapy. The drug, dosage, route of administration, and laboratory method of monitoring thrombolytic activity must be detailed. The frequency and results of sequential contrast phlebograms or duplex scans must be specified. A perfect result requires complete phlebography- or duplex scanning–determined patency and vascular laboratory determination of normal valvular competency as determined by duplex scanning assessment of valve closure times.[24]

Results of lytic therapy should be reported as the percentage of limbs experiencing *total clot resolution with overall preservation of valve function.* Serial objective patient outcome data regarding patency and valve function should be presented in a life-table format. Patency may be evaluated by serial phlebography or duplex scanning. Assessment of *segmental* valvular competence should be by duplex scanning–derived valve closure times. *Overall* reflux in the limb can be evaluated with direct ambulatory venous pressure measurements, venous recovery times (VRT), or air plethysmography.[25] Because reflux may develop late,[26] separate life-tables should be presented for patency and valvular competence.

Incomplete clot resolution should be reported as failure of thrombolytic therapy. Reporting outcome of thrombolytic therapy as a percentage of thrombus resolution is not acceptable. On occasion, restoration of patency in large veins, such as the vena cava or iliac veins, may be the goal of lytic therapy, and in these select cases restoration of valvular competency is irrelevant. Restoration of patency of infrainguinal veins without valvular competence should be noted.

Other drug therapy. The use of drugs other than heparin sodium, heparinoids, warfarin, or thrombolytic agents in the treatment of DVT should include an explanation why the particular agent was selected. Route of administration, dosage, duration of therapy, and the method of monitoring drug effect must also be specified.

Complications of drug therapy. Complications associated with drug therapy must be detailed. General complications and complications specific to individual agents should be described and their severity indicated as mild, moderate, or severe. Deaths directly related to the agent, in-hospital deaths, and deaths within 30 days of hospital discharge must be specifically noted.

Surgical therapy. Venous thrombectomy continues to be performed occasionally for lower extremity DVT.[27] Reports of such procedures should provide a detailed description of the surgical technique. The same reporting requirements pertain to venous thrombectomy as to thrombolytic therapy (see above). The same method of evaluation should be used for all patients included in an individual report.

Outcome of acute DVT

Pulmonary embolism as a complication of DVT should be specifically noted, with attention to the details listed in the section, "Pulmonary Embolism." The time course for resolution of local signs, such as edema and cyanosis, should be noted if relevant. The presence of recurrent episode(s) of DVT should be specifically noted together with the objective tests used to establish the diagnosis. If relevant, subsequent occurrence of the postthrombotic syndrome should be noted with attention to the details listed in the later section, "Chronic Venous Disease." Some vascular laboratory tests may not reliably establish the presence of a new episode of DVT because patients may have permanent vascular laboratory abnormalities after the first episode.

Venous thrombosis prophylaxis

The term *prophylaxis* should be used only in relation to patients who have not sustained a recent DVT. Many reports describe the outcome of various prophylactic measures for patients defined as being at high risk for DVT. Such reports should, as described in the first section, "Risk Factors," calculate a risk factor index for each patient, as well as an average for the study and control groups. In addition, the

Table I. Classification of chronic lower extremity venous disease*

C	Clinical signs (grade$_{0-6}$), supplemented by ($_A$) for asymptomatic and ($_S$) for symptomatic presentation
E	Etiologic Classification (**C**ongenital, **P**rimary, **S**econdary)
A	Anatomic Distribution (**S**uperficial, **D**eep, or **P**erforator, alone or in combination)
P	Pathophysiologic Dysfunction (**R**eflux or **O**bstruction, alone or in combination)

*See text for details.

following should be included in reports on the efficacy of venous thrombosis prophylaxis.

Mechanical methods of prophylaxis. Leg exercises should be described as active or passive, and type, frequency, and duration should be specified. For compression stockings, design (whether gradient or not), length, compression measured in torr, and the frequency and duration of use should be specified. Studies with pneumatic leg compression should detail the number of individual compression chambers, the pressure and time sequence of the pumping pattern, and the frequency and duration of treatment.

Drug prophylaxis. The agents, route of administration, dosage, duration, and method of monitoring drug prophylaxis should be specified. If prophylaxis is directed toward prevention of postoperative thrombosis, the timing of the initiation of prophylaxis in relation to operation and the type and duration of operation should be stated.

CHRONIC VENOUS DISEASE

Chronic venous disease is defined as an abnormally functioning venous system caused by venous valvular incompetence with or without associated venous outflow obstruction, which may affect the superficial venous system, the deep venous system, or both. Venous dysfunction may result from either a congenital or an acquired disorder.

The term *postthrombotic* may be used if the patient has experienced an objectively documented prior episode of DVT. The term *postphlebitic syndrome* should not be used because this implies the presence of an inflammatory component that is infrequently confirmed. In the absence of a clear documentation of a prior episode of DVT, the condition should be termed *chronic venous disease* without additional suggestion of origin.

Physical examination—descriptive terms

Venous dilation. Mild chronic venous insufficiency (CVI) is signified by the occurrence of a submalleolar venous flare. Greater degrees of venous dilation are apparent by both observation and palpation. Telangiectases are defined as dilated intradermal venules less than 1 mm in size. Reticular veins are defined as dilated, nonpalable, subdermal veins 4 mm in size or less. Varicose veins are defined as dilated palable subcutaneous veins generally larger than 4 mm.

Edema. The presence of edema indicates more functionally advanced venous disease than the presence of venous dilation alone. In individual case reports and when otherwise applicable, the location and extent of edema should be noted and objectively documented with circumferential limb measurements.

Skin pigmentation. Pigmentation changes and other cutaneous manifestations of chronic venous disease (venous eczema, lipodermatosclerosis) are important signs of severe chronic venous disease. They should be described along with a subjective assessment of severity.

Venous ulceration. The location and measurements of any venous ulcer should be described, and the presence or absence of granulation tissue should be noted. The presence of healed venous ulceration manifested by cutaneous scarring should be noted.

Classification of chronic venous disease (C,E,A,P)

Limbs with chronic venous disease should be classified according to clinical signs (C), cause (E), anatomic distribution (A), and pathophysiologic condition (P). The classification system detailed below and summarized in Table I was developed in 1994 by an international consensus conference on chronic venous disease held under the auspices of the American Venous Forum. It replaces clinical classes 0 to 3 outlined in the 1988 version of Reporting Standards in Venous Disease. This updated method of classifying chronic venous disease is designed to provide the additional details necessary to accurately compare limbs in medical and surgical treatment trials.

Clinical classification (C_{0-6}). Any limb with possible chronic venous disease is first placed into one of seven clinical classes (C_{0-6}) according to the objective signs of disease listed in Table II.

Limbs in higher categories have more severe signs of chronic venous disease and may have some or all

Table II. Clinical classification of chronic lower extremity venous disease

Class 0	No visible or palable signs of venous disease
Class 1	Telangiectases, reticular veins, malleolar flare
Class 2	Varicose veins
Class 3	Edema without skin changes
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

of the findings defining a less severe clinical category. Each limb is further characterized as asymptomatic (A), for example, $C_{0\text{-}6,A}$, or symptomatic (S), for example, $C_{0\text{-}6,S}$. Symptoms that may be associated with telangiectacic, reticular, or varicose veins include lower extremity aching, pain, and skin irritation. Therapy may alter the clinical category of chronic venous disease. Limbs should therefore be reclassified after any form of medical or surgical treatment.

Etiologic classification (E_C, E_P, or E_S). Venous dysfunction may be congenital, primary, or secondary. These categories are mutally exclusive. Congenital venous disorders are present at birth but may not be recognized until later. The method of diagnosis of congenital abnormalities must be described. Primary venous dysfunction is defined as venous dysfunction of unknown cause but not of congenital origin. Secondary venous dysfunction denotes an acquired condition resulting in chronic venous disease, for example, DVT. (Table III).

Anatomic classification ($A_{S,D,P}$). 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. For reports requiring greater detail, the involvement of the superficial, deep, and perforating veins may be localized by use of the anatomic segments listed in Table IV.

Pathophysiologic classification ($P_{R,O}$). Clinical signs or symptoms of chronic venous disease result from reflux (P_R), obstruction (P_O), or both ($P_{R,O}$) (Table V). Measurement by superficial venous cannulation of the foot venous pressure at rest in the upright position and the change in pressure on walking has historically represented the "gold standard" for the *overall* objective assessment of chronic venous disease.[25] It is now recommended that reports of patients with chronic venous disease be accompanied by sufficient objective measurements of venous hemodynamics and anatomy to document adequately the individual pathophysiologic changes, reflux, obstruction, or both, accompanying chronic venous disease. Phlebographic or vascular laboratory studies can objectively assess the presence of venous outflow obstruction (P_O), as well as the presence of venous reflux (P_R) in the superficial, communicating, and deep venous systems.

Ascending phlebography defines areas of obstruction, recanalization, and collateral vein formation. Descending phlebography demonstrates competency of venous valves by assessing the magnitude of contrast reflux. Comparisons of different reports of descending phlebography are facilitated by knowledge of the technical details of performance of the procedure. Accordingly, reports of descending phlebography should include details of cannulation; type, volume, and injection rate of contrast media; tilt angle of the table; and maximal timed descent of the contrast column in the deep venous system.

Assessment of maximal venous outflow (MVO) by one of various plethysmographic techniques provides objective noninvasive information on the presence and amount of venous *obstruction* (P_O). Sequential examinations are helpful in monitoring the development of venous collateral vessels or recanalization after DVT. MVO must be related to simultaneously determined venous capacitance. Pressure gradients (e.g., arm/ankle) at rest or after induced hyperemia may also permit the objective assessment of outflow obstruction.

Directional continuous wave-Doppler examination with proximal compression or Valsalva maneuver is a qualitative test for assessing *reflux* (P_R) in both the superficial and deep venous systems. Because the test is qualitative and uncontrolled, its use is discouraged in modern reports concerning chronic venous disease.

Venous refill time (VRT) provides a measure of *overall* venous reflux that can be used in the objective assessment of chronic venous disease. It can be obtained invasively with foot venous cannulation used in conjunction with ambulatory venous pressure or noninvasively with photoplethysmography. Normalization of a shortened VRT by application of a leg tourniquet compressing superficial veins indicates that significant venous disease is limited to the superficial venous system.

The venous filling index (VFI) as determined by

Table III. Etiologic classification of chronic lower extremity venous disease

Congenital (E_C)	The cause of the chronic venous disease has been present since birth
Primary (E_P)	Chronic venous disease of undetermined cause
Secondary (E_S)	Chronic venous disease with an associated known cause (postthrombotic, post-traumatic, other)

air plethysmography is another quantitative method of evaluating overall venous reflux.[28] Like VRT, the test can be performed with and without tourniquets in an effort to identify significant reflux limited to the superficial veins.

Because the severity of venous dysfunction is influenced by the anatomic location of the reflux or obstruction,[29] it may be desirable to report the anatomic segments involved with either reflux or obstruction with the venous segments outlined in Table IV. Currently, duplex ultrasonography performed with the patient in an upright position and with the limb examined in a nonweight-bearing position, in combination with proximal deflation of a venous occluding blood pressure cuff,[24] is the best-documented noninvasive method of quantifying reflux, by measuring reflux duration in specific axial superficial or deep venous segments. Duplex scanning is also suggested as a means of identifying reflux in individual communicating veins.

Surgical procedures

Superficial system insufficiency. Valvular competence of the deep veins has an obvious relationship both to the occurrence and recurrence of superficial varicosities. Thus reports of superficial venous ligation and stripping must describe communicating and deep venous valvular competence as assessed by appropriate phlebographic or vascular laboratory testing.

Deep venous obstruction. When various conduits are used to bypass areas of venous obstruction, a detailed description of type, extent, and configuration of the bypass conduit must be provided. Procedural reports, in addition to the patients' subjective assessment of benefit, should include vascular laboratory measurements of MVO obtained before operation and at least 6 months after operation to aid in objectively assessing surgical outcome. Patency of the bypass conduit should be assessed with either duplex scanning or phlebography and presented in a life-table format.

Table IV. Segmental localization of chronic lower extremity venous disease

Segment no.	
	Superficial veins (A_{S1-5})
1	Telangiectasias/reticular veins
	Greater (long) saphenous vein
2	*Above-knee*
3	*Below-knee*
4	Lesser (short) saphenous vein
5	Nonsaphenous
	Deep veins (A_{D6-16})
6	Inferior vena cava
	Iliac
7	*Common*
8	*Internal*
9	*External*
10	Pelvic: gonadal, broad ligament
	Femoral
11	*Common*
12	*Deep*
13	*Superficial*
14	Popliteal
15	Tibial (anterior, posterior or peroneal)
16	Muscular (gastrointestinal, soleal, other)
	Perforating veins ($A_{P17,18}$)
17	Thigh
18	Calf

Deep venous valvular insufficiency. The type of procedure used to correct this defect should be categorized as one of the following:

Valvuloplasty. Include a brief technical description of the procedure and the precise location and number of valves repaired.

Venous segmental transposition. Specify anastomotic connection, such as, saphenous to deep femoral vein.

Venous segmental transplantation. Specify donor and recipient sites, the length of the transplanted segment, and the number of valves contained in the transplanted segment.

Evaluation of operative results. All publications describing patients undergoing surgical repair of deep venous insufficiency or obstruction must include vascular laboratory measurements of lower extremity venous hemodynamics before and after operation to permit objective assessment of results. The tests must include, at a minimum, an overall evaluation of venous function with VRT or ambulatory venous pressure, and preferably both. MVO provides helpful additional information in patients undergoing operation for venous obstruction, whereas duplex assessment of valvular competence, including the operative repair, should be performed

Table V. Pathophysiologic classification of chronic lower extremity venous disease

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

in patients undergoing operation for valvular incompetence.

Authors may, at their discretion, report the use of new and/or nonstandardized vascular laboratory tests performed in an attempt to document objectively the functional results of deep vein reconstruction, but the report of such tests does not exempt authors from reporting the requisite standardized preoperative and postoperative vascular laboratory tests of venous function. All reports of deep vein repair should report at least 6 months of postoperative vascular laboratory follow-up and preferably 12 months of follow-up because progressive deterioration of initially satisfactory surgical results has occurred with some frequency. Reports must state whether the patient regularly used postoperative elastic compression hosiery. If so, the compression, measured in torr, must be included. Reports should also state whether the patient used intermittent leg elevation during the day. A suggested categorization of clinical outcome is presented in Table VI. It is recommended that no clinical outcome grade be assigned until at least 6 months after operation.

UPPER EXTREMITY DEEP VENOUS DISEASE

Diagnosis. The clinical diagnosis of thrombosis of the deep upper extremity and cervical veins is imprecise. Reports of axillary-subclavian or internal jugular vein thrombosis must therefore use contrast or isotope phlebography or duplex scanning to establish the diagnosis objectively.

Cause. The author should specify the presumed cause of the venous thrombosis. Recognized causes include clavicular and proximal humeral fracture, either acute or with malunion or chronic malposition; central venous cannulation; injection or infusion of hypertonic or irritating solutions; or septic phlebitis. Diagnosis of the latter requires organism identification. In the absence of any of these factors, the thrombosis may be presumed to be idiopathic. The term *idiopathic* as used here includes the condition termed *effort thrombosis.* In many such idiopathic cases there is venosclerosis at the level of the head of the clavicle that may or may not have resulted from repetitive venous compression in the region of the thoracic outlet.

Upper extremity postthrombotic syndrome. Obstruction not reflux is the cause of upper extremity venous symptoms. Description should include the patient's subjective assessment of discomfort either at rest or in relation to specific activities and should also include objective descriptions such as the presence of cyanosis, and the presence and extent of limb swelling documented by circumferential limb measurements compared with the uninvolved limb. Hand edema should be specifically noted, as well as any resulting functional impairment of the fingers.

Vascular laboratory. A number of vascular laboratory tests have been used in the diagnosis of acute upper extremity venous thrombosis or documentation of upper extremity postthrombotic syndrome. Thus far only duplex scanning for the diagnosis of upper extremity and cervical deep venous thrombosis has achieved widespread acceptance. Therefore if vascular laboratory results other than duplex scanning are reported in the diagnosis of acute thrombosis, the author should describe in detail both the performance and results of the tests selected and include results from normal persons, as well as from the contralateral normal upper extremity.

Treatment. The same details of medical treatment should be included as described earlier in the section, "Acute Lower Extremity Venous Thrombosis." Reports of the results of surgical treatment for either acute axillosubclavian vein thrombosis or upper extremity postthrombotic syndrome should include descriptions of the postoperative phlebograms or duplex scans visualizing the area of operation. Patency of a reconstructed axillosubclavian vein must be documented by either phlebography or duplex scanning and presented in a life-table format. Attempts to assess the efficacy of surgical therapy with only the patient's subjective assessment of benefit may be grossly inaccurate on the basis of the well-described tendency to spontaneous improvement with either of these conditions.

PULMONARY EMBOLISM

The author should state the type of pulmonary embolus (PE) being described. The most common is aseptic embolism. Less common but well-recognized PE include septic PE, organism identification required; tumor PE; air PE; fat PE; and foreign body PE, including such items as catheters, heart valves, bullets, pellets, and caval interruption devices.

Table VI. Clinical outcome after surgery

+3	**Asymptomatic**, no symptoms of chronic venous disease, improvement of VRT to normal or at least +5 seconds, improvement in AVP to normal or at least −10 torr
+2	**Moderate improvement**, mild symptoms of chronic venous disease, improvement of VRT to normal or at least +5 seconds, improvement in AVP to normal or at least −10 torr
+1	**Mild improvement**, clinical improvement **or** improvement in vascular laboratory test results (VRT or AVP)
0	**Unchanged**, no change clinically or by vascular laboratory test results
−1	**Mild worsening**; worsening of symptoms of chronic venous disease **or** vascular laboratory tests (VRT or AVP)
−2	**Significant worsening**, worsening of symptoms and worsening of vascular laboratory test results (VRT or AVP)
−3	**Marked worsening**, same as **−2** accompanied by either new or worsening ankle claudication

AVP, Ambulatory venous pressure.

Diagnosis

The clinical manifestations of pulmonary embolism are similar to other cardiopulmonary disorders, often making a clinical diagnosis difficult or impossible. Arterial blood gas determinations and electrocardiographic changes may be suggestive, but they have insufficient sensitivity and specificity to establish a firm diagnosis.[30] Reports concerning pulmonary embolism must therefore confirm the diagnosis objectively with an imaging study.

Pulmonary arteriography. This remains the "gold standard" against which all other diagnostic tests must be compared.

Ventilation/perfusion scanning (V/Q scans). Perfusion lung scanning alone is of unacceptably low specificity to establish a diagnosis of PE. High probability (but not intermediate probability) V/Q scans may be used to establish objectively the diagnosis of PE. A high probability scan requires an embolus sufficiently large to occlude arterial circulation to an entire pulmonary segment and requires that the patient have a normal chest x-ray film. Low probability V/Q scans are missing one or both of these requirements and are inadequate to establish the diagnosis of PE.

Classification

PE has been classified anatomically and functionally. The term *massive embolism* has traditionally required significant filling defects in two or more lobar arteries, which implies greater than 40% obstruction of the pulmonary circulation. The term *submassive embolism* defines a PE with obstruction of less than two lobar arteries. However, these anatomic definitions are of limited usefulness and are less accurate in predicting death than a functional definition based on hemodynamic measurements. The hemodynamic response to acute embolization is a function not only of the size of the embolus but also of coexisting heart and pulmonary disease and the magnitude of both the neurohumoral and vasoconstrictor response to the embolism. Serial hemodynamic measurements essential to the functional definition of PE include blood pressure, pulse, central venous pressure, cardiac output, pulmonary artery pressure, pulmonary capillary wedge pressure, and pulmonary vascular resistance. The presence or absence of cardiac arrest or shock should be described because these are strong predictors of death associated with PE. The definition of shock is not standardized and must be clearly stated along with the duration of shock. Such a definition should include standard hemodynamic alterations (pulse rate and reductions in blood pressure and cardiac output), systemic responses (based on arterial pH, mixed venous blood gas determinations, oxygen consumption, and reductions in urinary output), and the need for vasopressors and inotropic support. A classification of PE on the basis of clinical, anatomic, and hemodynamic modifiers is given in Table VII.

The term *recurrent PE* can be used only when either a pulmonary arteriogram or high probability V/Q scan documents both a new and a previous embolism.

Chronic PE. Use of the term *chronic PE* requires sequential documentation of recurrent PE on multiple occasions over a period of months by either high probability V/Q scans or multiple pulmonary arteriograms. Alternately, a single pulmonary angiogram that shows emboli in both lungs and obstruction of more than half the pulmonary vasculature along with a classic history of exertional dyspnea progressing to severe respiratory insufficiency in the setting of documented multiple recurrent DVTs is also sufficient to establish the diagnosis of chronic pulmonary embolism syndrome. The term must not be used in the absence of such documentation.

Table VII. Classification of pulmonary embolism

Clinical class	*Characteristics*
0	Asymptomatic PE
1	Symptomatic PE No hemodynamic alterations <40% pulmonary arterial circulatory obstruction
2	Symptomatic PE Minor or no hemodynamic alterations >40% pulmonary arterial circulatory obstruction
3	PE with major hemodynamic alterations and shock regardless of degree of pulmonary arterial circulatory obstruction
4	PE with cardiac arrest regardless of degree of pulmonary arterial circulatory obstruction

Therapy for acute pulmonary embolism

Therapy for acute pulmonary embolism includes supportive measures for cardiopulmonary failure, as well as anticoagulation, thrombolysis, and surgical treatment. The same details of anticoagulant treatment should be included as earlier described in the section, "Acute Lower Extremity Venous Thrombosis."

The surgical treatment of PE includes both caval interruption, as well as open pulmonary artery embolectomy, and percutaneous suction embolectomy. Descriptions of caval interruption should specify how this was accomplished. If a percutaneous device is used, the author must specify the type of device, route of placement, final location of device, and whether placement was confirmed phlebographically.

Reports describing thrombectomy for chronic PE should include postoperative pulmonary function tests, as well as pulmonary arteriography with measurement of pulmonary artery pressure because it is recognized that patients may display markedly improved postoperative arteriograms without improvement in pulmonary function.

Pulmonary embolism prophylaxis

Caval interruption, by whatever means, should only be described as prophylactic for patients who have not had a recent PE. The type, location, and timing of venous interruption in relation to subsequent or concomitant operations and whether concomitant anticoagulation is used and its intensity should be specified. The technique of device insertion should be described (i.e., operative or percutaneous), and radiographic confirmation of the localization of the device after insertion should be noted.

Autopsy verification of fatal PE

Probably lethal PE. Probably lethal PE consists of thrombus or thrombi in the main pulmonary artery trunk or bifurcation, a portion of which may be in the right ventricle; thrombi in both right and left pulmonary arteries; thrombi in one or more contralateral lobar arteries.

Possibly lethal PE. The designation of possibly lethal PE requires consideration of both the extent of obstruction of the pulmonary arterial system and the patient's underlying cardiopulmonary state. A number of factors may aggravate hypoxemia and diminish the capacity of the right side of the heart to accept an increased afterload, therefore affecting the patient's potential to survive an acute embolus. Such factors include thrombus occluding the main right or left pulmonary artery; thrombi in two or more lobar arteries of one lung and in one or more contralateral lobar arteries; combination of thrombi in unilateral or bilateral lobar or segmental (and equivalent subsegmental) arteries equal to the above.

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