REVIEWS

Invasive treatment of deep venous disease. A UIP consensus

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Deep venous disease includes primary and/or secondary pathological changes in the deep venous system. These may consist of valve insufficiency, complete or incomplete vein obliteration and/or functional impairment.
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Pathophysiology

Deep veins constitute the outflow track of the lower extremities. In healthy individuals, the superficial veins function as tributaries of the deep veins, and the direction of the blood flow is from the superficial into the deep veins. This unidirectional flow is possible because of the pressure gradient directed towards central veins, low resistance of deep vein, work of the muscle pump, and the presence of competent valves. Recent studies revealed substantial deficiencies in current understanding of venous physiology. Pressure changes in veins during muscle activities are variable and do not always follow the model of ambulatory decrease in pressure.[1] Relationships between the flow in major veins and their tributaries have been shown to be more complex than pressure-based models suggest.[2] Additional functional roles of venous valves are suggested, such as flow facilitation by creation of jets, maintaining pulsatile flow, and providing "flushing" of sinuses.[3]

Disruption of the venous blood flow plays the key role in natural history of venous diseases. This can be due to incompetence of venous valves, acute or chronic occlusion of a vein, increase of resistance to blood flow (as a result of stenosis, intralumenal synechia, or increased rigidity of the venous wall), or any combination of these abnormalities. Cascade of biological reactions was identified as a result interaction of disturbed flow and endothelium, and resulted in prothrombotic and proinflammatory changes in endothelial cells.[4] This suggests that pathological processes can be initiated by minor disturbances in flow, perhaps preceding major hemodynamic consequences detectable by current diagnostic techniques. Changes in collagen in patients with CVD appear to be systemic and not limited to the venous wall.[5] This suggests that processes other than those that relate to changes in blood flow may play role in natural history of venous diseases.

Diagnosis

Management of chronic deep vein disease requires accurate objective diagnosis of abnormalities in the venous tree from the lower calf to the diaphragm. Segment by segment diagnosis of reflux and obstruction is the standard, performed by Duplex scan. Venography with or without intravenous ultrasound is required for definitive diagnosis in the iliac and IVC vessels. Physiologic studies with pressures and volume methods are useful to evaluate global function and differentiate dominant obstruction from reflux.[6] These studies are all complementary and need be applied selectively as the severity of the clinical problem increases. New diagnostic modalities provide information that is potentially useful for identification and evaluation of venous abnormalities. B-flow ultrasound allows direct observation of the function of venous valves, instead of indirect inferences made from conventional duplex studies. Earlier and more precise identification of valvu-

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TABLE I.—*Results of deep vein reconstruction.*

Author, Year	Surgical technique	Number of limbs (number of valves repaired)	Etiology PVI/Total	Follow-up month (Mean)	Ulcer recurrence or non healed ulcer (%)	Hemodynamic results		
						Competent valves (%)	AVP improved	VRT improved
Masuda 1994	I	32	27/32	48-252 (127)	-28	24/31 (77)*	81%	50%
Lehtola 2008	I	12	12-May	24-78				
	TMEV	7	7-Mar	-54	/	-55		
	±TMEV	1	0/1					
Perrin 2000	I	85 (94)	65/85	12-96 (58)	10/35 (29)	72/94 (77)		Normalized
Raju 1996	I	68 (781)	/	12-144	16/68 (26)	30/71 (42)		
Raju 1996	TMEV	47 (111)	/	Dec-70	14/47 (30)	72/111		
Lurie 1997	I	52/52	52/52	36-108	4/19 (21)	42/52 (81)		
Raju 2000	TCEV	141 (179)	98/141	1-42	-37	-59	15%	Normalized
Rosales 2006	TMEV	17 (40)	17/17	3-122 (60)	3/7 (43)	-52	50%	
Sottiurai 1988	I	143	/	9-168 (81)	9/42 (21)	107/143 (75)		
Tripathi 2004	I	90 (144)		-24	-32	-79.8		
	TMEV	12 (19)	118		-50	-31.5		

TABLE II.—*Banding, cuffing, external stent, wrapping.*

Author, Year	Number of limbs (number of valves repaired)	Site	Etiology PVI/Total	Follow-up month (Mean)	Ulcer recurrence or non healed ulcer (%)	Hemodynamic results		
						Competent valves (%)	AVP improved	VRT improved
Akesson (Venocuff) 1999	20 (27)	F, P	7/20	5-32 (19)	2 (PTS)/10 (20)	PVI 7/7 (100) PTS 7/10 (70)	PVI-10% PVS-10%	PVI-10% PVS-100%
Camilli (Dacron) 1994	54	F	54/54	4-63		41/54 (76)		
Lane (Venocuff) 2003	42 (125)	F, P	36/42	64-141 (93)	(20)	(90)		100%
Makhatolov (Vedenski Spiral) 2009	24 (54)	F	28/28	12-60 (29)	No C6	?		
Raju (Dacron)	96	F, P, T				60-72 (83)		

lar dysfunction may become possible with this technique. The use if intra-venous ultrasound scanning (IVUS) opens the opportunity of more reliable and precise identification of venous obstructions.[7] Magnetic resonance imaging is a developing non-invasive method with potentially leading role in identification of venous abnormalities.[8] The elements of the CEAP classification including clinical, etiologic, anatomic and pathophysiologic features are all necessary aspects of the definitive workup.[9] These principles have been routine in the management of deep vein disease for decades and represent a benchmark for venous diagnosis that far exceeds the diagnostic detail followed in the management of infra-inguinal superficial and perforator disease.

TABLE III.—*Transposition results.*

Author, Year	Number of limbs	Etiology PVI/Total	Follow-up month (Mean)	Ulcer recurrence or non healed ulcer (%)	Hemodynamic results		
					Competent valves (%)	AVP improved	VRT improved
Cardon 1999	16	16/16	24-120	4/9 (44)	12/16 (75)		
Johnson 1981*	12	12/12	12	4/12 (33)		Unchanged	Unchanged
Kistner 1994	14		48-252	7/14 (50)	10/13 (77)	70%	70%
Lehtola 2008	14	12/14	24-78		(43)		
Perrin 2000	17	19/17	12-168	2/8 (25)	9/17 (53)		
Sottiurai 1996	20		9-119	9/16 (56)	??????		

TABLE IV.—*Transplantation results.*

Author, Year	Number of limbs (number of valves repaired)	Site	Etiology PVI/Total	Follow-up month (Mean)	Ulcer recurrence or non healed ulcer (%)	Hemodynamic results		
						Competent valves (%)	AVP improved	VRT improved
Bry 1995	15	P		15-132	3/14 (21)	7/8 (87)	Unchanged	Unchanged
Eirksson	35	F, P	35/35	6/60		11/35 (31)		Unchanged
Lehtola 2008	29	F, P	25/29	24-78 (54)		(16)		
Mackiewicz 1995	18	F		43-69	5/14 (36)			
Nash 1988	25	P	25/25		3/17 (18)	18/23 (77)	18%	
Perrin 2000	32	F	31/32	12-124 (66)	9/22 (41)	8/32 (25)		19%
Raju 1999	83	F. P. T	8,/83	12-180	(40) 6 yrs	(38) 4 yrs	Unchanged	
Raju 1996	54	F		12-180		16/44 (36)		
Rosales 2008	22 inclusing 2 double Tr. Tr.+ other procedure	F. P	22/22	6-108		GSV tr. 14/26 AV Tr 3/6		
Sottiurai 1988	18	F. P		7-144	6/9 (67)	6/18 (33)		
???????	??	F. P		?????	???	??????	?????	

Treatment strategies

Following accurate diagnosis and clinical assessment, the alternatives for successful treatment can be identified. In planning surgery for advanced venous disease advantage can be taken of the fact that the venous system has broad capacity for self correction via new collaterals and other pathways of venous return that have yet to be understood.

Within these limitations, certain principles appear to be useful and clinically appropriate at this time:

1. partial correction of venous defects may have enormous influence on the clinical state, allowing the extremity to achieve a clinically compensated state consistent with improved or normal function for the future. However, in advanced disease it is seldom that the veins can be restored to a totally normal state;[10]

2. axial reflux in the superficial veins is poorly tolerated by the skin and subcutaneous tissues of the lower extremity and deserves surgical correction. Axial deep vein reflux need not be corrected as the initial step when deep and superficial refluxes co-exist. In the short term, deep axial reflux appears to be better tolerated than superficial axial reflux. There is however limited data indicating that correction of deep reflux can

TABLE V.—*Neovalve.*

Author, Year	Technique	Number of limbs	Etiology PVI/Total	Follow-up month (Mean)	Ulcer recurrence or non healed ulcer (%)	Hemodynamic results		
						Competent valves (%)	AVP	VRT improved
Plagnol 1999	Bicuspid Neo valve	44	4/44	6-47 (17)	3/32 (17)	38/44 (86)		
Labas 2009	Wilson technique on transplanted axillar vein±FV Valvuloplasty Sclerotherapy	56		48-52 (127)	(18)	51/56		
Maleti & Lugli 2009	Monocuspid or Bicuspid Neo Valve Constructed with deep vein wal	19+21 =40	40/40	2-78 (28.5)	7/40 (17)	13/19 (68) 21/21 (100)		75
Opie 2008	Monocuspid Neo valve Constructed with deep	14		(48)	0/6	13/14 (92%)		

improve clinical outcomes and if not corrected can contribute to recurrence of varicose veins.[11-14] Failure of the extremity to thrive following correction of superficial reflux is an indication for deep vein reconstruction;[15-18]

3. correction of deep venous reflux in primary disease can be accomplished by both internal and external repair. The internal repair is the more durable, the external is simpler to perform [16, 17] (Tables I, II);

4. correction of post-thrombotic reflux can be achieved by direct repair when the proximal valve has not been destroyed. When the valve has been deformed transposition of the refluxing segment to an adjacent competent vein segment, or transplantation of a competent valve from another source may be successful. The long-term durability of these repairs is less than internal valve repairs in primary disease [17-20] (Tables III, IV);

5. recent reports of open surgical creation of an autogenous valve from post-thrombotic scarring within the vein provide the alternative of effective long-term success in the post-thrombotic refluxing extremity. The neovalve technique is based on the creation of a flap by dissecting the vein wall: while not impeding the normal flow of blood, it is able to withstand the reflux.[21, 22] Reports of non-autogenous valve substitutes have not shown clinically acceptable results to date;[23]

6. early restoration of iliac vein patency at the time of acute ilio-femoral thrombophlebitis has shown improved long term results and is becoming the norm for treatment in the acute phase to prevent post-thrombotic disease;[24, 25]

7. with modern imaging techniques, it is now recognized that iliac vein obstruction is ubiquitous and is often present in silent form in the general population.[26] In symptomatic (CEAP clinical class 3-6) primary and postthrombotic CVI patients, such lesions are present in >90% when

TABLE VI.—*One-seven year results of three independent series of deep vein reconstruction.*

	PVI	PTS
Kistner		
JVS: 1994		
Disease-free		
1 yr:	86%	75%
5 yr:	73%	50%
7 yr:	73%	43%
Raju		
JVS: 1996		
Ulcer-free		
1 yr:	85%	75%
5 yr:	60%	64%
7 yr:	60%	48%
Perrin		
JVS: 1997		
Ulcer-free		
1 yr:	93%	81%
5 yr:	71%	60%
7 yr:	83%	42%

From Kistner.[31]

examined with IVUS: at arterial crossover points in primary disease and as focal or diffuse lesions (Rokitanski stenosis) in postthrombotic limbs.[27] Iliac vein obstructive disease has been treated effectively by balloon angioplasty and stenting. This technique has replaced most of the attempts to bypass iliac obstructions, and provides good intermediate term (2-6 years) results with minimal morbidity, less than 5% in-stent restenosis, and significant improvement of pain, swelling, and quality of life.[28] Its success depends upon the availability of adequate distal inflow and proximal outflow from the stented segment. Details of the diagnostic criteria for this procedure are under investigation;[29]

8. the indications for deep vein reconstruction are limited at this time to cases in which simpler forms of venous repair have failed to control the problem and the patient is healthy enough to benefit from the correction with increased activity or decreased need for medical care.[15-17]

The risk of deep vein surgery has proven surprisingly low. Mortality has been rare throughout the 40-50 year history of reporting from around the world. The morbidity of operating inside the veins includes several considerations. Thromboembolic complications are rare in primary disease, more frequent in post-thrombotic disease.[30]

The chance exists in operating upon pure primary disease of converting the problem to postthrombotic disease if the reconstruction becomes clotted. This complication is minimized by adherence to proper principles of surgery including provision of adequate inflow and outflow and removal of endovenous obstructions to flow.

Venous reconstructions are usually performed on anticoagulated patients which can raise the risk of post-operative hematoma to the 5-10% level. When higher doses of anticoagulation are used the higher risk of wound hematoma is balanced with the risk of thrombosis when the anticoagulation is at a lower dosage (Tables V-VI).

An important factor in venous surgery is that the procedure is performed to improve the quality of life and complications which might worsen the quality of life are poorly tolerated.

Future questions to be answered

Recent progress in treatment, diagnosis and evaluation of venous diseases revealed significant gaps in our knowledge and understanding of venous physiology and patho-physiology.

Relationships between changes in deep, superficial and perforator veins in natural history of chronic venous disease remains poorly understood. The impact of the presence, severity and extend of reflux and obstruction on venous function, and their interaction when present in the same extremity remain unknown. Investigation of these and other fundamental aspects of venous disease may lead to further advantages in management of patients with deep vein abnormalities.

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