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Original Article

Pathogenetic mechanisms of venous congestion after the Fontan procedure

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Abstract *Background:* The hemodynamic status after a Fontan type procedure for definitive palliation of functionally univentricular hearts is dominated by a high central venous pressure, which seems to be one of several factors responsible for venous congestion appearing as a frequent complication in the early and late postoperative course. The purpose of our study was to find other hemodynamic parameters correlating with the presence of venous congestion and effusions in these patients. *Methods:* We compared the hemodynamic data of 18 patients who had an uneventful long-term course after a Fontan type procedure with the respective data of 10 patients who developed symptoms of venous congestion in the immediate postoperative period. Based on a theoretical model, we developed an algorithm to calculate mean hydrostatic capillary pressure from mean arterial pressure, systemic vascular resistance index and central venous pressure. *Results:* Pulmonary vascular resistance index (2.1± 1.0 mmHg L-1 min m2), mean left atrial pressure (9.7± 4.0 mmHg) and cardiac index (3.6± 0.6 l/min/m2) are mainly normal in patients with venous congestion in the immediate postoperative period, but mean hydrostatic capillary pressure is significantly higher compared to patients without venous congestion (24.3± 3.1 vs 18.3± 4.0 mmHg). Lower mean hydrostatic capillary pressures in these patients are due to a highly significant increase of systemic vascular resistance index (18.6± 4.2 versus 33.6± 6.6 mmHg L-1 min m2) and a concomitant decrease of cardiac index to 2.4± 0.3 l/min/m2. *Conclusions:* The increase of mean hydrostatic capillary pressure, caused by high central venous pressures but also by relatively low systemic vascular resistance indexes, seems to be the hemodynamic key parameter responsible for venous congestion and effusions in patients after a Fontan type procedure in the immediate postoperative period.

Keywords: Congenital heart disease; congestive heart failure; Fontan operation; microcirculation; medical therapy

After a Fontan type procedure for definitive palliation of functionally univentricular hearts, the circulation of blood depends on high central venous pressures required for an effective transpulmonary gradient, thus generating an adequate flow into the lungs in the absence of a subpulmonary pumping chamber.[1,2] The increase of central venous pressure seems to be responsible for venous congestion occurring as a frequent complication in the early and late postoperative course.[3,4] On the other hand, significant correlations between chronic effusions and absolute levels of central venous pressure have not been consistently observed.[5] The purpose of this study was to reevaluate other hemodynamic parameters possibly correlating with the presence of venous congestion in these patients.

Thus, we compared hemodynamic data of patients who did not develop signs of venous congestion in their long-term course after a modified Fontan procedure with the corresponding data from patients with effusions in the early or late postoperative course. Focusing on the crucial role of mean hydrostatic capillary pressure in the pathogenesis of edema,[6,7,8] we used an algorithm for its calculation from mean arterial pressure, central

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venous pressure, and sytemic vascular resistance based on Starling's theories concerning ultrafiltration in the capillaries.[9]

Patients and methods

At our institution, the Fontan procedure was established in 1975[10,11,12] and a total number of 110 patients have now undergone this operation or, since 1990, a total cavopulmonary connection[13] as the presently preferred surgical technique. Cardiac catheterization was performed for clinical reasons in 24 patients, 1–14 years postoperatively. We used data from these hemodynamic evaluations for further analysis in this study. The patients are grouped into a subset of 18 patients without signs of venous congestion in the long-term course, having a mean age of 15.5 years, and a second set of 6 patients suffering from protein-losing enteropathy with diuretic resistant pleural effusions and ascites. The latter group with late congestion had a mean age of 19.2 years.

Cardiac output was calculated by the Fick method in the earlier experience, but has been double checked by thermodilution and dye-dilution techniques since 1991 (COLD™system, Pulsion Medical System, Munic), for which we were able to reproduce the previously reported good correlation between calculated and measured cardiac outputs.[14] For the purpose of these measurements, we performed central venous injections of cold Cardiogreen solution and detected the dilution curves with a fiberoptic catheter placed in the aorta.[15,16,17] Left atrial pressures were estimated from pulmonary capillary wedge pressure readings. In addition, a group of 10 patients was enrolled to evaluate early postoperative hemodynamic constellations. We had the opportunity to use data acquired in the intensive care unit setting from a prospective clinical study of patients after total cavopulmonary connection.[16] At this early stage, all patients had venous congestion, with an average of 470 ml/24 hours chest tube drainage. This group with early congestion had a mean age of 8.5 years. Cardiac output was measured 3 hours after weaning from the respirator by thermodilution. Mean arterial pressure, central venous pressure, and left atrial pressure were recorded simultaneously. Capillary leakage syndrome was excluded clinically in view of normal values for gas exchange and in the absence of any need to use vasoconstrictors. Clinical data of all parients are listed in detail in Table 1.

As a control cohort, we used hemodynamic data from 18 patiets studied in our catheterization laboratory who had mild abnormalities, such as pulmonary or aortic stenoses, or small shunts, for which an intervention was not warranted. This control group had a mean age of 7.5 years.

Statistical analysis was carried out using Student's t-test, comparing selected hemodynamic parameters from the groups with either no congestion or early congestion. The group with late congestion, having only 6 patients, was too small for statistical evaluation.

Calculated hemodynamic parameters were derived according to the following equations (the algorithm for equation 3 is derived in the appendix):

- Systemic vascular resistance index
  SVRI = (MAP – CVP) / CI [mmHg L-1 min m2 = 80dyn s cm-5 m2 = 8 kPa L-1 s m2]
- Pulmonary vascular resistance index
  PVRI = (CVP – LAP) / CI [mmHg L-1 min m2 = 80dyn s cm-5 m2 = 8 kPa L-1 s m2]
- Minimal mean hydrostatic capillary pressure

Pcap(min)) = [2 mmHg L-1 min m2 × MAP + CVP(SVRI – 2 mmHg L-1 min m2)]/SVRI [mmHg]

Results

Central venous pressure and mean arterial pressure

Central venous pressure was increased in all patients after a Fontan type procedure (Fig. 1) as compared to controls. In patients without venous congestion, we found significantly lower values for central venous pressure (13.4 ± 3.8 mmHg) in comparison to patients with venous congestion in the early (17.1 ± 2.9 mmHg) or late (17.2 ± 2.9 mmHg) postoperative course. Despite this difference, there is a wide overlap of central venous pressure values between these 3 groups.

Mean arterial pressures of 82 mmHg in the early postoperative course were normal but significantly lower than in the late postoperative group without congestion (93 ± 11 mmHg). As shown in Table 1, three patients developed arterial hypertension after a total cavopulmonary connection later requiring medical therapy.

Cardiac Index

Using the Fick method in patients both with and without venous congestion in the late postoperative course after a Fontan type procedure, we found markedly reduced values for cardiac index (2.5 and 2.4 l/min/m2) in comparison to controls.

In the early postoperative course measured cardiac indexes were in the low normal range (3.6 ± 0.6 l/min/m2), but significantly higher than those of the late postoperative patients.

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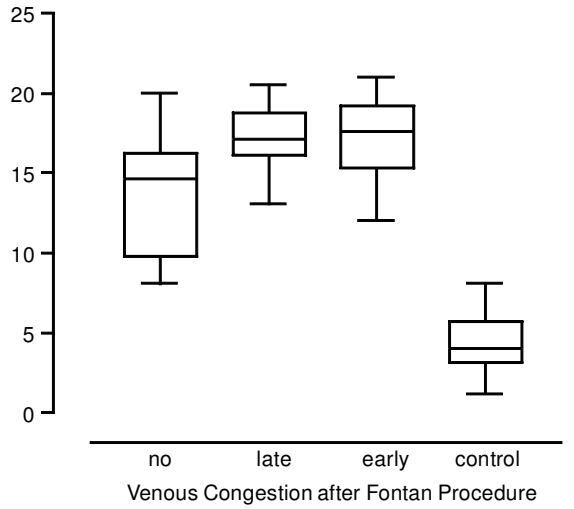

Figure 1.
Values for central venous pressure in patients after a Fontan procedure without and with symptoms of venous congestion in the early or late postoperative period in comparison to a control group. Median, minimum, maximum, 25% and 75% percentile are shown by box and whiskers.

Left atrial pressure and pulmonary capillary wedge pressure

Mean values for left atrial pressure or pulmonary capillary wedge pressure were in the high normal range in all groups of patients, while a few patients showed increased values up to 14mmHg. We found no significant differences for left atrial pressure or pulmonary capillary wedge pressure between the control group (9.2 ± 2.2 mmHg) and patients after a Fontan type procedure in the early (9.7 ± 4.0 mmHg) or late (8.0 ± 2.5 mmHg) postoperative course (Table 2).

Vascular resistances

The mean value for pulmonary vascular resistance indexes is normal in patients after a Fontan type procedure, and there is no significant difference between patients with venous congestion in the early postoperative course and patients without venous congestion in the late postoperative course (Table 2). Only those 6 patients with protein-losing enteropathy in the late postoperative course are showing higher values for pulmonary vascular resistance index (3.3 ± 1.0 mmHg L-1 min m2).

The most impressive difference between patients with early postoperative congestion and those without congestion in the long-term course is seen in respect to their systemic vascular resistance index, which is nearly twice as high in patients without congestion (33.6 ± 6.6 mmHg L-1 min m2 vs 18.6 ± 4.2 mmHg L-1 min m2).

Minimal mean hydrostatic capillary pressure

The calculated minimal mean hydrostatic capillary pressure values in Fontan patients without congestion (18.3 ± 4.0mmHg) were significantly lower than those in patients with congestion after a Fontan type procedure in the early (24.3 ± 3.1 mmHg) and late (22.2 ± 2.9 mmHg) postoperative course. Above a cut-off line of 21 mmHg, virtually all patients developed congestion, whereas those below that level did not (Fig. 2).

Discussion

Facing the problems of venous congestion in a substantial number of patients with a Fontan circulation, many investigators have pointed out the important role of increased venous pressures, possibly due to increased pulmonary vascular resistance or an impaired ventricular function of the more or less hypertrophied dominant ventricle.

It was not the aim of our study to deny these already well known facts. Moreover, we do not intend to compare groups of patients who, in many respects, are uncomparable, because of the differences in recruitment to the different groups, the design of the study, selection criterions, and the eras

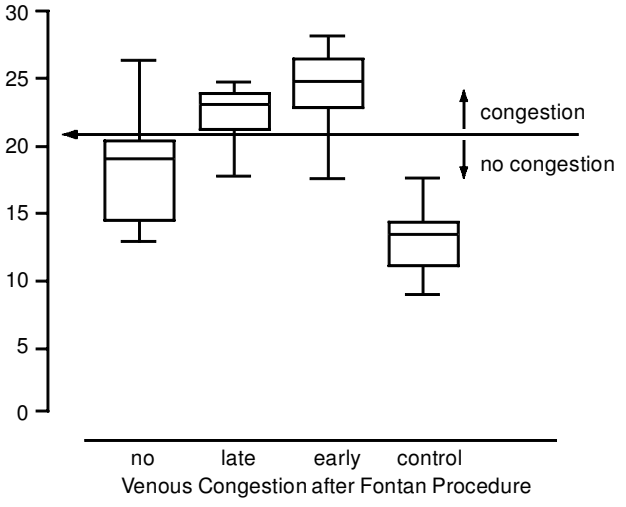

Figure 2.
Calculated values of minimal mean hydrostatic capillary pressures (the algorithm is derived in the appendix) in patients after a Fontan procedure without (group 1) and with (group 2 and 3) symptoms of venous congestion in the early or late postoperative period in comparison to a control group. Median, minimum, maximum, 25% and 75% percentile are shown by box and whiskers. More than 75% of patients developed congestion above a cut-off line of 21 mmHg.

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Table 1. Clinical profile of the patient groups

Patient group	Diagnosis	Surgical Procedure	Clinical Status
No congestion:	DILV	RA-PA An	SV-Tc
	DILV	TCC	systemic hypertension
	TA Ib	TCC	systemic hypertension
	DILV	TCC	AF,PM
	UVH (right morphology)	RA-PA Cd	AF
	UVH (mixed morphology)	TCC	AF
	UVH (left morphology)	RA-PA Cd	AF
	TA Ib	RA-PA Hanc	alive + well
	UVH (left morphology)	RA-PA An	SV-Tc
	DORV, AVSD	TCC	AF,PM
	UVH (left morphology)	RA-PA Cd	alive + well
	DILV	RA-PA Cd	pulmonary embolism
	PA/VSD	TCC	systemic hypertension
	PA/IVS	TCC	alive + well
	TA Ib	TCC	SSS,PM
	TA IIb	RA-PA Cd	alive + well
	TA Ib	TCC	SV -Tc
	DILV	RA-PA-Cd	AF
Late Congestion:	TA Ib	RA-PA Cd	PLE, died
	Hypoplastic RV	RA-RV Hanc	PLE, died
	UVH (left morphology)	RA-PA Cd	PLE, died
	TA Ib	RA-RV Cd	PLE, died
	TA Ib	RA-PA Hanc	PLE, died
	DILV	RA-PA An	PLE, died
Early Congestion:	UVH (left morphology)	TCC	alive + well
	DILV, MA	TCC	alive + well
	Criss-Cross-Heart	TCC	alive + well
	DILV	TCC	alive + well
	TA Ib	TCC	alive + well
	PA,VSD	TCC	alive + well
	DILV	TCC	systemic hypertension
	DILV	TCC	alive + well
	DILV,MA	TCC	alive + well
	Criss-Cross-Heart	TCC	alive + well

DILV = typical double inlet left ventricle, TA = tricuspid valve atresia, UVH = functional univentricular heart, DORV = Double outlet right venticle, AVSD = atrioventricular septal defect, PA = pulmonary valve atresia, VSD = ventricular septal defect, IVS = intact ventricular septum, MA = mitral valve atresia, TCC = total cavopulmonary connection, RA-PA An = right atrial to pulmonary anastomosis, RA-PA Cd = right atrial to pulmonary valveless conduit, RA-PA Hanc = right atrial to pulmonary Hancock conduit, SV-Tc = supraventricular tachycardia, AF = atrial flutter, PM = pacemaker, SSS = sick sinus syndrome, PLE = protein loosing enteropathy

of surgical and intensive care. All of our patients have one thing in common, nonetheless, a Fontan-type circulation. One group does not have signs of venous congestion, whereas the two other groups have effusions in the early or late postoperative course. Our aim was not to compare patients, but simply to look closely at hemodynamic constellations in either presence or absence of venous congestion in patients after the Fontan-type procedure. We focused on the key parameters of circulation, namely the flow, pressures and resistances upstream, within and downstream of the capillary bed, because it is due to an imbalance of these physical forces that excess fluid accumulates outside the vascular bed (Fig. 3).

In accordance with previous studies[18,19] we did not find any difference in cardiac indexes between patients with or without venous congestion in the long-term postoperative course. On the other hand, we confirmed the observation of others, that higher central venous pressures go along with the complication of effusions. Widely overlapping values indicate, however, that other determinants must contribute to these complications.

Based on our results, the significantly increased central venous pressure in the early postoperative course after a Fontan type procedure is not related to a significantly higher pulmonary vascular resistance. Also, ventricular impedance values, expressed as the ratio of left atrial pressure to cardiac index, in patients with early congestion after a Fontan operation are not different from those of the 18 patients without congestion in the late postoperative course (2.8 ± 1.2 mmHg L-1 min m2 vs 3.4 ± 1.2 mmHg L-1 min m2) . In accordance with Ohm's law [CVP = (PVRI +

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Table 2: Hemodynamic data and calculated mean capillary filtration pressure after Fontan procedure

	CVP	TPRI	PVRI	PCWP,LAP	CI	SVRI	MAP	Pcap
	[mmHg]	[dyn s cm^{-5} /m^2]	[dyn s cm^{-5} /m^2]	[mmHg]	[l min^{-1}/ m^2]	[dyn s cm^{-5}/m^2]	[mmHg]	[mmHg]
early congestion	17.1 ± 2.9	390 ± 102	168 ± 79	9.7 ± 4.0	3.6 ± 0.6	1486 ± 340	82 ± 11	24.3 ± 3.1
no congestion	13.4 ± 3.8	450 ± 134	181 ± 94	8.0 ± 2.5	2.4 ± 0.34	2687 ± 527	93 ± 11	18.3 ± 4.0
significance	p< 0,01	n.s.	n.s.	P< 0,05	P< 0,01	P< 0,0001	P< 0,05	P< 0,001
late congestion	17.2 ± 2.9	552 ± 131	267 ± 81	7.3 ± 4.9	2.5 ± 0.34	2023 ± 393	80 ± 10	22.2 ± 2.9
control	4,3 ± 2,3	283 ± 61	108 ± 63	9.2 ± 2.2	4.4 ± 1.0	1484 ± 477	81 ± 11	13.2 ± 2.4

CVP = central venous pressure, TPRI = total pulmonary resistance index, PVRI = pulmonary vascular resistance index, PCWP = pulmonary capillary wedge pressure, LAP = left atrial pressure, CI = cardiac index, SVRI = systemic vascular resistance index, MAP = mean arterial pressure, P_{cap} = mean capillary filtration pressure

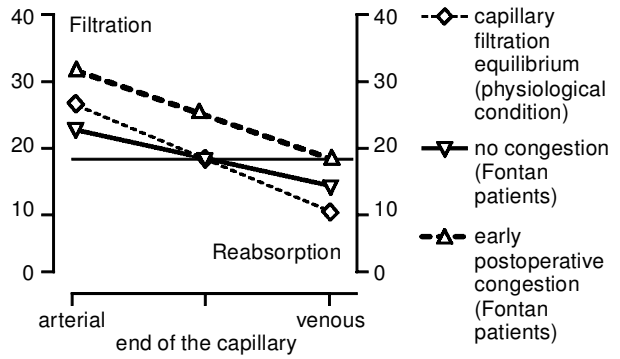

Figure 3:
Simple model of hydrostatic pressure decrease in the capillaries. Average data of Fontan patients with and without venous congestion at the arterial and venous end of the capillaries were calculated with the algorithm, derived in the appendix and compared with physiologic conditions. The physiologic capillary filtration equilibrium" based on following assumptions: plasma oncotic pressure = 18 mmHg, capillary resistance index = 4 mmHg L^{-1} min m^2, normal Cardiac index = 4 l/min/m^2, postcapillary resistance = 0 mmHg L^{-1} min m^2. Patients without congestion after a Fontan procedure have higher hydrostatic pressures at the venous end of their capillaries due to higher central venous pressures. Capillary filtration equilibrium" was attained by reducing the hydrostatic pressure at the arterial end of the capillary due to higher systemic vascular resistances. As a consequence these patients have lower cardiac indices. Patients with congestion after a Fontan procedure have higher hydrostatic pressures both at the venous and arterial end of their capillaries. If the plasma oncotic pressure remains constant filtration predominate reabsorption with fluid accumulation in the lymphatic system.

Abbreviations for hemodynamic parameters:
CI = cardiac index
CVP = central venous pressure
LAP = left atrial pressure
MAP = mean arterial pressure
P_{art} = *hydrostatic pressure at the arterial end of the capillary*
P_{cap} = *mean hydrostatic capillary pressure*
$P_{cap(min)}$ = *minimal mean hydrostatic capillary pressure*
P_{ven} = *hydrostatic pressure at the venous end of the capillary*
PCWP = pulmonary capillary wedge pressure
PVRI = pulmonary vascular resistance index
SVRI = systemic vascular resistance index

LAP/CI) × CI} it therefore seems possible that the significantly higher cardiac indexes in the early postoperative period may also cause higher central venous pressures. Undoubtely, relatively high cardiac indexes are necessary to sustain mean arterial pressure, because in this situation systemic vascular resistance is nearly half as high compared to patients without congestion in the long-term course after a Fontan type procedure.

Our data identify the increased mean hydrostatic capillary pressure as the main hemodynamic cause for effusions in patients after a total cavopulmonary connection. Indeed, nearly all patients with a calculated mean hydrostatic capillary pressure of greater than 21 mmHg developed symptomatic venous congestion as shown in Figure 2. Whereas our calculated values for minimal mean hydrostatic capillary pressure of 24.3 ± 3.1 mmHg in patients with early congestion would be well within the physiologic range for experimentally measured mean hydrostatic capillary pressures of 23 – 25 mmHg, real values in this group are likely to be considerably above the normal range. Real values for mean hydrostatic capillary pressures include the pressure difference between the venous end of the capillary and the central venous pressure. This value cannot be measured in a clinical setting. To support this observation, calculated minimal mean hydrostatic capillary pressures in our control group are significantly below the normal range for true mean hydrostatic capillary pressure.

As shown in Figure 3, patients without clinical symptoms of venous congestion after a Fontan procedure seem to be in a capillary filtration equilibrium", with reduced hydrostatic pressures at the arterial end due to a high systemic arteriolar resistance. Normal ejection fractions and left atrial pressures are evidence for a normal ventricular function in the majority of these patients, who obviously tolerate a higher afterload due to higher systemic vascular resistances without alterations of systolic function, in contrast to patients with myocardial damage.

Patients with early congestion after the Fontan operation had elevated hydrostatic pressures across the whole length of the capillary bed, and thus did not reach filtration equilibrium. This increase in capillary filtration pressure causes excessive filtration in the interstitial space as reabsorption capacity is limited. Fluid that cannot be reabsorbed has to be drained by the lymphatic system and via the thoracic duct into the central venous system.[20,21] The transportation capacity of the lymphatic system is also limited, most of all in patients with increased central venous pressures. Accumulation of fluid in the lymphatic system results in pleural effusions[22] and edema.

This mechanism is more pronounced in the 6 patients with hypoproteinaemia because of protein losing enteropathy (mean total protein 4.4g/dl versus 7.5g/dl and 6.7g/dl in the two other groups), where the oncotic pressure is also reduced.

Based on this hypothesis, we are able to explain the striking incidence of peripheral edema and pleural effusions occuring in three patients with the Fontan procedure who received treatment with the calcium channel blocker verapamil, and who initially did not have congestion. The underlying pathomechanism of formation of edema due to

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calcium antagonists is well documented by animal trials,[23] and is in accordance with our observations.

As recently demonstrated in a cohort study, medical treatment with angiotensin converting enzyme-inhibitors in postoperative patients with the Fontan circulation failed to show an association between treatment with captopril and duration of pleural drainage.[24]

As some patients with effusions after a cavopulmonary connection were resistant to diuretics, we started a therapeutic trial in three of these patients with a betablocker in order to decrease neurohormonal stimulation and cardiac index.[25] Under a dosage of 1.7 mg/kg bodyweight propranolol, giving an average dose of 0.9 – 2.7 mg/kg, we observed a decrease of mean heart rate from 109/min to 100/min without significant changes of mean arterial pressures. All patients improved clinically and the effusions resolved within one week. Along with the clinical improvement, we observed a decrease of enhanced plasma renin and aldosterone levels.[26] Thus, it seems possible to decrease mean hydrostatic capillary pressure by β-blockade, with an additional beneficial effect by inhibiting renin release via β-receptor blockade.

Limitation of the study

The hemodynamic status of patients in their early course after the Fontan procedure was influenced by intensive care, and some patients were on treatment with catecholamines (dopamine 1 – 5 μg/kg/min, dobutamine 3 – 5 μg/kg/min and epinephrine 0.5 – 4 μg/kg/min). Additional, vasoactive drugs were not in use. We still cannot exclude the possibility that some hemodynamic differences between patients during the early and late postoperative course may depend on this medical treatment.

Other possible causes for effusions after the Fontan procedure, such as neurohormonal activation or the presence of collaterals, were not investigated in this study, which is focused on the hemodynamic causes of venous congestion.

Since arterial blood flow is pulsatile, a better way to describe the flow would be to use impedance. For the purpose of the algorithm, we calculated vascular resistance by using mean vascular pressure, assuming a constant and not pulsatile flow.

Appendix

Calculation of minimal mean hydrostatic capillary filtration pressure (Pcap(min))

Measurement of mean capillary filtration pressure is not established for clinical uses. For this study we developed an algorithm to calculate mean capillary filtration pressure from routinely measured hemodynamic data, based on the concept of ultrafiltration in the capillaries, first described by Starling in 1897.

The capillary filtration pressure (Pcap) is the mean of hydrostatic pressures at the beginning (Part) and end (Pven) of the capillaries:

$$\Rightarrow \text{Pcap} = (\text{Part} + \text{Pven})/2$$

Taking into account that the unknown postcapillary resistance is low the pressure at the end of the capillary is equal or greater than central venous pressure (CVP) :

$$\Rightarrow \text{Pcap} \geq (\text{Part} + \text{CVP})/2 \quad \text{(equation A)}$$

In a simplified model of two serial resistances (systemic vascular resistance, SVR, and capillary resistance, Rcap) connected in a closed circuit with a given flow index the pressure at the beginning of the capillary Part can be calculated as follows:

$$\Rightarrow (\text{Part} - \text{CVP}) / \text{Rcap} = (\text{MAP} - \text{CVP}) / \text{SVR}$$
$$\Rightarrow \text{Part}/\text{Rcap} = \text{MAP}/\text{SVR} - \text{CVP}/\text{SVR} + \text{CVP}/\text{Rcap}$$
$$\Rightarrow \text{Part} = \text{MAP} \times \text{Rcap} / \text{SVR} - \text{CVP} \times \text{Rcap}/\text{SVR} + \text{CVP}$$
$$\Rightarrow \text{Part} = [(\text{MAP} - \text{CVP}) \times \text{Rcap} + \text{CVP} \times \text{SVR}]/\text{SVR}$$

Capillary resistance Rcap is not regulated and known to be relatively constant at 4 mmHg× L-1 min m2. To account for body surface area, we use systemic vascular resistance index (SVRI) for further calculation:

$$\Rightarrow \text{Part} = [(\text{MAP} - \text{CVP}) \times 4\ \text{mmHg} \times \text{L-1 min m2} + \text{CVP} \times \text{SVRI}] /\text{SVRI} \quad \text{(equation B)}$$

To calculate mean hydrostatic capillary pressure Pcap, Part in equation A has to be replaced with equation B:

$$\Rightarrow \text{Pcap} \geq (\text{MAP} \times 2\text{mmHg L-1 min m2} - \text{CVP} \times 2\ \text{mmHgL-1 min m2} + \text{CVP}\times \text{SVRI} / 2 + \text{CVP} \times \text{SVRI} / 2) / \text{SVRI}$$
$$\Rightarrow \text{Pcap} \geq [\text{MAP} \times 2\ \text{mmHg L-1 min m2} + \text{CVP} \times (\text{SVRI} - 2\ \text{mmHg L-1 min m2})] / \text{SVRI}$$
$$\Rightarrow \text{Pcap (min)} = [\text{MAP} \times 2\ \text{mmHg L-1 min m2} + \text{CVP} \times (\text{SVRI} - 2\ \text{mmHg L-1 min m2})] / \text{SVRI}$$

Based on this algorithm minimal mean hydrostatic capillary pressure depends not only on central venous and mean arterial pressure but also on systemic vascular resistance.

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