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Effect of External Positive and Negative Pressure on Venous Flow in an Experimental Model

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WHAT THIS PAPER ADDS

Current concepts regarding the effect of external pressure on transmural pressure and flow in veins are based on a closed static model; however, an open, dynamic model is more appropriate to simulate in vivo conditions. Substantially different results are obtained with the dynamic model. These new insights may enhance understanding of basic flow mechanics and pathophysiology of venous flow.

Objective: Positive external pressure is said to decrease transmural pressure; negative pressure in the pleural cavity is widely believed to result in negative pressure in systemic chest veins. The discrepancy between erect column height and foot venous pressure has been explained on this basis.

Methods: These core concepts rest on static closed models that may not be appropriate. This study examined the effects of external pressures in a dynamic open model that may better reflect in vivo conditions. Flow in a Penrose drain enclosed in a chamber that could be positively or negatively pressurized was used. Input and output reservoirs with pressures in the physiological range provided flow. Flow and pressure were monitored in horizontal and erect models with modifications to suit particular experiments.

Results: The discrepancy between foot venous pressure and erect venous column height was shown in this experimental model to be a result of two flows in opposite directions (superior and inferior vena cavae) meeting at the zero reference level at the heart; the upper column pressure therefore does not register at the foot. Positive external pressure results in slowing of velocity with conversion to pressure. Internal and transmural pressures therefore do not decrease. Negative external pressure has only a marginal effect on flow; importantly, internal pressure does not become negative. In an experimental set-up it was shown that negative pressure in chest veins was not necessary for air embolism to occur.

Conclusion: Persistent negative pressure in systemic chest veins probably does not occur. The reason for the discrepant foot venous pressure is likely to be a result of dynamic flow and not negative pressure in chest veins. External positive pressure results in slowing of velocity but the transmural pressure remains largely unchanged.

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INTRODUCTION

Current concepts of venous hemodynamics largely rest on work carried out during the mid portion of last century. The central view is that the venous system largely behaves as a passive reservoir without the extensive neuro-hormonal controls evident in the arterial system.[1] Much of the blood volume (>70%) resides in veins. Because veins are thin walled and collapsible, a non-linear pressure volume relationship prevails and they are subject to the influence of external pressure (positive or negative) in tissues and body compartments. Another complexity is orthostasis in humans. As erect bipedal animal models are sparse, much of the volume/pressure relationships in orthostasis were derived from human tilt table studies and bench models (often static).

A puzzling observation that attracted early attention was that foot venous pressure measured 5–10 mmHg less than the height of the erect blood column extending from the foot to a few cm above the clavicle; the jugular vein collapses above this point as blood "translocates" (a misnomer, see later) to the lower parts of the trunk in orthostasis and the contained volume is not enough to fully distend the erect venous tree. The discrepancy was attributed to the negative pressure in the thorax "holding up" as it were, a portion of the upper blood column ("dead column segment") such that it does not register in the pressure at the foot level.[2]

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The concept of transmural pressure (internal pressure minus the external pressure) was introduced to account for the influence of external pressure on venous flow mechanics.[3] The prevailing concept is that an increase in external pressure reduces the transmural pressure. This central concept has been used to explain certain anomalous flow behavior in inferior vena cava, specific flow patterns in varicose veins, and a variety of ancillary observations such as the therapeutic benefit of compression stockings.[4–6] However, the influence of external pressure on fluid within a collapsible tube will vary depending on whether the ends of the tube are closed or open, and if the contained fluid is stationary or in motion. Some of the prevailing venous concepts are based on a static closed model.

The aim of the present study is to show that several pivotal concepts in common currency are not consistent with dynamic flow open bench models and in vivo observations. One question that arose as a direct result of the experimental findings herein was the mechanism of air embolism, traditionally explained on the basis of negative pressure in the chest veins.[2] An alternative explanation is offered based on specific experiments devised to explore this mechanism.

MATERIAL AND METHODS

Dynamic flow model

Penrose tubing is a commonly used experimental proxy for studying volume/pressure relationships in iliac-caval veins; flow mechanics are largely similar with minor differences in the shape of non-linear flow curves prevailing in veins.[7] Water is frequently used (easy to handle) to qualitatively simulate blood flow mechanics, as both are known to behave in a Newtonian manner (viscosity independent of shear rate) in large conduit flows.[3,8] Quantitative differences arising from viscosity differences are not central to the general principles illustrated in the experiments.

Penrose tubing (10″ long; 7/8″ flat, 1/2″ ID when full) was mounted within a sealed transparent PVC cylinder ("Starling resistor") as previously described (Fig. 1).[9] The chamber could be positively or negatively pressurized (referred to herein as "Starling pressure" or "external pressure") to the desired level. Positive chamber pressure

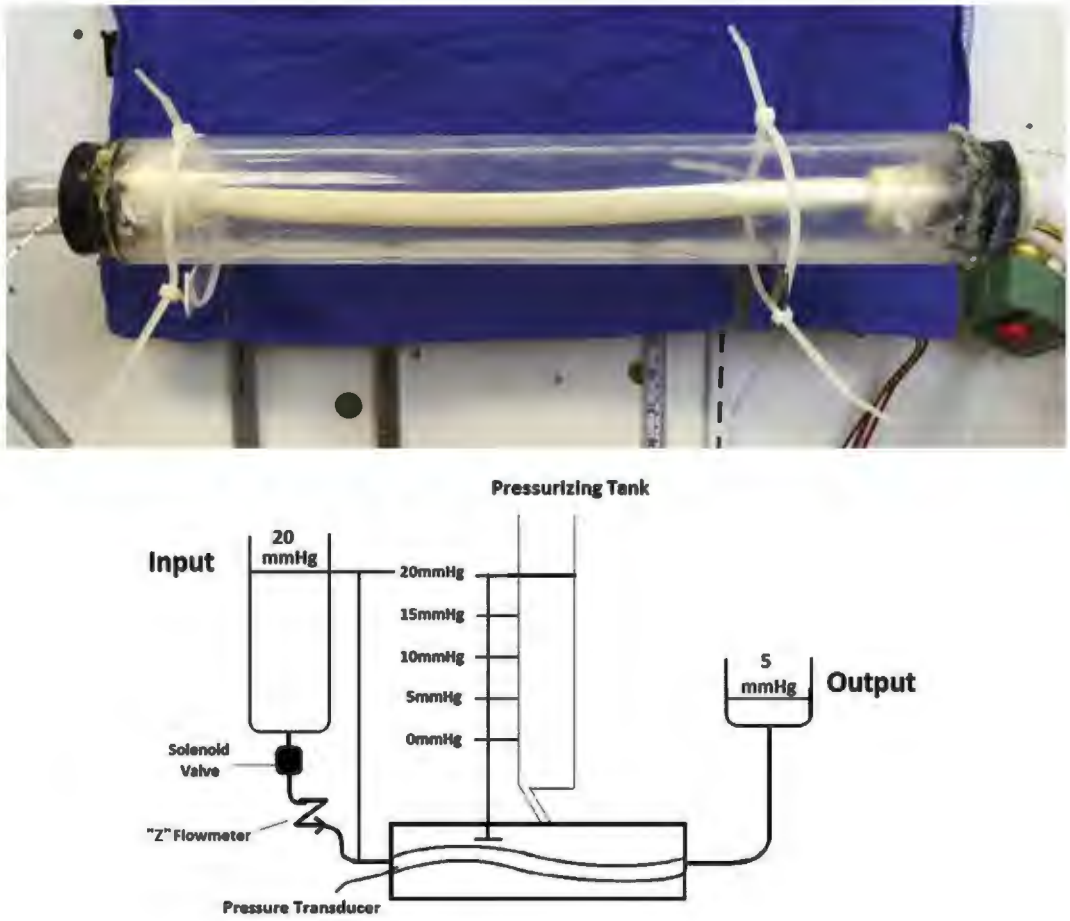


Figure 1. Dynamic venous model. Flow through the Penrose in the Starling resistor (picture detail, top) is controlled by input and output reservoirs with 20 mmHg and 5 mmHg pressure heads, respectively. External pressure over the Penrose in the Starling resistor can be controlled by fluid from a pressurizing tank; the pressure applied can be varied by adjusting the water level in the pressurizing tank. Known negative pressure is applied with a commercially available chest-tube suction system (not shown). Flow and pressure within the Penrose are continuously monitored.

was applied from a 10″ diameter water tank of known height and pressure head. The large diameter minimizes pressure head variations from minor level fluctuations. Known negative pressure was applied to the Starling chamber through a standard chest tube suction set-up used clinically. Flow and pressure within the Penrose were monitored by an in-line flowmeter (Leviflow LFS-04, Levitronix, Zurich, Switzerland) and a pressure transducer (Model 840081, Sper Scientific, Scottsdale, AZ, USA) connected to a 5 Fr. side-hole catheter inserted into the Penrose respectively, as shown in Fig. 2. Flow through the Penrose was controlled through input and output reservoirs mounted at known levels to provide 20 mmHg and 5 mmHg pressure heads approximating physiologic norms (venous capillary and atrial pressure, respectively).

This basic model was modified as needed to examine particular effects such as, for example, orthostasis (vertical Starling resistor), and static models in some experiments to show the difference from dynamic models. Specific set-up details are shown in model diagrams or described in context.

The results shown are averaged from two or more repetitive "runs" at the same settings. Variation between individual "runs" at the same settings were $<0.4\%$ for pressure readings and $<1.2\%$ for flow measurements.

Statistics

Paired two-tailed *t* test was used to assess significance. A *p* value $<.05$ was considered significant. IRB permission was granted for acquisition and publication of intravascular ultrasound (IVUS) and pressure data obtained in the course of endovascular interventions.

RESULTS

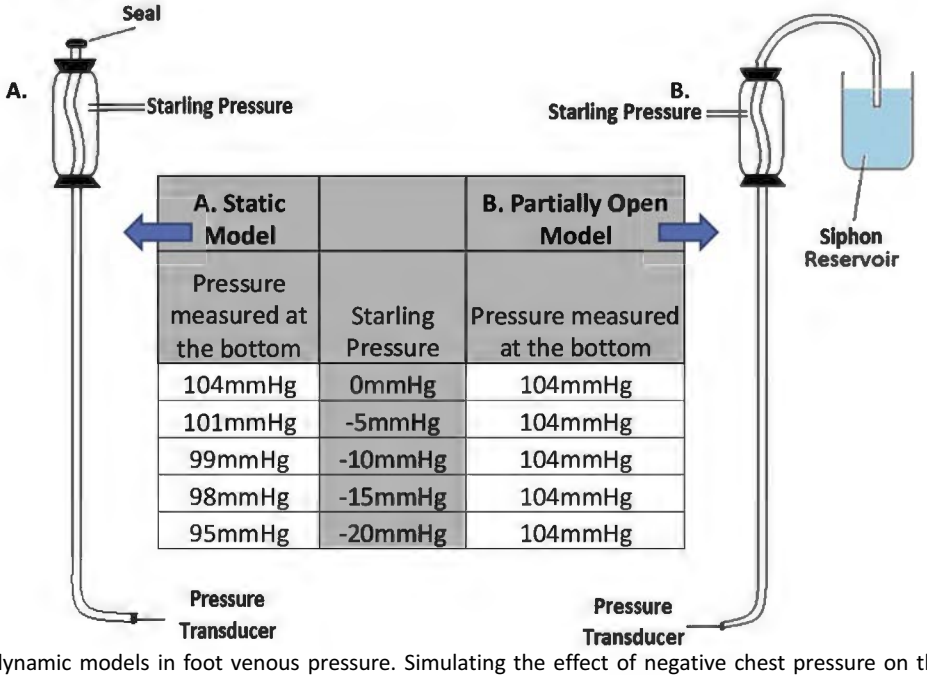
Bench flow model

Foot venous pressure in orthostasis. A static closed model, a static open model, and a dynamic model were used in these experiments as shown in Figs. 2 and 3.

In the closed static model, the Penrose sealed at both ends passed through a Starling resistor (chest) near its upper end. The pressure at the lower end read $\approx$100 mmHg, similar to erect foot venous pressure. A range of negative Starling pressure was applied to the Penrose simulating negative pressure in the chest. A pressure drop of 3–9 mmHg with incremental negative pressure in the Starling resistor as shown (see Fig. 2) was observed at the lower end, consistent with current "dead column" theory.

The experiment was repeated in a static open model with the upper end of the Penrose connected to a siphon allowing ingress or egress of fluid in response to external pressure changes. There was no pressure drop at all in this set-up at the lower end when negative Starling pressure was applied. Water from the siphon tank flowed into the Penrose cancelling the negative pressure created. While different from the previous static model, the results do not explain the in vivo discrepancy between observed column height and the pressure recorded at the foot level.

In the erect human, the caval flow is not unidirectional as in the previous model but consists of two different flows streaming in opposite directions in the superior and inferior



A. Static Model		B. Partially Open Model
Pressure measured at the bottom	Starling Pressure	Pressure measured at the bottom
104mmHg	0mmHg	104mmHg
101mmHg	-5mmHg	104mmHg
99mmHg	-10mmHg	104mmHg
98mmHg	-15mmHg	104mmHg
95mmHg	-20mmHg	104mmHg

Figure 2. Static and dynamic models in foot venous pressure. Simulating the effect of negative chest pressure on the measured venous pressure of the foot. Negative pressure applied to the Starling resistor near the top in a closed static model (A) significantly lowers foot venous pressure from resting levels (up to 9 mmHg). This is consistent with the current concept but does not correctly explain in vivo pressure discrepancy. With the siphon arrangement (B), the space created in the Penrose by the negative Starling pressure is quickly filled from the siphon. Pressure at the bottom remains unchanged.

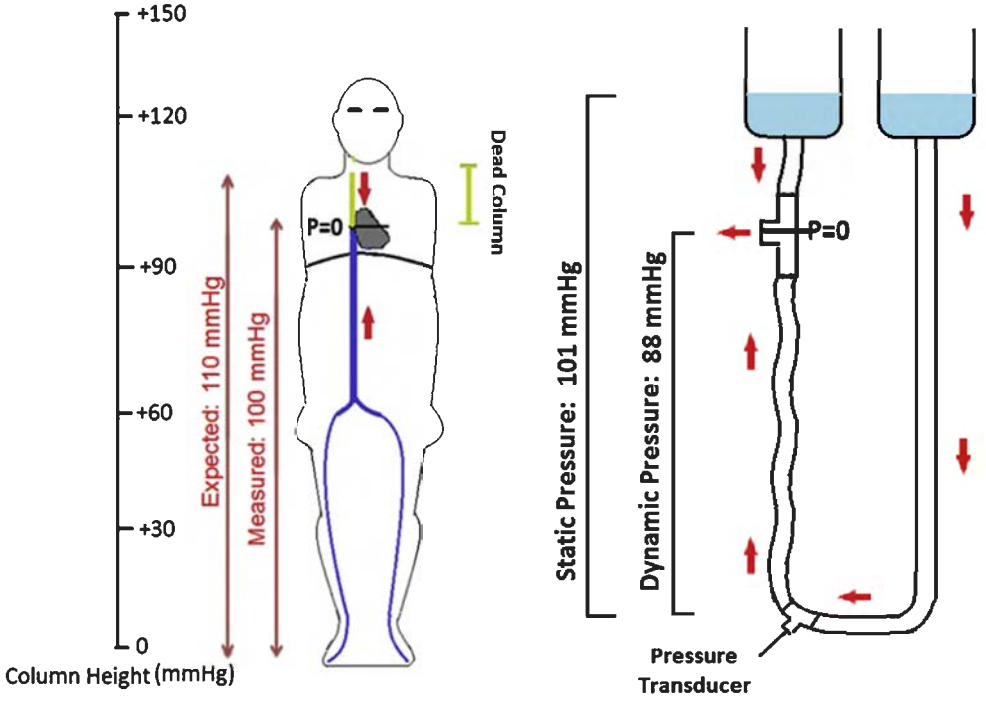


Figure 3. Discrepancy between foot venous pressure and column height. The venous pressure measured at the foot level is 5—10 mmHg less than expected from the height of the venous column in the erect position. Thus, a portion of the venous column at the upper end appears "dead" as it does not register at the foot level (left). A fully dynamic model more in line with in vivo flow conditions is shown on the right. The flow in the superior and inferior vena cava are in opposite directions fed by respective feeder tanks. Both flows exit through the side-arm of a "T," representing the heart. The pressure represented by the column in the superior vena cava does not register at the bottom of the set-up explaining the discrepancy between total column height and measured pressure. Pressure at the "T" measured 0 mmHg, similar to the zero pressure level at the heart.

vena cava, respectively (Fig. 3). To simulate opposite directional flows occurring in vivo, a dynamic model was devised (Fig. 3): water from two header tanks (one in a U tube configuration and the other from a siphon) maintained flow in the Penrose in opposite directions. Both flows exited through the large bore (ID 5/8″) side-arm of a 'T' connector "heart" mounted towards the upper end of the Penrose in place of the Starling resistor. The large bore side-arm ensured no restriction of flow and the pressure in the outlet open to the atmosphere measured zero. Once both flows were established, flow was momentarily stopped, cutting off flow from both tanks and closing the side-arm of the "T." Static pressure at the bottom "foot" end of the set-up was read at 101 mmHg representing the full motionless column height. When flow was resumed with the opposing flows exiting through the side-arm of the "T," the "foot" pressure dropped to 88 mmHg, representing only the column height to the "T" (heart).

This experiment shows that in a dynamic setting, the column pressure represented by the flow in the opposite direction above the "heart" will not be reflected at the foot level and appears as a "dead column."

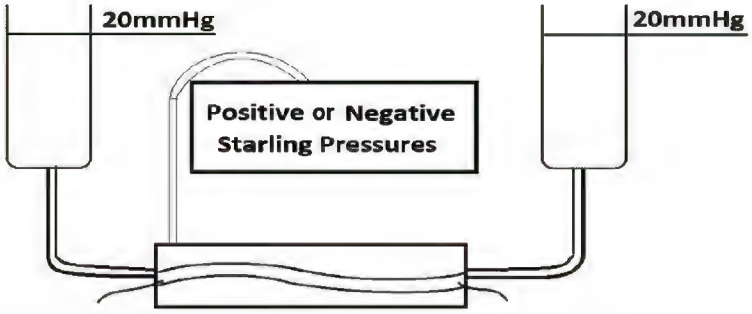
Transmural pressure and internal pressure. Current concepts in this arena are best illustrated by a static model as shown in Fig. 4. Internal Penrose pressure is maintained at 20 mmHg through input and output reservoirs both set at this level. This internal pressure remains unchanged regardless of negative or positive external pressure (up to point of collapse at 20 mmHg) (Fig. 4). As a result, the calculated transmural pressure (external minus internal pressure) declines with increasing external pressure and increases with negative external pressure.

Although consistent with current concepts, the static model is untenable; a dynamic model better represents in vivo conditions.

Dynamic model

The basic dynamic flow model with pressure settings shown in Fig. 1 was used. The effect of incremental range of both positive and negative pressures on internal pressures and flows is shown in Fig. 5.

Positive Starling pressure. With increasing external Starling pressures, the flow rate decreases progressively, whereas internal Penrose pressure rises stepwise with Starling external pressure. This is because of progressive conversion of flow velocity to pressure energy (not possible in a static model) as per the Bernoulli theorem.[9] The net result is that the calculated transmural pressure shows only minor change until flow has slowed considerably. When the Starling pressure is raised to 15 mmHg, flow dramatically slows with insufficient velocity energy left for conversion to pressure, resulting in a fall in transmural pressure. When external pressure exceeds the 20 mmHg input threshold, the Penrose collapses into a dumbbell in cross-section with



Starling Pressure	Internal Pressure (mmHg)	Transmural Pressure (mmHg)
+30mmHg	Collapsed	Collapsed
+20mmHg	Collapsed	Collapsed
+15mmHg	20	5
+10mmHg	20	10
+5mmHg	20	15
0mmHg	20	20
-5mmHg	21	26
-10mmHg	21	31
-15mmHg	21	36
-20mmHg	21	41

Figure 4. Transmural pressure in a static model. In this static model, there is no flow. Internal pressure remains unchanged up to 15 mmHg Starling pressure. The Penrose collapses at higher Starling pressures. Calculated transmural pressure progressively decreases as shown in the right column. Such a reduction in transmural pressure is a central prevailing concept. Negative external pressure prevents the Penrose from collapsing and the internal pressure remains unchanged, but calculated transmural pressure increases. Penrose volume changes with positive and negative Starling pressure are shown in Table 1.

microchannels at transverse ends; there is no recordable flow in the collapsed center and the internal and transmural pressures are unmeasurable.

Negative Starling pressure. In tested ranges, negative Starling pressures had a trivial increase on internal pressure (≈ 1 mmHg), but quantity of flow remained unchanged. Of note, internal pressure remained positive.

Transmural pressure in orthostasis. A "U" tube arrangement with "arterial" input 20 mmHg higher than the venous side was used (Fig. 6). A vertical Starling resistor was mounted on the venous limb ≈25 cm from the bottom of the "U" simulating compression (e.g., stockings) at the level of the human calf. The set-up added ≈85 mmHg gravity component at the lower end of the Penrose in the Starling resistor. Incremental Starling pressure had no effect on

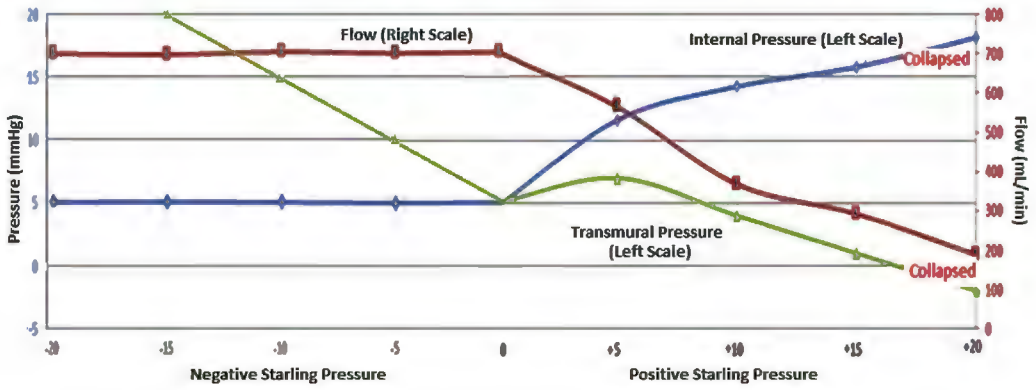


Figure 5. Effect of positive and negative external pressure in a dynamic model. With increasing positive external (Starling) pressures, the flow rate progressively decreases and the internal pressure progressively increases. This is because of conversion of velocity into pressure energy (Bernoulli theorem). The transmural pressure remains roughly unchanged until the Penrose collapses from high external pressures and collapses in the form of a dumbbell in cross-section. There are two microchannels at either end of the collapsed center. A trickle of flow continues through these corner microchannels. With increasing negative external pressures, there is little change in internal pressure or flow.

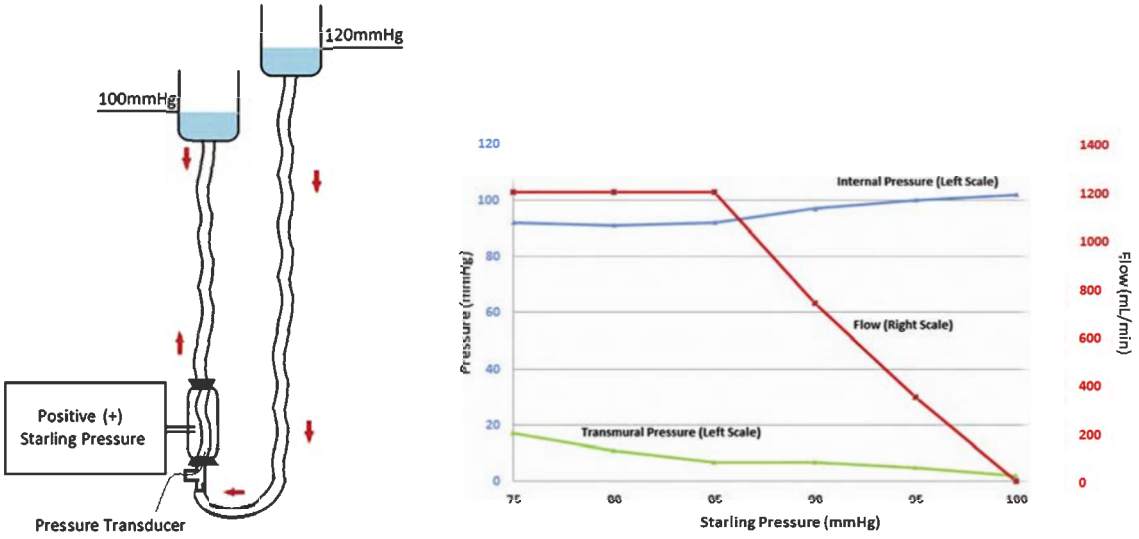


Figure 6. Transmural pressure in orthostasis. A "U" tube arrangement (left) added ≈85 mmHg of gravity pressure to the Penrose in the Starling chamber, which was mounted at the level of the calf. Incremental Starling pressures had no effect until the gravity component was exceeded (graph to the right) on flow, internal pressure (starting value 91 mmHg), and transmural pressure. Further increase in Starling pressure resulted in a sharp decline in flow and an increase in internal pressure because of conversion of flow energy into pressure (Bernoulli). Because of the increase in internal pressure, transmural pressure remains stable as in the horizontal model shown in Fig. 5.

Penrose internal pressure or flow until the external pressure in the resistor exceeded the gravity component of 85 mmHg; further increase in Starling pressure resulted in an increase in internal pressure with slowing of flow velocity (stable transmural pressure) similar to that seen in the horizontal set-up described above.

Penrose volume changes

The dynamic flow model was slightly modified to monitor Penrose volume changes occurring with both positive and negative Starling pressure changes (Fig. 7). A narrow bore graduated water manometer of negligible capacity (<2 cc) was used as the pressurizing head tank for positive Starling pressure. For negative Starling pressure, the chest tube suction set-up was connected to the upper end of the manometer to deliver known negative pressure. Zero Penrose volume was established with the Penrose completely collapsed flat by applying 30 mmHg Starling pressure (highest pressure tested) and the input and output reservoirs disconnected. Flow was then allowed to occur with the reservoirs reconnected at standard inflow/outflow settings with varying positive and negative pressures. When flow occurred, the Penrose expanded displacing an equal amount from the Starling chamber that could be measured by withdrawing the overflow (while maintaining the manometer level unchanged) into a graduated syringe connected to the bottom of the manometer through a three-way stopcock. Penrose volume changes at various settings are shown in Table 1. With increasing positive Starling pressures, Penrose volume declined roughly parallel with flow volume but had no correlation to transmural pressure as internal pressure rose to keep it roughly constant.

Negative Starling pressure expands the tube to the extent allowed by the stretchability and elastic wall properties of the tube. There was a slight (3—8%) increase in Penrose volume and a small increase (≈ 1 mmHg; NS.) in internal pressure from slowing velocity. Tube expansion quickly levels off with as little as −5 mm negative external pressure. Incremental negative pressure beyond this level is progressively less effective in tube expansion because of parabolic increase in resistance to expansion by wall tensile properties. The external negative pressure simply converts the Penrose into a functionally stiff tube at this stage. The flow channel is somewhat larger (Table 1), the velocity likely somewhat slower (not measured), and the internal pressure slightly higher because of slowing velocity, but quantity of flow remains unchanged (Fig. 5, Table 1).

Air embolism and internal pressure. Experiments described herein and in vivo pressure measurements show that persistent negative pressures in systemic chest veins likely do not occur. Experiments described in Fig. 8 show that negative pressure at the lower end of the Penrose is not necessary to favor air embolism. Using transparent collapsible polyethylene tubing, an air pocket was seen to form around the side-hole narrowing the flow stream providing a Venturi effect for suction of air.

DISCUSSION

The key findings in the experimental model are (a) external pressure does not reduce transmural pressure, but reduces flow velocity to maintain transmural pressure constant over a range of external pressures; (b) negative external pressure has little effect on internal flow or pressure, which does not become negative but remains positive. These conclusions are strictly valid only for the experimental model. Some inferences to human physiology are possible subject to limitations. These are elaborated below.

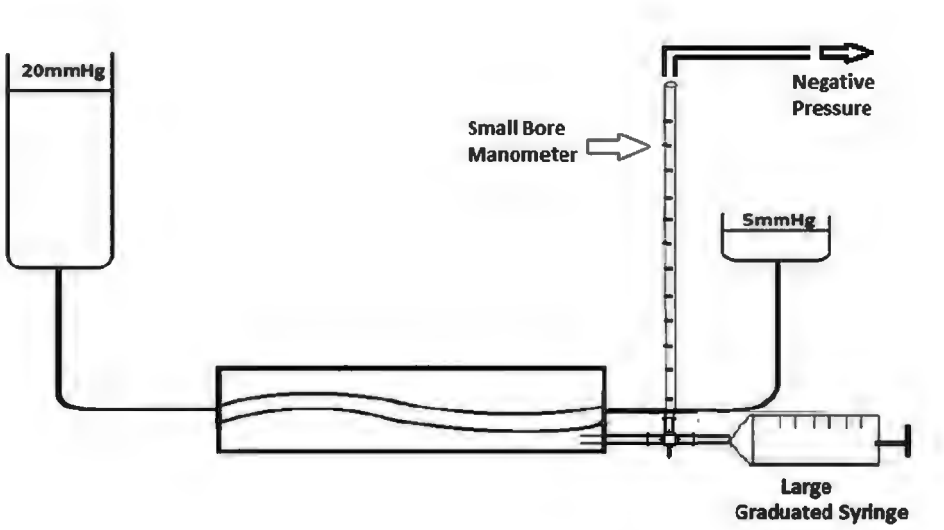


Figure 7. Penrose volume changes in the Starling resistor. A small bore manometer of negligible capacity is used as a pressurizing tank for positive pressure and as a connector between the Starling chamber and the chest suction set-up for applying desired negative Starling pressures. Baseline Starling chamber volume is established with the Penrose completely collapsed. Resumption of flow in the Penrose will lead to expansion of the Penrose and a reciprocal contraction of Starling chamber volume. These volume changes can be monitored by noting the measured volume required to be injected or withdrawn from the chamber while maintaining the manometer level constant for a particular Starling pressure setting.

Transmural pressure

The effect of external pressure on fluid pressure contained in a collapsible tube will vary if the tube ends are closed or not and if the fluid is continuous motion or not. If the ends are closed, internal pressure will rise and fall with applied positive and negative external pressures respectively (see Fig. 2 for negative pressure effect). If at least one end of the tube is open, the stationary fluid inside will egress or ingress in response to external positive and negative pressure, respectively; In either case, internal pressure will remain unchanged (see Fig. 4). If the fluid in the tube is in motion because of a gradient (Poiseuille flow), internal pressure responses to external pressure become complex, more so if gravity field is added. Many current concepts in this area are based on the notion that the venous circulation is part of a closed system with fixed volume/pressure relationships. It is argued that the venous circuit should be viewed as a dynamic open flow system, as modeled in Fig. 1.

Table 1. Penrose volume and flow changes with positive and negative Starling pressures.

Starling pressure, mmHg	Penrose volume, cc	Flow, mL/min
+30	2 (5%)[a]	2 (.3%)[a]
+20	6 (15%)	115 (17%)
+15	18 (46%)	245 (36%)
+10	26 (67%)	411 (60%)
+5	30 (77%)	685 (100%)
0	39 (100%)	686 (100%)[b]
−5	41 (105%)	672 (98%)
−10	42 (108%)	666 (97%)
−15	43 (108%)	665 (97%)
−17	44 (108%)	680 (99%)[b]

With increasing positive Starling pressures, Penrose volume progressively decreases roughly paralleling the decrease in flow. Negative Starling pressures result in only a small increase in Penrose volume (maximum 8%); there is no significant change in flow.

[a] At high external pressures, the Penrose collapses into a "dumbbell" with small tubular channels at each corner, allowing a trickle of flow.

[b] Not significantly different.

Venous system as an open dynamic model

The venous system is unique in that it is collapsible with a capacitance that can expand or contract on demand to fulfill its reservoir function. The energy and pressure required to recruit partially or fully collapsed parts of the system to full capacitance are very low.[10] Because of the non-linear compliance curve, internal pressure varies very little for a wide range of volumes, helping homeostatic stability. In essence, it behaves like an open system expanding or contracting as needed around the available blood volume. Positive and negative pressures that change conduit caliber in opposite directions often occur simultaneously in different sections (abdomen, chest, and limbs), offsetting each other on their volume impact. With a cardiac output of 5—6 L per minute or 80—100 cc per second, combined with the ability of the venous reservoir to contract or expand in compensatory fashion, it would seem that any net imbalance in volume/conduit size will be filled easily. Any such local volume change will show as variation in flow channel caliber as it is dynamic.

Role of the heart in maintaining venous volume

The heart appears to fill passively with blood that overflows the venous tank, that is the heart pump does not "suck" blood out of the venous system (ventricular diastole may have a minor suction effect) but merely empties the overflow from it like a sump pump.[11] Hence venous filling

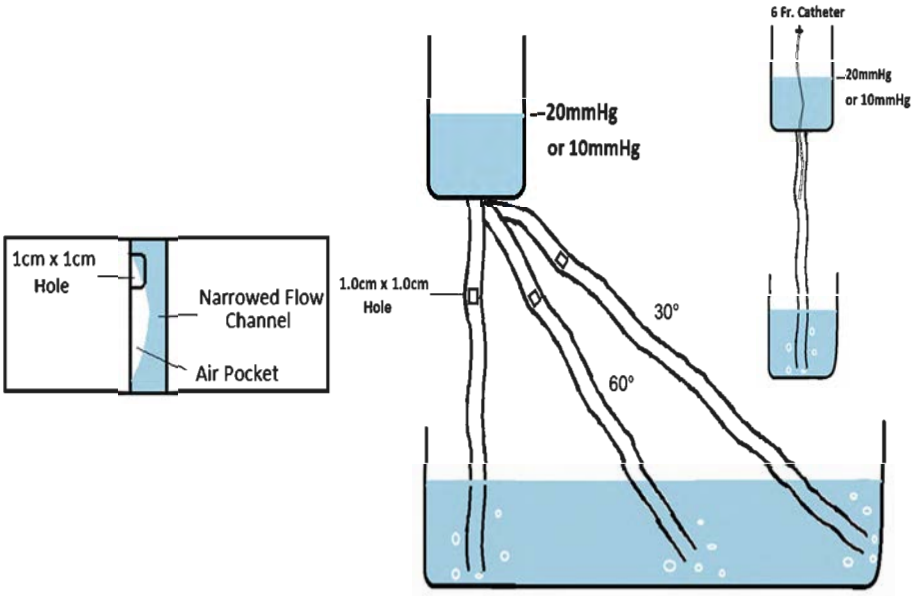


Figure 8. Air embolism. Flow was allowed to occur from a head tank with a pressure head of 20 mmHg through a long Penrose into a glass tank. The lower end of the Penrose was kept submerged $\approx$7 cm under water in the receiving tank. An aperture 1 cm $\times$ 1 cm was cut out from the side of the Penrose midway between the two ends. As flow occurred in the Penrose large amounts of air were sucked in from the side-hole which could be visually seen discharging from the lower end. Similar results were obtained with a 10 mmHg pressure head and also when the Penrose was vertical, slanted at 60° or even when slanted only 30° from the horizontal. In a companion experiment (right), a 6 Fr catheter (similar to a central line) was inserted through the head tank into the Penrose without the side-hole. The upper end of the catheter was open to the atmosphere. Air embolism was also noted in this experiment. A Venturi type of mechanism appears to be responsible for sucking in air through the side-hole (left inset). A similar mechanism may occur around the catheter tip because of flow separation and flow acceleration.

controls cardiac output and not the reverse. This means that cardiac ejection per se will not produce negative pressure in chest veins because it pumps only overflow from the venous reservoir. As an overflow pump, it tends to maintain venous volume in balance with available venous capacitance. Thus, the action of the heart and the compliance characteristics of venous conduits effectively convert it to an open system. The regional volume changes in orthostasis are often described as "displacement" of volume from above as though it is a motionless static column. Clearly, this is redistribution of regional flow channel sizes occurring in the context of voluminous flow.[1—3,12]

In the horizontal model, internal flow and pressure changes are different for positive and negative external Starling pressures, as shown in Fig. 5. This is because external pressures in the physiologic range can completely collapse the Penrose whereas expansion from negative external pressure will be limited (Table 1). The slowing of flow velocity in response to positive external pressure is a fundamental characteristic of collapsible tube flow demonstrated to occur also in vivo.[9,13—16] In the experimental set-up simulating orthostasis, flow and transmural pressure do not change until the external pressure exceeds the gravity component. Once the gravity component is exceeded, changes in internal and transmural pressures are identical to those in the horizontal dynamic model, shown in Figs. 1 and 5.

The current prevailing concept that application of positive external pressure results in a reduction of internal and transmural pressures (based on a static view of internal pressure, Fig. 4) is probably incorrect.

Is the pressure in systemic chest veins negative?

It is often stated or implied that venous pressure in chest veins is negative.[11,14,17—20] The occurrence of air embolism has reinforced this belief. Results of the model experiments and clinical observations suggest that this is unlikely except as a brief transient during deep inspiration.

Persistent negative internal pressures are possible only in a closed system. Transient negative pressures are possible in an open dynamic system if there is a lag between tube expansion from negative external pressure and the incoming flow to fill the expanding space. Because the Penrose is already "full" when negative external pressure is applied, the Penrose wall properties limit expansion to $<$10%. Expansion will be greater if the Penrose was in the collapsed state. Central veins in the chest are also observed to be "full" on intravascular ultrasound examination during endovenous interventions. Their capacitance and limits to their expansion are unknown, although compliance curves between canine thoracic vena cava and Penrose are nearly identical.[7]

Clinical measurements of central venous pressure can clarify this issue. Thoracic IVC pressures recorded during endovenous interventions and central venous pressures monitored through old-style water manometers are in the positive range as well, even during respiratory oscillations.

Observable jugular venous pulse in 45° tilt position seldom dips below the clavicle, indicating positive internal pressure above the heart level. Reported right atrial pressures in normal individuals are above zero (2—6 mm Hg).[21] Very brief transient atrial pressure dip below zero during atrial diastole and inspiration is possible as the prevailing pressure is close to zero. However, it is doubtful that central venous pressures in the chest ever become persistently negative in normovolemic individuals not in shock. As it is not possible to prove a negative by its absence in a small sample, a large sample size powered to obtain a high Z score in support is required to clarify this issue.

Mechanism of air embolism

As persistent negative pressure seems unlikely, it became necessary to explore alternative mechanisms. Experiments shown in Fig. 8 suggest that negative pressure in the chest veins is not necessary for air embolism, which can occur from a Venturi mechanism. Gravity flow in collapsible tubes is known to exert no lateral pressure,[22] hence no fluid spurts out of the side-hole in the set-up. Further studies to clarify the exact mechanism of air embolism in vivo are warranted. Neck and chest veins are held open by fascial attachments; a free hanging Penrose does not duplicate this anatomical feature.

Discrepancy between column height and foot venous pressure

In the dynamic orthostatic experiment (Fig. 3) the 'T' connector is the datum line for the gravity field, with flows in opposite directions meeting at the datum line. Only the gravity component of the column below the datum registers at the foot level in the experimental model, explaining the observed discrepancy between the total column height and the foot pressure.[1—3,12] Such a dynamic explanation was indeed offered by Clark previously but was ignored.[23]

Study limitations

The results described herein are from an experimental model using Penrose tubing to simulate venous flow. Although substantially similar in fluid mechanical behavior, important differences remain. For example, Penrose collapses into a dumbbell shape in cross-section with two microchannels at each transverse end, while venous collapse is more concentric.[7] The pressure volume curve is less non-linear. Wall properties show substantial regional variations.[24] Importantly, systemic veins are tethered at multiple points to fascia, muscle, and to multiple tributaries, unlike the Penrose hung between two end connectors in this experiment. These factors may significantly modify collapse/expansion behavior, influencing results of some experiments (e.g., air embolism).[25] Tube expansion with negative external pressure, especially with deep inspiration, may be more substantial in human chest veins than in the stiffer Penrose used here. Tube collapse in the Starling set-up is not uniform; regional collapse can occur depending on certain combinations of Starling/output pressure settings. Anomalous flow behavior resembling a "waterfall" has been shown to occur in the collapsed segment which has been postulated to occur also in vivo, particularly in the pulmonary circulation. Interested readers may find detailed treatment of this subject in the seminal works of Holt, Shapiro, and Fung.[10,26—28]

Despite these biological differences from the experimental model, the authors view current results as a valid qualitative framework for understanding some aspects of venous flow, but in vivo validation is required, especially of hypotheses contrary to traditional explanations. For example, venous flow and pressure changes in response to external pressure (e.g., abdominal compression suit; compression stockings) are apt subjects for human study. The question of negative pressure in systemic chest veins is easily resolved with modern more accurate transducers (electronic zero) and larger numbers for statistical proof.

CONFLICT OF INTEREST

None.

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