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Reconstructive

Basic Perforator Flap Hemodynamic Mathematical Model

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Background: A mathematical model to help explain the hemodynamic characteristics of perforator flaps based on blood flow resistance systems within the flap will serve as a theoretical guide for the future study and clinical applications of these flaps.
Methods: There are 3 major blood flow resistance network systems of a perforator flap. These were defined as the blood flow resistance of an anastomosis between artery and artery of adjacent perforasomes, between artery and vein within a perforasome, and then between vein and vein corresponding to the outflow of that perforasome. From this, a calculation could be made of the number of such blood flow resistance network systems that must be crossed for all perforasomes within a perforator flap to predict whether that arrangement would be viable.
Results: The summation of blood flow resistance networks from each perforasome in a given perforator flap could predict which portions would likely survive. This mathematical model shows how this is directly dependent on the location of the vascular pedicle to the flap and whether supercharging or superdrainage maneuvers have been added. These configurations will give an estimate of the hemodynamic characteristics for the given flap design.
Conclusions: This basic mathematical model can (1) conveniently determine the degree of difficulty for each perforasome within a perforator flap to survive; (2) semiquantitatively allow the calculation of basic hemodynamic parameters; and (3) allow the assessment of the pros and cons expected for each pattern of perforasomes encountered clinically based on predictable hemodynamic observations. *(Plast Reconstr Surg Glob Open 2016;4:e714; doi: 10.1097/GOX.0000000000000689; Published online 20 May 2016.)*

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The perforator flap as an entity was first clinically introduced in 1989 by Koshima and Soeda.[1] Many advances of this concept have since been made over the past 2 decades.[2–5] Detailed descriptions of the anatomical location, caliber, and potential pedicle length of each perforator and connecting or linking patterns between perforators that can enhance the territorial range of the chosen flap are currently available.[6,7] In 2001, the Gent consensus on perforator flap terminology[8]; the first com-

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pendium on perforator flaps in 2006, *Perforator Flaps: Anatomy, Technique, and Clinical Applications*[9]; and the idea of the perforasome proposed by Saint-Cyr et al[10] in 2009 have all greatly standardized and promoted the development of this perforator flap concept.

Although observations of the anatomical structure of perforator flaps have been incisive, many clinical questions remain unanswered. For example, what is the precise territory that a given perforator can nourish? How do supercharging and superdrainage ancillary maneuvers increase the survival of a perforator flap? Does pressure or flow via the recipient artery have a greater effect on the survival of a perforator-free flap?

Knowledge of the anatomical structure of a perforasome alone is insufficient for answering these aforementioned questions; and further studies on the hemodynamic aspects of perforator flaps are required. Although some pertinent studies in this regard have already been conducted,[11,12] these have mostly been only observations from clinical or animal experiments and as such have been limited to empirical or descriptive conclusions. Our objective instead is to establish a theoretical hemodynamic model for perforator flaps that can explain existing phenomena noted by others, provide a capability for predicting future and even unknown possibilities, and overall provide a good mathematical tool that can then be applied later for both clinical and basic laboratory studies on perforator flaps.

A MATHEMATICAL MODEL OF INTRAFLAP BLOOD FLOW RESISTANCE NETWORKS

Hagen-Poiseuille Equation

The Hagen-Poiseuille equation in nonideal fluid dynamics relates to the pressure drop in an incompressible and Newtonian fluid during laminar flap through a long cylindrical pipe of constant cross section. Although a blood vessel is a nonlinear biological tube of variable diameter, the Hagen-Poiseuille equation can be used to approximately analyze multiple intravascular hemodynamic parameters to simplify calculations and make clinical research easier: in this case,

$$\text{where } R = 8\eta L / \pi r^4 \quad (1)$$

$$\text{and } F = \Delta P / R \quad (2)$$

$$\text{then } F = \pi \Delta P r^4 / 8\eta L \quad (3)$$

where R, resistance; L, vessel length; η, coefficient of fluid viscosity; r, vessel radius; F, flow; ΔP, pressure difference; π, constant.

Relative Blood Flow Resistance Within a Perforator Flap

Taylor and Palmer[13] showed 2 connection patterns between angiosomes, that is, via choke vessels or true anastomoses. With choke vessels, vessel calibers are gradually reduced and angiosomes are connected via small indirect vessels, thereby making blood flow resistance immense. The converse is the case in true anastomoses, where angiosomes are directly connected without reduction in vessel caliber, and the blood flow resistance is small. In a similar manner, Saint-Cyr et al[10] proposed the perforasome, which also has 2 connection patterns between adjacent perforasomes, that is, via indirect or direct linking vessels. Through animal and cadaver perfusion and clinical observations, they found that the blood flow resistance of a perforator flap mainly originated from the sites of choke vessel obstruction and indirect connections between perforasomes.[14]

This same conclusion can be derived by using the Hagen-Poiseuille equation. Assume the diameter of the perforator vessel is r, the intralumen blood flow resistance R, and the radius of the choke vessel is 0.1 r. Therefore, based on Equation (1), blood flow resistance inside the choke vessel will be approximately 10,000 times that within the perforator vessel itself, which is comparatively huge as would be expected. Flow via the perforator vessel can usually cross the first choke zone (the area where the choke vessels or indirect linking vessels are located) but rarely the second choke zone due to this high resistance. Similarly, choke zones in the venous linking areas among perforasomes and capillary regions between the perforator arteries and the veins will be sites with the highest blood flow resistance.

Blood Flow Resistance Networks Within a Perforator Flap

For convenience, blood flow resistance within the choke zone between perforator arteries can be denoted as $R_{(a\text{-}a)}$, that between perforator veins as $R_{(v\text{-}v)}$, and finally between perforator arteries and veins as $R_{(a\text{-}v)}$, which will be considered the 3 major network systems causing resistance within a perforator flap. The degree of difficulty for a flap to survive will then be dependent on the number of these blood resistance networks it needs to cross, that is, the fewer it is required to cross, the easier will it be to survive and vice versa.

To better demonstrate this simplified concept that can predict flap survival, we will start with an analysis of the most rudimentary perforator flap, that is, a flap containing only a single perforasome (Fig. 1). If this perforator flap is to survive, only 1 blood flow resistance network will be encountered, in this case $R_{(a\text{-}v)}$, as blood flow only has to cross from the perfora-

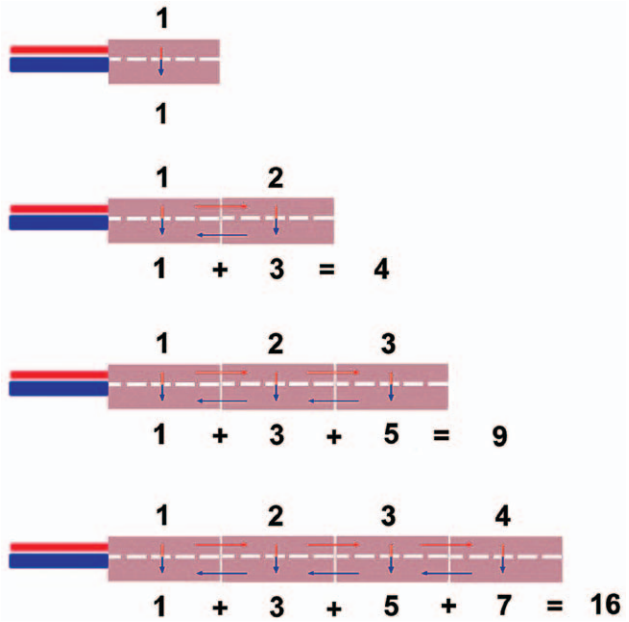

Fig. 1. Intraflap blood flow resistance networks.

tor artery to the perforator vein. The total number of crossed blood flow resistance networks will be 1. In order for a perforator flap consisting of 2 perforasomes (Fig. 1) to totally survive, flow across the first perforasome again needs to cross 1 $R_{(a\text{-}v)}$, whereas to capture the second perforasome blood flow needs to cross 1 $R_{(a\text{-}a)}$, 1 $R_{(a\text{-}v)}$, and 1 $R_{(v\text{-}v)}$; and thus, the total number of blood flow resistance networks crossed will be 1 + 3 = 4. Similarly, if a perforator flap has 3 perforasomes, the number of crossed blood resistance networks will be 1 + 3 + 5 = 9 (Fig. 1). Thus, if the vascular pedicle is arranged as in this preceding example, for a perforator flap containing "n" perforasomes, flow to the nth perforasome needs to cross $(2n - 1)$ blood resistance networks, so the total number of crossed intraflap blood resistance networks will be:

$$\sum_{n=1}^{n}(2n-1)=n^2.$$

However, if the connection patterns of the vascular pedicles or perforasomes are altered, the method of calculation of the blood flow resistance networks will also be altered. For example, in Figure 2 with the outflow tract on the opposite side of the flap from the inflow, the total number of blood resistance networks for 4 connected perforasomes will instead be 4 + 4 + 4 + 4 = 16.

Comparison of Blood Flow Resistance Networks to an Electrical Circuit

A blood flow network can be considered to be very similar to an electrical network according to

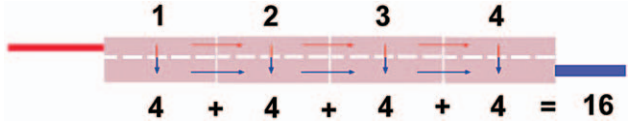

Fig. 2. Variations in the blood flow resistance networks after a vascular pedicle alteration.

Ohm's law,[15,16] with blood pressure, blood flow, and blood flow resistance resembling voltage, electrical flow, and electrical resistance, respectively. Thus, when blood flow resistance networks are connected in series, the total resistance R_t will be:

$$R_t = R_1 + R_2 + R_3 + \ldots R_n \qquad (4)$$

When blood flow resistance network systems are connected in parallel, the total resistance R_t will be:

$$\frac{1}{R_t} = \frac{1}{R_1} + \frac{1}{R_2} + \frac{1}{R_3} + \ldots \frac{1}{R_n} \qquad (5)$$

CALCULATION OF INTRAFLAP RESISTANCE NETWORKS ACCORDING TO CONNECTION SYSTEMS

As outlined in the preceding overview, the 3 major blood flow resistance network systems of the perforator flap have been described, their relationship to the resistance network found in each perforasome and the whole perforator flap enumerated, methods to calculate their numbers within a given flap elaborated; and 2 patterns of series and parallel connections of perforasomes analyzed. How these are connected to hemodynamic parameters within a perforator flap and algorithms for using and understanding them in this regard will be as follows.

Single Perforasome Flap

When a perforator flap contains 1 perforasome, there is only 1 blood flow resistance network $R_{(a\text{-}v)}$ (Fig. 3). After construction of the related "circuit diagram," a blood flow equation can be derived: $F = \Delta P / R$. Because both F and ΔP are measurable, $R = \Delta P / F$ is known. Thus, the hemodynamics of this perforator flap is measurable or quantifiable.

Dual Perforasome Flap

When the perforator flap contains 2 perforasomes (Fig. 4), the flap should be divided into the first and second perforasomes. Total blood flow resistance of the first perforasome is $R_1 = R_{(a\text{-}v)}$, whereas total blood resistance of the second perforasome is $R_2 = (R_{(a\text{-}a)} + R_{(a\text{-}v)} + R_{(v\text{-}v)})$. Again, after the construc-

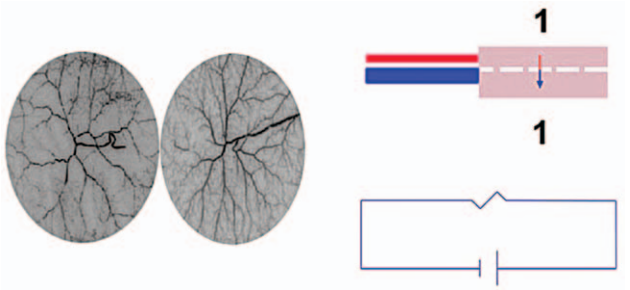

Fig. 3. Blood flow pathway and circuit diagram of the single perforasome.

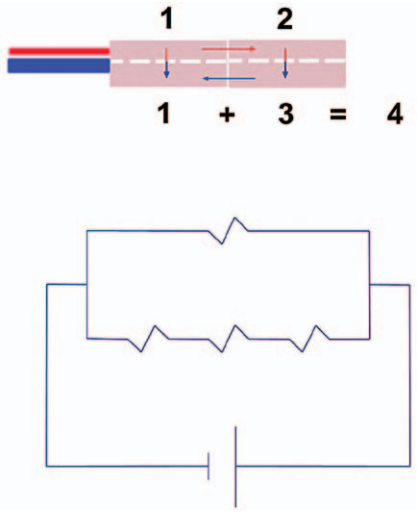

Fig. 4. Blood flow pathway and circuit diagram of dual perforasomes.

tion of the apropo circuit diagram, the total blood flow resistance of the entire perforator flap will be $\frac{1}{R_t} = \frac{1}{R_1} + \frac{1}{R_2} = \frac{1}{R_{a-v}} + \frac{1}{R_{a-a} + R_{a-v} + R_{v-v}}$. Similarly, as for a single perforasome flap, each parameter of this perforator flap can also be calculated.

Multiple Perforasome Flap

When the perforator flap contains multiple perforasomes, the flap should first be divided into multiple parts based on the number of perforasomes. Next, the blood resistance network systems to be crossed for each perforasome to survive should be counted. Then, the corresponding circuit diagrams according to the connection patterns (in series or parallel) of each perforasome should be drawn. This allows the blood flow resistance of each component part based on the corresponding circuit diagrams and equations and the total blood flow resistance to be calculated. Finally, the survival probability of each portion and the entire perforator flap can be estimated based on the results of this calculation.

REPRESENTATIVE CLINICAL APPLICATIONS OF THE BLOOD FLOW RESISTANCE NETWORK MODEL IN PERFORATOR FLAPS

Location of the Vascular Pedicle and Degree of Difficulty for a Perforator Flap to Survive

As an example of the clinical utility of this model, consider 2 perforator flaps, each with 3 identical perforasomes of the same size and vessel caliber, with the only difference being the location of their vascular pedicle, that is, one located laterally on the same side and another at the midline of the flap (Fig. 5). If questioned which has the greater chance of survival, intuitively the one with its pedicle located at the midline should have the better probability. Now to explain this using our model. First, each flap is divided into 3 parts based on the number of perforasomes. If the perforator vascular pedicle is located at the midline, the number of blood flow resistance networks crossed is 3 + 1 + 3 = 7; but if located laterally with inflow and outflow on the same side, the number of blood resistance networks will be 1 + 3 + 5 = 9 (Fig. 5). Because the greater the number of blood flow resistance networks crossed, the less likely the flap will survive; based on these calculations when the vascular pedicle is in the middle of the 3 perforasomes, total flap survival chances would be improved as predicted.

Supercharging or Turbocharging and Superdrainage

Clinical and animal experiments have verified that supercharging or turbocharging and superdrainage can enhance flap survival.[17–19] This is accomplished by either increasing the arterial pressure or reducing the venous pressure, which will increase the pressure difference (ΔP) within the flap. Based on Equation (2), if ΔP increases, flow (F) increases, making it more likely for the flap to survive. In addition, supercharging, turbocharging, and superdrainage change the blood flow pathways of each part of the perforator flap to lower the encountered number of blood flow resistance network systems. The perforator flap containing 3 perforasomes described in Figure 1 will be used as an example. When the vascular pedicles are located laterally on the same side, the number of blood resistance networks is 1 + 3 + 5 = 9. If supercharging or superdrainage is added, this number drops to 1 + 3 + 3 = 7 (Fig. 6), which not only reduces the total number of

Fig. 5. Locations of the perforator vascular pedicles and the blood flow pathways.

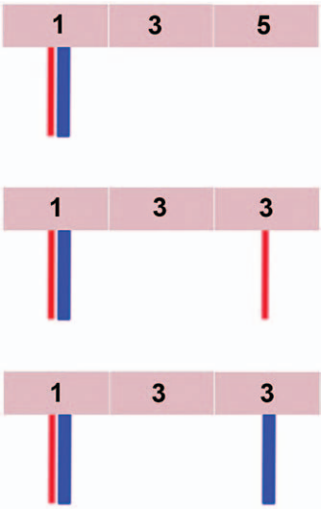

Fig. 6. Supercharging and superdraninage tracts and the alternation of the total resistance networks encountered.

intraflap resistance networks, but more importantly, the number of blood flow resistance networks for the third perforasome from 5 to 3, altering it from a potentially dangerous state (5) to a safe state (3).

Extent of Flap Viability

Several recent studies[20–22] have focused on the territory a flap perforator can successfully nourish, which can be explained by our model. From Equation (2), $F = \Delta P / R$, increased blood flow and theoretically enhanced flap perfusion can be achieved either by increasing the pressure difference (ΔP) or by reducing the blood resistance (R). There are many ways to increase the ΔP; for example, by increasing the arterial pressure, selecting a large perforator, inducing supercharge or turbocharge, delaying the connection of choke vessels; or reducing the venous pressure by inducing superdrainage. Reducing the total intraflap resistance by changing the blood flow pathways, such as with delay maneuvers, is also feasible to reduce the R. More experimental data are needed to substantiate our basic mathematic model, which still serves primarily as a guideline.

LIMITATIONS OF THE PRESENT PHYSICAL MODEL

This mathematical model should not be considered as anything but the most rudimentary as a preliminary guideline upon which future laboratory or clinical studies can be established to clarify numerous variations in the usual biological systems. For example, to simplify our calculations using the Hagen-Poiseuille equation, the variable of vessel length (L) was not considered to be in any way an influencing factor. Of course, a blood vessel is not a homogeneous and rigid tube but instead subject to the elastic dilatation and contraction due to pulsatile blood flow within the arterial vessel wall or distension of venous valves. In turn, no difference was distinguished among $R_{(a\text{-}a)}$, $R_{(a\text{-}v)}$, and $R_{(v\text{-}v)}$ when calculating the total number of blood flow resistance networks, although capillary and venous networks would intuitively cause far greater resistance than $R_{(a\text{-}a)}$. This model has been predicated for ideal conditions and does not take into account variability in regional differences of perforasomes nor the effect of temperature, vasodilators, blood pressure, or other systemic factors that could affect network resistance and in turn flow. In addition, the clinical situation may be more complicated since perforasome connection patterns, how these relate to adjacent tissues, variations of vascular patterns, and their integrity upon harvest of a perforasome can all potentially affect the expected results as to flap perfusion and viability. Indeed, the intent here has been to provide only a starting point that will require modification by more exact future animal and clinical experiments.

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