Eur J Vasc Endovasc Surg **28**, 182–192 (2004)
doi: 10.1016/j.ejvs.2004.04.002, available online at http://www.sciencedirect.com on SCIENCE DIRECT®

Venous Flow Restriction: The Role of Vein Wall Motion in Venous Admixture

S. Raju,* G. Cruse, M. Berry, S. Owen, E. F. Meydrech and P. N. Neglen

River Oaks Hospital and University of Mississippi Medical Center, Jackson, MS, USA

Objectives. *There are wide differences in flow between vascular beds at rest, even more during stress. The hydrodynamic energy (*Energy grade line *or EGL) of venous outflows must also vary considerably between vascular beds. We explored the mechanism of venous admixture of differing energy flows using a mechanical model.*
Materials and methods. *The model simulated two venous flows coalescing at a venous junction and then flowing through collapsible venous pumps. Flow rates and pressures were monitored when the venous pumps were full (steady state) and when they were compressed and allowed to refill inducing wall motion (pump flow).*
Results. *With increasing EGL differences between two coalescing venous flows, reduction or cessation (*venous flow restriction*) of the weaker flow occurred during steady state; higher base EGL of both flows ameliorated venous flow restriction and lower base EGL the opposite. Outflow obstruction favoured venous flow restriction. Pump action in the vicinity of the venous junction abolished venous flow restriction and maximized both venous flows.*
Conclusion. *The model suggests a pivotal role for vein wall motion in venous admixture and regional perfusion. Observations in the model are explained on the basis of* network flow *principles and collapsible tube mechanics.*

Key Words: Venous flow; Venous circulation; Vein wall motion; Venous pump; Venous pressure; Venous network; Network flow; Tissue perfusion; Collapsible tube.

Introduction

The bulk of peripheral resistance resides in the small arteries and arterioles.[1,2] Arterioles are vaso-reactive through a heterogeneous set of effectors (neural, hormonal and local) and receptors, particularly a family of alpha receptors that dynamically modulate the resistance offered according to varying regional and systemic flow requirements.[3] There are wide variations in the distribution and efficacy of these units that account for regional perfusion differences at rest, stress or metabolic activity. Examples of low and high vascular resistance beds at rest include: muscle and skin of the lower limb, internal and external carotid circulations in the head and neck, and visceral and non-visceral vascular beds in the abdomen. Some outflows (e.g., renal, hepatic) have passed through two sets of vascular beds before joining the inferior vena cava. Response to stress also varies: some vascular beds vasoconstrict such as renal and mesenteric beds in shock while others vasodilate (e.g. muscles in exercise, mesenteric bed during digestion). Regional blood flow variations of 10–20 times the resting levels occur and as great as one hundredfold difference between extremes of vascular tone has been recorded in some vascular beds.[4] The venous effluents from these beds with widely diverging resistances likely emerge with different hydrodynamic energy.

While much work has focused on arterial dynamics, venous outflow has been regarded as occurring through passive conduits. If such were the case, admixture of differing energy flows could not be efficiently accommodated. The human circulation can be viewed as a *network*,[5] with the venous circulation forming a sub network with multiple input points, *loops* and *nodes*. When considered in this light, venous flow can have a significant effect upstream (arterial inflow) under certain conditions.

We explored venous flow characteristics in a mechanical model incorporating a venous junction and two coalescing venous flows of differing hydrodynamic energy. *Venous flow restriction* and other novel flow phenomena observed in this model are of general interest in understanding network dynamics in collapsible tubes. The model suggests a basic mechanism for venous admixture, with potential physiologic and pathologic importance but requires biologic validation. A tentative network model of venous circulation is presented for further work.

*Corresponding author. Seshadri Raju, 1020 River Oaks Dr. #420, Flowood MS 39232, USA.

1078–5884/020182 + 11 $35.00/0

Material and Methods

Terminology

Network terminology conventions are used. Total hydrodynamic energy of pipe flow when plotted as a line along the course of fluid flow is called *Energy Grade Line* (EGL or simply EL).[5] EGL = total head (pressure head + elevation head + velocity head). The concept of 'head' derives from the Bernoulli equation: total hydrodynamic energy(E) = pressure energy(P) + gravitational potential energy (ρgh)+kinetic energy ($1/2\rho V^2$), respectively. The energy terms in the equation are expressed per unit weight of the fluid to arrive at the corresponding 'head'. Another term used here is *Hydraulic Grade Line* (HGL); HGL = pressure head + elevation head. Therefore, EGL = HGL + velocity head; practical application is that EGL can be calculated from pressure and velocity data. HGL also referred to as Piezometric head (after the measuring device) is indicated by lateral pressure. Although 'lines' by definition, EGL and HGL terms are used synonymously with corresponding 'heads' at specific points in flow.

Experimental design

Basic setup of the mechanical venous model is shown in Fig. 1(a). It simulates the venous limb of the circulation starting at the venous end of the capillary.

The experimental model was constructed to simulate many of the physiologic parameters prevailing in the lower limb venous circulation of an erect adult subject.[2] Particularly, ambulatory venous pressure changes in the foot (resting and post-exercise pressure, recovery time) and ejection volume of the calf venous pump were quantitatively simulated. The model exhibited phasic flow variations as with respiration. The latex tube used in the model for calf and abdominal pumps has many of the physical characteristics of human veins and is considered a satisfactory proxy for venous flow modelling.[6]

Since the elevation and velocity heads of the two flows at their origin from the two venous reservoirs were identical (same height[a] zero velocity), EGL differences between the two flows could be set solely by varying the pressure head applied to each of the two reservoirs. HGL variations at the reservoir level are easier to set and monitor than are direct EGL measurements.

Three different modes of flow were tested using the above basic set-up: (1) varying the base pressure head applied identically to both venous reservoirs, simulating a likely range of physiologic pressures at the venous end of the capillary resulting from systemic flow changes; (2) varying the pressure head differential between the two venous reservoirs simulating flow conditions when there are differences in vascular tone between vascular beds, and (3) varying the external pressure applied to the abdominal pump.

Venous flow model

The set-up consisted of two venous flows fed from separate reservoirs filled with water through plastic tubing (ID $1/8''$) forming a venous junction through an inverted 'Y' connector (ID $1/8'$). The conjoined flow was then fed into the 'calf pump' immediately above. The 'calf pump' consisted of a collapsible Penrose tube ($1'' \times 10''$) mounted between two short rigid PVC connectors (ID $1/2''$), and enclosed in a rigid transparent plastic sleeve sealed around the connectors. The sleeve was pressurized from a compressed air tank, with input and dump valve controls allowing compression and ejection of the enclosed Penrose tubing (calf pump) when desired. The calf pump was then connected to a higher 'abdominal pump' through a check valve and intervening plastic tubing. The abdominal pump consisted of Penrose tubing ($1/4''$[b] $\times 6''$), enclosed in a plastic sleeve similar to the calf pump. The sleeve was also pressurized from compressed air tank through a calibrated pressure regulator, ingress and dump valves allowing compression of the abdominal pump with known external pressures.

The 'abdominal pump' emptied upwards into a 'right atrial' reservoir through a check valve. The various components with attached flexible plastic tubing and adjustable mounts were on vertical rails. The calf and abdominal pump heights were adjusted to approximate their vertical locations in a 6 foot man. The venous and 'atrial' reservoirs were mounted at the same height so that there was no flow unless additional pressure head was applied to the venous reservoirs; a minimum pressure head of ± 10 mmHg was required to produce stable fluid motion in the system. Each venous reservoir was pressurized from the compressed air tank through individual mercury manometer-controlled pressure regulators (1 mmHg

[a] Elevation head was also zero as all three reservoirs were mounted at the same height.

[b] Phasicity was lost if $1'$ Penrose was used; compression resulted in clover leaf like collapse with trilateral channels larger than the $1/8'$ feeders from the venous reservoirs offering no increased resistance to flow.

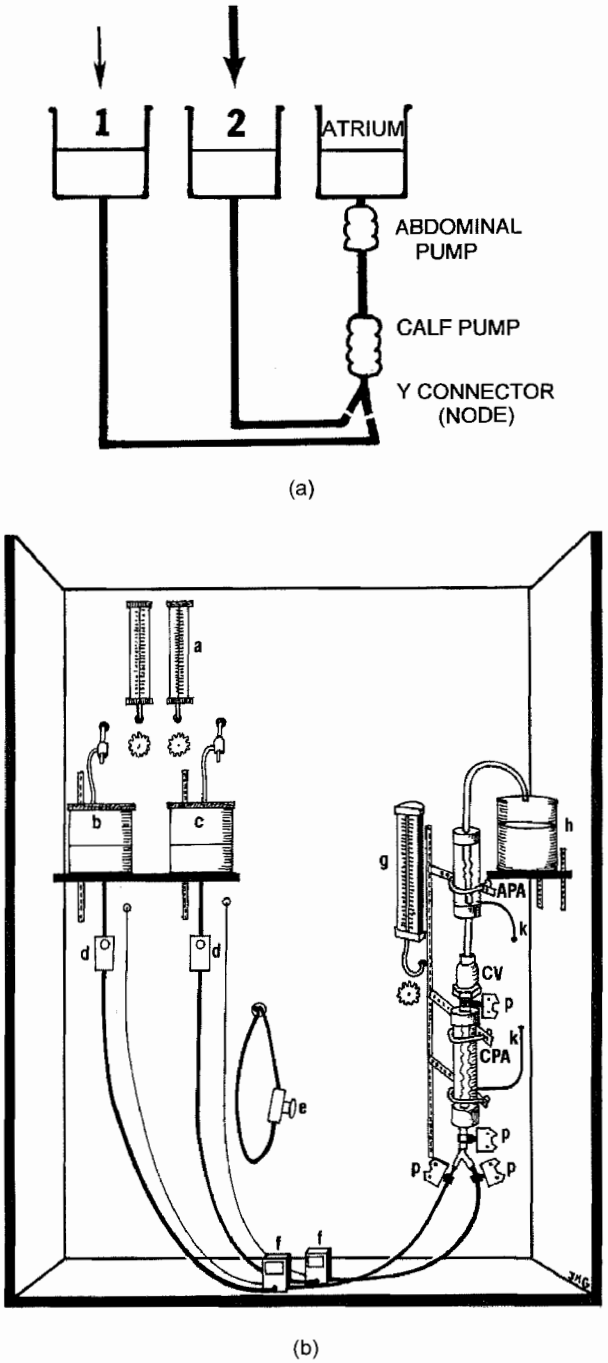

Fig. 1. (a) Schematic diagram of venous flow model used in the experiments. Two 'venous' reservoirs (#1 and 2) of identical elevation confluence through a 'Y' connector ('node') to drain into an 'atrial' reservoir of same elevation as #1 and 2 through 'calf' and 'abdominal' pumps mounted in series. Energy for flow through the system is provided by additional graduated pressure head into reservoirs #1 and 2 from a pressurized air tank. The flow pattern when the pressure head to the two reservoirs are identical ('base pressure head') is different when reservoir #2 receives a differentially higher pressure head than reservoir #1 as shown by the heavier arrow. The flow pattern when the two pumps are full ('steady state flow') is also different from the pattern observed when the pumps are empty and collapsed ('calf pump refill' and 'abdominal pump flow'). (b) Detailed diagram of venous flow model used in the experiments. (a) Mercury manometer-controlled regulators to set pressure head into the two reservoirs. (b) and (c) Reservoirs #1 and #2. (d) Shut off solenoid valves. (e) Pressurization valve (pressurized tank, not shown) for calf pump sleeve. (f) Flow meters. (g) Manometer regulator to control abdominal pump compression. (h) 'Atrial' reservoir. (k) Dump lines for abdominal and calf pump sleeves. P, pressure transducers; CPA, calf pump assembly; APA, abdominal pump assembly; CV, check valve.

tolerance) such that known variable pressure head could be provided to each reservoir. This base pressure head applied identically to both venous reservoirs ranged from 15 to 40 mmHg. Additional pressure head over the base, ranging from 10 to 55 mmHg (pressure differential) were provided to reservoir #2. Large diameter 1.2 l venous reservoirs were used so that the initial pressure differential set between the two reservoirs would not degrade significantly ($>$3 mmHg.) per each run, even if all of the flow ensued from only one reservoir.

Electro-optical in-line flow meters and pressure transducers were mounted at multiple locations (Fig. 1(b)). The pressure recording obtained from the various pressure transducers represented lateral pressure (Piezometric head) or HGL.

The apparatus was primed with water and the levels in the three reservoirs were allowed to equilibrate with the occlusive solenoid shutoff valves (Fig. 1(b)) on the venous lines open; the valves were then closed and each venous reservoir was pressurized to the desired level. Each 'run' was initiated with reopening the shutoff valves. There was steady flow from one or both venous reservoirs, depending upon the differential pressure applied. At this stage, the abdominal pump was partially compressed (+5 to +25 mmHg) to simulate inspiration and the flow allowed to stabilize again ('steady state' Fig. 2). The calf pump was then activated, allowing ejection through the check valve and the partially compressed abdominal pump into the atrial reservoir. The calf pump sleeve was then depressurized, allowing the empty calf pump to refill ('calf pump refill-flow') from the venous reservoirs. After steady flow resumed through the system, abdominal pump compression was released, simulating expiration and the 'abdominal pump flow' noted. The 'run' was then terminated by activating the shut-off valves.

All critical device inputs and outputs for each experiment such as valves and flow monitoring devices were controlled in their appropriate sequence by a personal computer with an AD board. Data

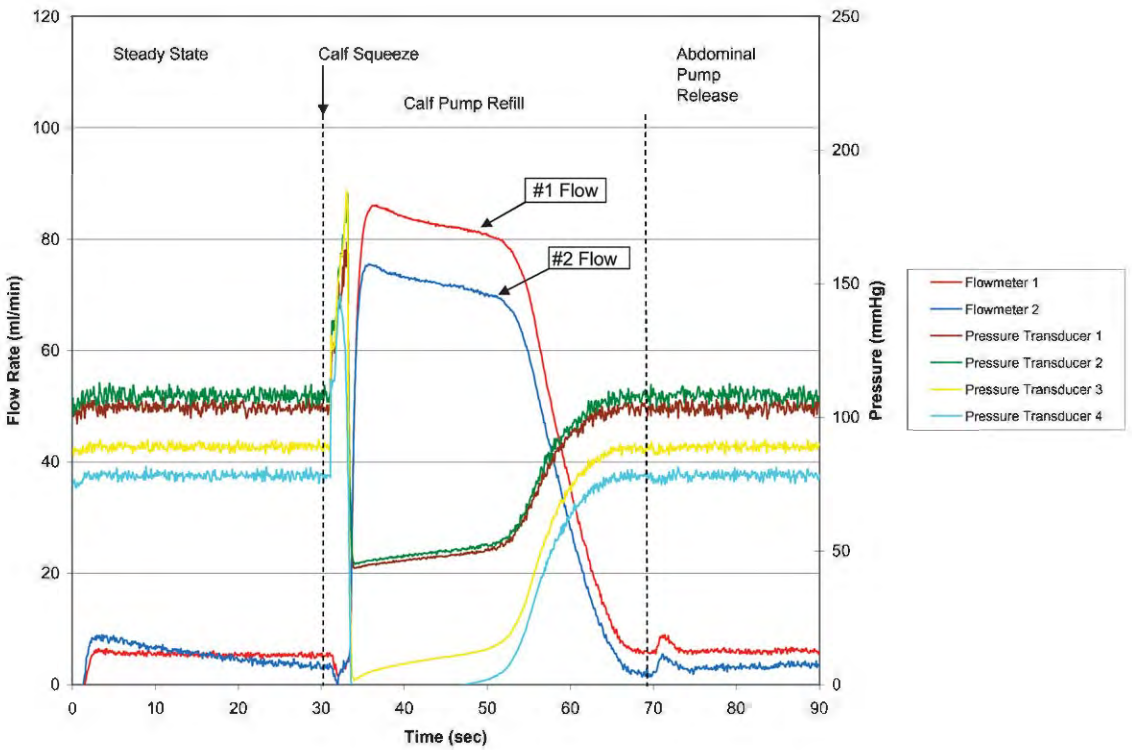

Fig. 2. Basal flow. Flow curves for identical base pressure head setting of 15 mmHg (zero pressure differential) at the reservoirs with abdominal compression of +5 mmHg. Note huge augmentation of flow rate (velocity) during calf pump refill increasing several folds from the steady state flow. Calf pump pressure (yellow line, right scale) falls to near zero.

sampling was accomplished 10 times/second during each run and downloaded into a commercially available spreadsheet program. Four repetitions of each run with the same settings were carried out and the data averaged by the spreadsheet program. Curves shown were generated from averaged data. Calf pump refill-flows are reported both as peak and time averaged mean flows.

Dynamic similarity of the model

For considerations of dynamic similarity[7] of the model to the biologic equivalent, the following comments apply: (1) neuro-hormonal mechanisms are weak or absent[2,8] in large veins, thus mechanical modelling can provide insight. (2) Water was used as the fluid medium as blood, though a suspension, is known to behave in Newtonian manner in veins of the calibre modelled.[1] (3) Vertically positioned collapsible tubes display Poiseuille flow in the steady state flow regimen[2,9] hence the use of plastic tubing between the critical latex pump elements was not considered objectionable. (4) Blood flow in the veins being modelled is laminar.[1] The flow in the model was determined to be laminar as the pressure-gradient/flow relationship in the model was linear.[1] Reynolds number of model flows ranged from 20 to 345. Reynolds number derived from duplex flow velocities and diameter measurements in eight healthy limbs of erect volunteers in our vascular laboratory ranged from 39 to 110 for the posterior tibial vein, 177 to 421 for the popliteal vein and 114–272 for the femoral vein. (5) Frictional energy losses in the model (± 10 mmHg) approximates *in vivo* estimates.[2,10]

Statistical analysis

Flows from #1 to #2 venous reservoirs were compared as part of a $2 \times 3 \times 6 \times 5$ factorial design where the other factors were the three base pressure head settings, six differential pressures between the #1 and #2 venous reservoirs, and five abdominal pump pressures. In the absence of replication for this experiment, the third order interaction term was used as experimental error. A significance level of 0.05 was used to test all main effects and first and second order interaction hypotheses. Analysis was performed using PROC GLM of Version 8e of the personal computer SAS Program.

Results

When there was no pressure differential between the venous reservoirs, both venous flows yielded nearly identical curves through the various phases of each 'run' (Fig. 2). Flows from the two reservoirs at zero pressure differential with a particular combination of base pressure head and abdominal pump compression were similar (shown in boldface, Table 1) and will be referred to hereafter as 'basal flow'.

Venous flow restriction

Increasing the pressure differential between the reservoirs

The flow rates from the reservoirs with a range of differential and base pressure head settings for abdominal pump compression of +5 mmHg are shown in Table 1. Increasing the base pressure head to both reservoirs resulted in progressive increases in flow rates from both reservoirs during the steady state and abdominal pump phases. With increasing pressure differentials for the given base pressure head setting, steady state flows from reservoir #2 became increasingly dominant and flow from reservoir #1 became progressively reduced and remained below basal flow rates. This relative restriction of flow from reservoir #1 compared to basal flow rate ('venous flow restriction') occurred at the lower range of base pressure head settings with as little as a 5 mmHg differential; higher pressure differentials were required at higher base pressure head settings. Calf pump refilling flows averaged approximately 4 × the basal steady state flow for most settings, were virtually identical between the two reservoirs, and were nearly impervious to base pressure head or differential pressure settings employed. i.e. calf pump refill flows were exempt from venous flow restriction. Abdominal pump flow was ineffective in relieving venous flow restriction.

Varying the abdominal pressure

Table 2 shows flow patterns from the two reservoirs with a range of abdominal pump pressures (excess external pressure over internal pressure) for a variety of base and differential pressure head settings. Higher abdominal pressures tended to promote venous flow restriction. Abdominal pressure setting variations had little effect on calf pump refill flows; peak flows of ±80 ml from each reservoir were present at all settings.

Pressure changes

Lateral pressures (HGL) of the respective flows from the two reservoirs were monitored by transducers 1 and 2 mounted below the two limbs of the inverted 'Y' connector; confluent flow pressure was monitored by transducer 3 mounted between the inverted 'Y' connector and the calf pump assembly; transducer 4 monitored pressure immediately above the calf pump assembly (Fig. 1(b)).

During the steady state, lateral flow pressures recorded by all four transducers ranged between 75

Table 1. Flows recorded in the model for the indicated range of pressure settings

Reservoir input pressure (mmHg)		Steady state (ml/min)		Calf pump flow (peak) (ml/min)		Calf pump flow (avg.) (ml/min)		Abd. pump flow (avg.) (ml/min)	
Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2
15	**15**	**5**	**6**	**86**	**76**	**70**	**60**	**7**	**3**
15	20	5	13	86	79	68	63	6	13
15	25	1	23	84	81	52	58	2	26
15	30	1	29	84	83	49	59	3	31
15	35	0	35	84	86	47	63	1	37
15	45	0	44	83	93	45	70	0	45
20	**20**	**17**	**11**	**90**	**78**	**71**	**60**	**18**	**11**
20	25	15	11	89	78	71	61	16	11
20	30	14	28	88	86	56	60	15	30
20	35	12	33	88	88	55	63	15	35
20	40	14	39	88	91	52	65	14	40
20	50	10	47	87	97	51	73	13	49
25	**25**	**23**	**20**	**92**	**82**	**72**	**63**	**25**	**20**
25	30	22	26	94	86	74	68	24	27
25	35	21	32	91	88	59	62	23	36
25	40	21	38	91	91	58	66	23	41
25	45	20	43	91	94	58	69	22	45
25	55	18	50	91	98	58	76	21	53

Flows recorded with a range of base input and differential pressure settings at the reservoirs; abdominal compression pressure +5 mmHg. Basal flows (zero pressure differential) for different base pressure head settings are shown in boldface.

Table 2. Flows for selected differential pressure settings at the reservoirs with various abdominal compression pressures

Reservoir pressure (mmHg)		Abdominal compression pressure									
		+5 mmHg		+10 mmHg		+15 mmHg		+20 mmHg		+25 mmHg	
Steady state flow (ml/min)											
Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2
15	25	1	23	0	21	0	20	0	22	0	21
15	30	1	29	0	29	0	28	0	27	0	27
15	45	0	44	0	44	0	44	0	41	0	41
20	25	15	11	12	19	9	20	9	19	8	15
20	35	12	33	11	32	8	32	3	31	1	31
20	50	10	47	8	47	4	46	0	46	0	46
25	35	21	32	19	31	18	31	16	30	14	26
25	45	20	43	16	42	16	39	11	40	10	38
25	55	18	50	14	50	14	50	9	48	7	47
Calf pump peak flow (ml/min)											
Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2
15	25	84	81	86	84	88	84	86	84	86	85
15	30	84	83	86	88	88	87	86	86	87	88
15	45	83	93	86	95	87	95	85	93	86	95
20	25	89	78	89	82	90	83	91	83	91	82
20	35	88	88	91	90	90	90	89	90	89	91
20	50	87	97	90	97	90	97	88	98	89	98
25	35	91	88	93	89	94	89	93	90	95	89
25	45	91	94	92	95	93	94	92	96	94	96
25	55	91	98	92	99	93	100	92	101	94	100
Calf pump flow average (ml/min)											
Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2
15	25	52	58	61	65	67	66	63	67	62	66
15	30	49	59	60	70	64	70	61	68	62	69
15	45	45	70	57	77	59	78	58	76	56	75
20	25	71	61	69	64	69	64	69	63	69	63
20	35	55	63	67	72	65	71	63	71	63	72
20	50	51	73	62	79	61	79	59	79	60	80
25	35	59	62	69	70	70	70	68	69	70	68
25	45	58	69	67	76	67	75	65	75	66	75
25	55	58	76	64	81	66	81	62	81	64	80
Abdominal pump flow (avg., ml/min)											
Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2	Res #1	Res #2
15	25	2	26	1	24	3	24	0	24	0	26
15	30	3	31	0	32	1	31	0	30	1	32
15	45	0	45	0	46	0	46	0	44	1	44
20	25	16	11	14	20	13	21	13	20	13	17
20	35	15	35	14	35	11	36	8	36	7	36
20	50	13	49	11	49	9	49	5	51	5	50
25	35	23	36	21	35	21	36	21	36	21	32
25	45	22	45	20	45	20	44	17	45	19	45
25	55	21	53	18	53	19	53	15	53	15	52

and 110 mmHg (Figs. 2 and 3); pressure differences between the transducers were largely due to vertical positioning in the system (Fig. 1(b)). Varying the base pressure head, the pressure differential or the abdominal compression resulted in only minor changes (<10 mmHg) in the lateral pressure recorded at the various monitoring sites; pressure head variations were largely transformed into velocity changes, as evident in the resulting flow rate changes described. Notably, venous flow restriction was not reflected in pressure tracings. Transducers 1 and 2 recorded identical pressures regardless of the differential pressure head applied (Figs. 2 and 3).

During the calf pump refilling phase, lateral pressures at all four sites declined precipitously; at transducers 1 and 2 it declined to approximately 50 mmHg from about 110 mmHg and remained at this reduced level, exhibiting a near-flat curve for most of the duration of refilling when the inflow was high. Transducer 3, situated immediately below the calf pump recorded even lower pressures at or near 0 mmHg for most of the duration of calf refilling, rapidly recovering to pre-ejection levels once refilling

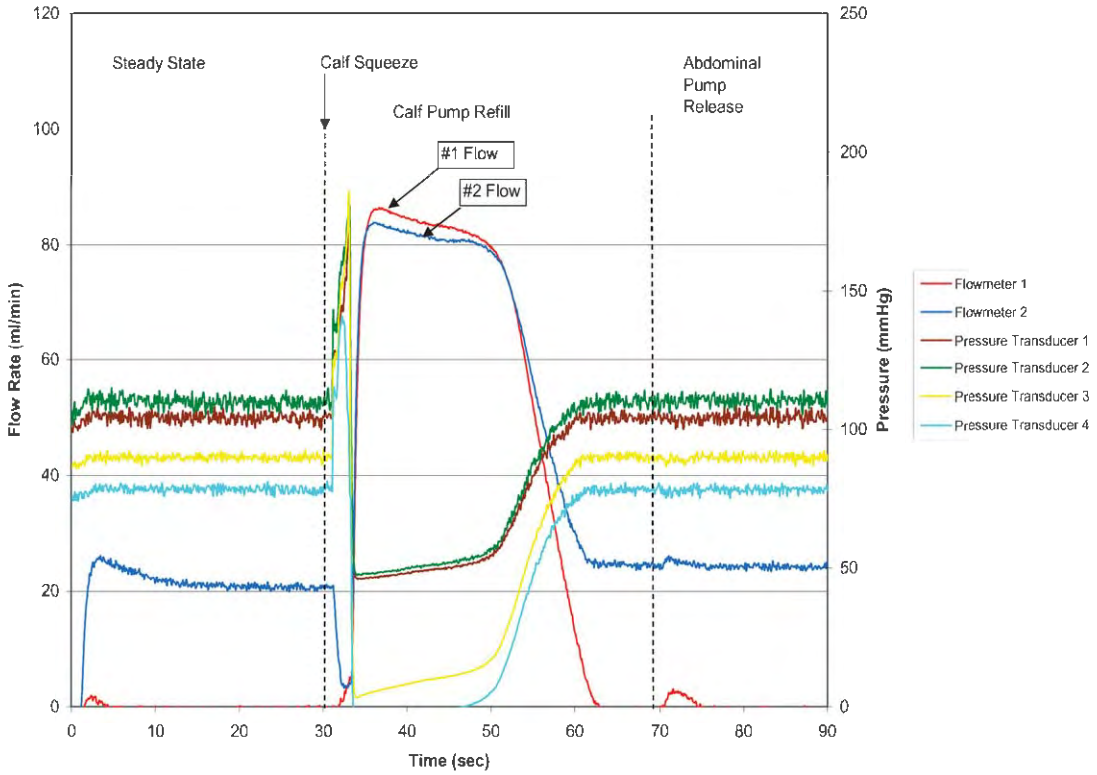

Fig. 3. Venous flow restriction flow recording for the two reservoirs with input pressures of +15 and +25 mmHg, respectively, (differential pressure +10 mmHg) at abdominal compression of +5 mmHg. There is no flow from reservoir #1 during steady state; flow resumes during calf pump refill with peak flow nearly the same as from reservoir #2 and similar to calf pump refill-flows shown in Fig. 1. There is a small insignificant (±3 ml) 'bump' in restricted flow from reservoir #1 during abdominal pump action. Note venous flow restriction is not detectable in the pressure curves (right scale) which are virtually identical to Fig. 1. See text.

was complete. Calf pump refill pressures were nearly identical for all of the various settings used in this experiment, and appeared to be determined primarily by calf pump action independent of the base, differential or abdominal compression pressure settings used.

Discussion

These experiments show: (1) when two venous flows of differing hydrodynamic energy (EGL) meet at a venous junction, reduction or cessation (venous flow restriction) of the weaker flow occurs. (2) The greater the EGL difference, the greater the trend towards venous flow restriction. (3) Minimum effective EGL differential required for venous flow restriction was a function of base EGL i.e. venous flow restriction can occur with even small EGL differentials when base EGL is low. (4) Increased abdominal pressure beyond the venous junction tends to favour venous flow restriction. (5) Pump action in the vicinity of the venous junction abolishes venous flow restriction, promotes admixture and increases both venous flows several fold from basal levels maximizing regional flow. Relevance to human circulation is discussed below.

Total energy (EGL) of 'ideal' inviscid, non-compressible fluid in steady flow remains constant along the streamline. There is a >90% decay in EGL from aorta to the cavae (from Burton's data)[10] requiring heart action for restoration. The EGL decline occurs mostly before flow reaches large veins and relatively less thereafter. This energy 'loss' as heat in the circulation is due to viscous flow shear and other departures from idealized flow. The pressure and velocity heads are inter-changeable, varying in reciprocal fashion during flow course (e.g. flow from tank, stenosis) according to local conditions. Yet total head (EGL) will remain nearly the same minus some EGL lost as heat during the energy transformations. Such to and fro transformations occur in normal blood flow with aggregate cross sectional flow area variations.[1] In the experimental model, energy inter-conversion clearly occurs at various points. Differential pressures, equilibrated rapidly at the node in the model with EGL

differences manifesting primarily as velocity (flow rate) differences. Velocity head, often ignored in venous energetics because it is a minor component,[10] nevertheless may be important in venous flow restriction and admixture.

A key assumption in this work is that there are significant EGL disparities in regional venous outflows at rest, even more during stress. Considering the differences in dimensions, architecture and variable flow rates between vascular beds, it would be surprising if it were otherwise. If the systemic resistance is lowered and cardiac output increases by 3×, there is roughly a 300% increase in vena caval EGL (from Burtons's data).[10] Selective increase in regional flow should behave likewise. Relevant real time data either at rest or stress is not available. Simultaneous regional HGL and velocity measurements are required with quiescent venous pumps; ever present cardiac and respiratory pumps pose a practical metric problem. If the assumption is valid, some type of mechanism would appear to be essential to effect venous admixture.

Circulatory network dynamics

Network principles and its analytics are well established.[5] Computerized models of even complex networks involving quasi steady or transient flows—also present in the circulation—are readily available. Application in biology, particularly in venous hemodynamics has lagged and may offer useful insights.

Human circulation contains all of the elements of a 'network'. A 'network' diagram of the human circulation is shown in Fig. 4. Key to understanding network flows is analysis of flow patterns prevailing at individual nodes in the network. Flow dynamics at the node are best illustrated by what is commonly referred to as the three-reservoir problem.[5] A node interconnects three reservoirs of different elevations through conduits of varying size and length thus yielding different flow rates (Fig. 5, left). While it is obvious that flow is outward from reservoir 'A' and there is inflow into reservoir 'C', flow direction with regard to reservoir 'B' may be more complex than it may first appear. Flow may occur into reservoir 'B' from 'A' or outward from reservoir 'B' into 'C,' depending upon the interrelationship between the elevations of the three reservoirs and their flow rates i.e., EGL relationships prevailing at the node. Engineering solutions to specific problems of this type are based on the continuity principle requiring iterative methodology, as analytical resolution is impossible. Continuity at the node (inflow must equal outflow) is the basic underlying principle of all network flows,[5] even complex networks with multiple inputs and outputs and nodes inter-connecting many conduits.

The venous model conforms to the classic three-reservoir problem except that the reservoirs are mounted on a 'U' tube arrangement to simulate orthostasis. This does not alter the hydraulic relationships during steady state flow as the hydrostatic advantage on the inflow side is counterbalanced exactly on the outflow side. The venous model also incorporates collapsible tubes as in Fig. 5 (right). Flow in distended tubes is similar to rigid conduits;[9] when collapsed they behave differently, altering the EGL relationships at the adjacent node, resulting in a flow pattern different from the steady state. Lateral pressure at the bottom of the calf pump falls to near zero (Figs. 2 and 3) converting pressure head into a large increase in velocity head. It is as if the three reservoir problem has temporarily changed to two reservoirs flowing freely into the atmosphere and the hydraulic influence of reservoir C′ and conduit c′ have been suspended.

Collapsible pumps function as energy or head storage devices releasing and accumulating it with pump action. Pump action disrupts stream line flow[11] and transients[5] develop, as the tube transitions into the collapsible regimen.[6] With pump refill the process is reversed till steady one dimensional flow is re-established. During this cycle, upstream and downstream flows to and from the pump become transiently decoupled[11] from each other like in a river dam; they are no longer synchronous in producing coordinated forward flow. In a vertical arrangement, pump action during refill prevents hydrostatic pressure of the column above the pump from being transmitted to the column below. Behaving rather like two different energy flows, both compete for the capacitance produced by pump action; reflux can occur. In a U tube arrangement with a substantial hydrostatic component, reflux flow can carry a considerable pressure head. Hydrostatic component favouring reflux is highest in pump(s) at the bottom of the U, less, if the pump is higher up. In mechanical systems, check valves may be designed to prevent the dangerously high pressures that may develop during transients with pump start up and shutdown. In man, they reserve pump capacitance for nodal flow as well. Flow disturbance at the collapsible pump is local; meaning only nodes close by will be affected. Hence, little pressure and velocity changes occur at the node with abdominal pump action (Figs. 2 and 3).

Pump effectiveness depends upon the capacitance created to satisfy regional flow needs and frequency of

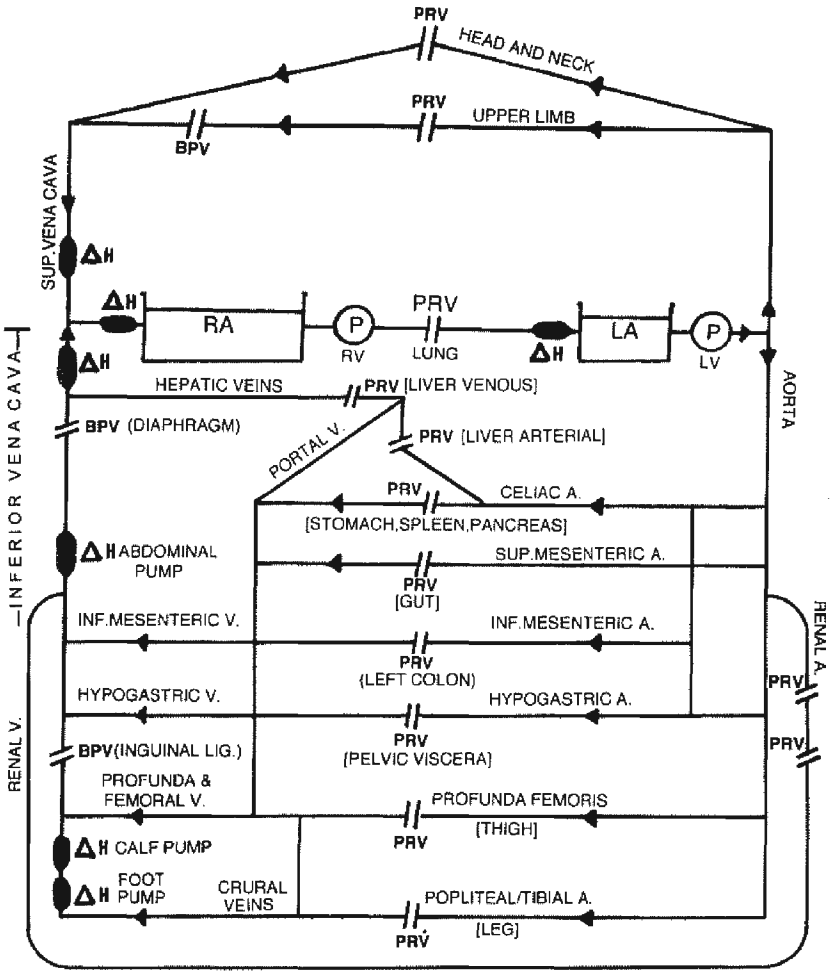

Fig. 4. Network diagram of the circulation. The left and right atria (LA and RA) are shown as reservoirs, the latter larger as the systemic circuit contains more blood volume than does the pulmonary circuit. The ventricles (LV and RV) are shown as pumps (P). Major branches of the aorta (A, artery) and major named veins (V, vein) are shown with intervening resistance depicted as pressure reduction valves (PRV). The renal and hepatic circuits contain two separate vascular beds and are each depicted with two PRVs. The pressurized abdominal cavity, the narrow diaphragmatic hiatus and the first rib produce back pressure to lower limb flows entering the abdomen, abdominal vena cava flow entering the chest, and upper limb flow entering thorax, respectively. This is represented as back pressure valves (BPV) at the respective sites. The various venous pumps function as booster pumps and reduce the pressure head at their respective sites. They are depicted as *differential head devices* or ΔH. Significant reversal of flow does not occur in vascular beds during venous flow restriction as the capillary in effect functions as a back pressure valve (BPV).

action. Pressure volume relationships in collapsible tubes are non-linear[2] with a bimodal regimen of compliance: an initial *bending* regimen, with low pressure, high-volume and a later *stretching* regimen with high pressure, low volume relationships. Pump action that drives the tube deep into the bending regimen will be more efficient than one restricted to the stretching regimen.

A 'network' view of venous circulation

Wall motion (distensibility) is a primordial characteristic of veins. This has been explained teleologically as essential for passive accommodation of capacitance changes.[4] Absence of regulatory responses such as vasoconstriction even in muscular veins in response to orthostasis or other challenges[12] has been explained similarly. The constant wall motion of veins by the many obligatory venous pumps, however, suggests a more active ongoing homeostatic function such as those suggested herein.

Venous pumps

To enhance regional flow and admixture, the main venous pathways are arranged as a series of *primary pumps* with active extrinsic (foot, calf, abdominal and thoracic pumps) or intrinsic (atrium) mechanisms and

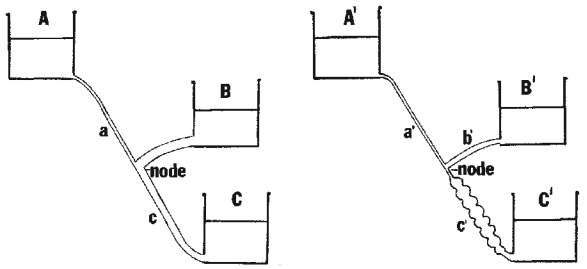

Fig. 5. Flow dynamics between three interconnected reservoirs. *Left*: Classic three-reservoir problem with rigid conduits. EGL relationships at the node determine whether reservoir B will flow outward or receive inflow from reservoir A. *Right*: Modification of flow with collapsible conduits: The presence of a collapsible tube in conduit c′ (calf pump in the model) allows for dynamic change in EGL relationships at the node influencing flow behaviour of reservoir B′. See text.

secondary pumps (e.g., saphenous veins[13]) which rely on passive elastance for wall motion. Pump action extends into the bending regimen in primary pumps and is confined to the stretching regimen in secondary pumps. Only the foot and calf pumps are under voluntary control of frequency of action. The venous pumps act as booster pumps during systole; in diastole they enhance flow and facilitate admixture at the adjacent node. They are appropriately described as *differential head devices* that produce a positive or negative change in prevailing head (ΔH, Fig 4). They are strategically located at or near major venous junctions. A 'thigh pump,' though hypothesized, has never been demonstrated, perhaps because the long femoral vein receives little major tributary flow requiring admixture.

The extrinsic compressive forces applied to the primary pumps are finely modulated. The foot pump, exposed to the highest venous pressure in the erect individual, is compressed by body weight and by plantar muscle contraction. The force of the calf pump has been measured over 250 mmHg,[4,14] exceeding the lateral pressure (HGL) of about 80 mmHg. The lateral pressure (HGL) in the abdominal pump when erect is about 25 mmHg; inspiration results in an increase of about 5–15 mmHg, adequate to induce significant collapsing wall motion. The thoracic cavae have a very low or even negative transmural pressure and may be partially collapsed;[2] the thoracic pump expands these venous structures causing wall motion during inspiration by increasing the negative intra-thoracic pressure. The cavae exhibit wall motion with cardiac cycle also, more frequent than in respiration though less in amplitude.

The HGL in the venous system is zero ('null point') at about the level of the right atrium.[2] Critical visceral and/or energy-poor outflows join the main flow stream through short conduits close to the 'null point', reducing viscous flow losses to a minimum. This arrangement moves the node closer to reservoir 'C' in Fig. 5, tending to diminish venous flow restriction of critical flows. Venous conduits draining critical vascular beds are in the bending regimen, allowing size adaptation to variable outflow with minimal restriction and energy expenditure.[9] They drain into pumps with obligatory wall motion.

Check valves

In orthostasis, lower limb circulation has a 'U' arrangement with arterial and venous limbs. Because of the hydrostatic component, deleteriously high pressures can occur at the microcirculation. Venoarteriolar reflux[12] tends to mitigate this on the arterial side. Lacking such auto regulatory mechanisms, the venous limb relies on check valves. When they fail, regional perfusion may be compromised by reflux; in addition extremely high pressures of transients (water hammer)[5] may get transmitted to the microcirculation. The combination may be even more damaging to tissue integrity. Because hydrostatic pressure is an important component of these mechanisms, valves are numerous in crural veins, decreasing higher up and are generally absent above the groin. The abdominal veins act both as a pump and as a back pressure valve via intra abdominal pressure.[2,9] Narrowing of the vena cava at the diaphragmatic hiatus and the negative intrathoracic pressure[2] may also discourage reflux.

Avenues for research

A flow pattern indistinguishable from venous flow restriction as described, is clinically observable by duplex or ascending venography. The saphenous flow admixes only minimally with deep venous flow through the perforators except after calf pump activation. Higher up, flow through the saphenofemoral junction occurs only phasically with abdominal pump action.

Venous flow restriction implies reduction or cessation of arterial inflow (upstream effect) and tissue perfusion for the duration. Since total perfusion is the sum of steady state flow and pump flow, pump dysfunction in the presence of venous flow restriction will be more serious. Clinical analogues of model conditions shown in parenthesis, favouring venous flow restriction are: regional vascular tone disparities (EGL difference), hypotension (low base EGL), outflow obstruction (abdominal pressure) and pump dysfunction (steady state). Clinical pump dysfunction occurs in valvular reflux, varicosity (loss of elastance),

post-thrombotic wall changes, pump compression by ascites, exudate or gravid uterus, tension pneumothorax, atrial fibrillation and 'stiff' lungs. Pump dysfunction and conditions favouring venous flow restriction often occur in many clinical hypo-perfusion syndromes such as: venous stasis ulceration; arteriovenous fistula; renal shutdown, lactic acidosis and organ failure in shock; hypotension related to rapid accumulation of transudate or exudate in the abdomen; eclamptic syndromes; shock with pneumothorax; adult respiratory distress syndrome (ARDS); hypotension with atrial fibrillation or pericardial tamponade; and organ failure after cardiopulmonary bypass. However, clinical extrapolation of experimental findings is not appropriate. Given the powerful auto regulatory mechanisms at the systemic and microcirculatory levels[15] that guard against tissue hypoxemia, the flow patterns of a purely mechanical venous model are not predictive, but are only suggestive for further research. Established animal models for many of the clinical conditions cited above exist. Cardiopulmonary bypass to halt the cardiac and respiratory pumps and intravenous balloon pulsations to restore or augment pump action may be useful tools in these animal models to test the flow hypotheses presented.

Summary

The mechanical venous model suggests a fundamental role for wall motion in collapsible tubes in enhancing regional perfusion and venous admixture. Venous flow restriction—a relative restriction or cessation of regional venous outflow—occurs in the absence of wall motion under certain conditions. This and other related flow phenomena can be explained on the basis of collapsible tube dynamics and 'network' flow principles. The venous circulation contains all of the elements of a 'network'. A tentative description of venous circulation in network terms has been provided. The experimental findings could be potentially important in circulatory physiology and pathology, but lack biologic verification to allow clinical extrapolation. However, potential implications are tantalizing enough to warrant exploration in biologic models.

Acknowledgements

The authors are grateful to David S. Sumner MD and William B. Brower Jr PhD for invaluable help with hydrodynamic principles and for reviewing the manuscript. Significant changes to the manuscript were made in response to their critique.

References

1 Nichols WW, O'Rourke MF. The nature of flow of a liquid. In: Nichols WW, O'Rourke MF, eds. *McDonald's blood flow in arteries*. London, UK: Arnold, 1998: 11–53.
2 Strandness DE, Sumner DS. Venous hemodynamics and control of venous capacity. In: Strandness DE, Sumner DS, eds. *Hemodynamics for surgeons*. New York, NY: Grune and Stratton, 1965: 120–160.
3 Hill CE, Phillips JK, Sandow SL. Heterogeneous control of blood flow amongst different vascular beds. *Med Res Rev* 2001; **1**: 1–60.
4 Guyton AC, Hall JE. In: Guyton AC, Hall JE, eds. *The circulation*, 9th ed. Philadelphia, USA: WB Saunders, 1996: 163–169.
5 Larock BE, Jeppson RW, Watters GZ. *Hydraulics of pipeline systems*. Boca Raton, FL: CRC press, 2000.
6 Shapiro AH. Steady flow in collapsible tubes. *J Biomed Engng* 1977; **99**:125–147.
7 Brower W Jr. *A primer in fluid mechanics*. Boca Raton, FL: CRC Press, 1999.
8 Hicks JW, Badeer HS. Gravity and the circulation: 'open' vs. 'closed' systems. *Am J Physiol* 1992; **262**:R725–R732.
9 Holt JP. Flow of liquids through 'collapsible' tubes. *Circ Res* 1959; **7**:342–353.
10 Burton A. *Physiology and biophysics of the circulation*. Chicago, IL: Year Book Medical Publishers, 1965.
11 Raju S, Fredericks R, Lishman P, Neglen P, Morano J. Observations on the calf venous pump mechanism: determinants of post-exercise pressure. *J Vasc Surg* 1993; **17**(3):459–469.
12 Gauer OH, Thron HL. *Postural changes in the circulation*. Handbook of physiology, **vol. 3**. Bethesda, MD: Am Physiol Soc, 1965. p. 2409–2439.
13 Raju S, Hudson CA, Fredericks R, Neglen P, Greene AB, Meydrech EF. Studies in calf venous pump function utilizing a two-valve experimental model. *Eur J Vasc Endovasc Surg* 1999; **17**(6):521–532.
14 Alimi YS, Barthelemy P, Juhan C. Venous pump of the calf: a study of venous and muscular pressures. *J Vasc Surg* 1994; **20**(5): 728–735.
15 Hershey SG. General principles and determinants of circulatory transport. *Anesthesiology* 1974; **41**:116–123.

Accepted 21 April 2004
Available online 7 June 2004