The Vasculosome Theory

Sir:

I read with great interest the article entitled "True and 'Choke' Anastomoses between Perforator Angiosomes: Part II. Dynamic Thermographic Identification," by Chubb et al. Interestingly, the findings of this study could better be explained by the "perforasome theory" rather than the "angiosome theory."[1] Both theories provide a guide to identify tissue units that can be reliably transferred in a single stage, based on a single source of blood supply. However, both theories cannot reliably predict the exact quantity of tissue that can be supported by a single feeding vessel.[2]

Taylor and Palmer defined the angiosome as a vascular territory, which is marked by the presence of reduced caliber vessels in its periphery.[3] The caliber by definition is narrow enough not to allow independent perfusion of the adjacent angiosome. Eventually, Taylor et al. also defined a perforasome as an inherent part of the angiosome.[4] In the current article, Chubb et al. investigate reduced caliber vessels connecting perforasomes.[5] It seems to contradict their angiosome theory, because by definition there cannot be narrow caliber vessels within an angiosome. The only reasonable explanation to sustain the description by Taylor et al. of a perforasome being a part of the angiosome, is to concede that angiosomes are not defined territories by themselves but are territories defined by several perforasomes (i.e., ∑perforasomes = an angiosome).

Saint-Cyr et al. state, "Each perforator has its own vascular territory, called a perforasome, which carries a multidirectional flow pattern that is highly variable and complex."[1] Each perforasome is connected to its adjacent perforasomes by means of linking vessels, which could be "large-caliber" or "narrow-caliber" channels. However, the direction in which a larger tissue territory could be harvested based on a single perforator cannot be predicted based on the perforasome theory.

McGregor and Morgan defined an axial flap as "a single pedicled flap which has an anatomically recognized arteriovenous system running along its long axis."[6] Branches of adjacent perforators, connected together by large-caliber channels, can form such a vascular axis. A unit of tissue incorporating this vascular axis and nourished by a single "feeder vessel" or perforator could be considered to be a "vasculosome." In other words, any feeder vessel arising from a source vessel and contributing to the "vascular axis" should be sufficient to support the entire vasculosome independently. In fact, the objective of "flap delay" is to induce the formation of a neovasculosome with a centrally placed "vascular axis."[7]

For a cutaneous vasculosome, suprafascial directionality of a perforator points toward the large-caliber channels with which it forms a vascular axis in the suprafascial plane.[8] A single perforator supplying this vascular axis should be adequate to support a flap including the two (or more) adjacent "skin-perforasomes,"[9] which will constitute a vasculosome (Fig. 1). Chubb et al. have suggested the use of thermography for detecting this vascular axis to aid flap elevation.[5] Indeed, accurate mapping of a particular cutaneous vascular axis will result in better delineation of the cutaneous vasculosome and thus elevation of a more reliable flap.

The muscle vasculosome has a vascular axis in the muscle. A single feeding vessel supplying any part of the muscle can nourish the entire muscle, provided that a vascular axis exists within the muscle (Fig. 2). If there are "narrow-caliber" channels linking adjacent muscle units, reliable harvest of both territories based on a single feeder is unlikely.[10] Even so-called traditional minor pedicles can

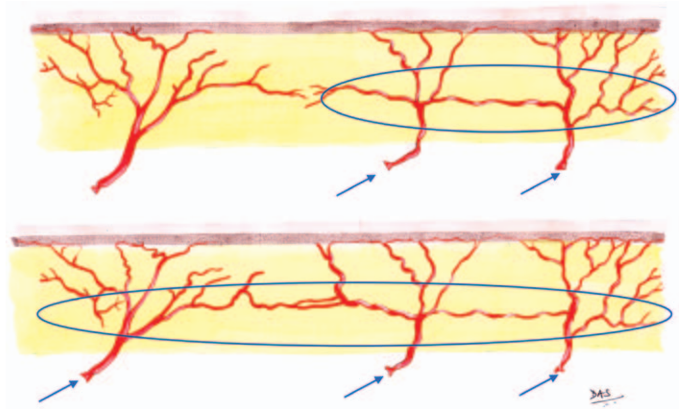

Fig. 1. Two cutaneous vasculosomes with their vascular axes (*blue circles*) in the suprafascial plane fed by two perforators (*blue arrows*) above and three perforators below.

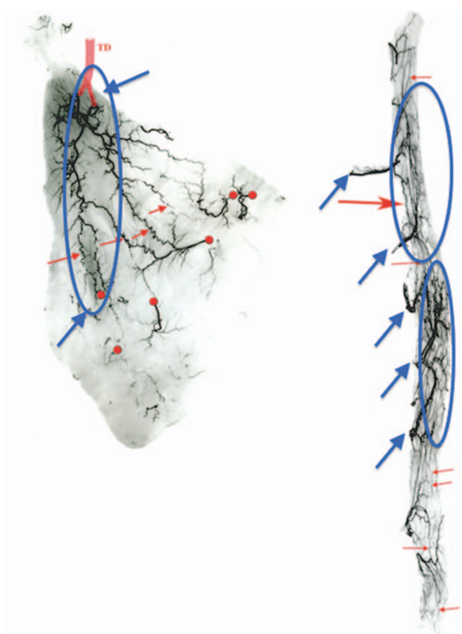

Fig. 2. Muscle vasculosomes with vascular axes (*blue circles*) in the muscles fed by perforators (*blue arrows*). (Adapted and republished with permission from Taylor GI, Chubb, DP, Ashton MW. True and "choke" anastomoses between perforator angiosomes: Part I. Anatomical location. *Plast Reconstr Surg*. 2013;132:1447–1464.)

perfuse an island-muscle flap based on a single perforator, provided that they feed the vascular axis.[11] Reliable preoperative imaging for identifying muscle vasculosomes is yet to be invented. However, intraoperative differential clamping of muscle pedicles can identify a muscle vasculosome.

The vasculosome theory provides a physiologic basis for flap elevation, unlike the anatomically rigid concepts of angiosomes and perforasomes. It is time we stopped investigating the branching pattern and course of an individual feeder vessel and start identifying the vascular axes and the perforators feeding those axes.[12] To conclude, it is the location of a hemodynamically robust perforator and the vasculosome it supplies that will determine the design of a perforator flap.

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Reply: The Vasculosome Theory

Sir:

We wish to thank Dr. Adhish Basu for his comments, directed toward our use of thermography to differentiate between the true and choke anastomoses that connect cutaneous perforators[1] to select the most robust flap along an axis that he has named a "vasculosome." Obviously, he likes this method of detection, but we hope his comments are a little "tongue in cheek" when he suggests that we should ignore the anatomy of the blood vessels when designing flaps and focus on their physiology, especially because some of the major advances in plastic surgery, beginning in the early 1970s, have focused on revisiting the vascular anatomy of the part. This "anatomical renaissance" started when McGregor and Jackson designed the groin flap[2] after reassessing the anatomy of the superficial circumflex iliac artery. This in turn led to the anatomical distinction between axial and random pattern flaps,[3] the free flap,[4,5] and now perforator flaps.[6]

When we subdivided the body anatomically into 40 angiosomes spanning between skin and bone, each supplied by a segmental or distributing source artery and linked together by true or choke anastomoses, we stated in our original article and since then[7–10] that each angiosome could be subdivided further, with each subdivision connected by either true or choke anastomoses. Not only did we define the cutaneous perforator as the smallest angiosome skin module, but we also identified the various angiosome modules within each muscle, supplied by vascular pedicles and connected again by true or choke anastomoses (Fig. 1). Later, we defined these vascular units in the nerves[11,12] and in bone.[13] Subsequently, Saint-Cyr et al.[14] have redefined these territories, renaming the same true and choke anastomoses "large and small caliber linking vessels," respectively, and the cutaneous angiosomes perforasomes.[14]

We have shown in animal models that not only will the tissue within the anatomical boundary of the perforator angiosome survive totally, but so will the next perforator angiosome connected radially in any direction by choke vessels (Fig. 2, *left*) or longer if connected by a true anastomosis (Fig. 2, *right*). This has been well documented in Part I and previously[8,9] and may have been overlooked by Dr. Basu.

Dr. Basu rather ambitiously states that he would like to design flaps without knowledge of their vascular anatomy yet have a long viable flap. Although impossible in the past without trial and error, recent advances in our knowledge of vascular anatomy have allowed surgeons to predict areas of the integument where the blood supply is more reliable.

Today, with a careful examination of the patient and the use of Doppler imaging, computed tomographic angiography, and recently thermography, the vascular reliability of a flap can be predicted preoperatively. By focusing on the fixed skin sites using these three modalities, the larger perforators can be identified as they emerge from the deep fascia. Next, their long branches can be located because they radiate along mobile skin folds such as the groin, loin, breast, and scalp (see Figs. 2 through 4 in Part I) and course in parallel with the cutaneous nerves, often connected by true anastomoses (see Figs. 4 through 6 in Part I), which in turn frequently parallel the large subdermal primary venous channels.[9,10] These veins can often be marked preoperatively. Examples include the long and short saphenous veins, the superficial inferior epigastric vein, and the superficial circumflex iliac vein. Thermography may help detect these true anastomoses as the basis for a longer, more robust flap. Finally, if the next dominant perforator is located along the chosen axis by the modalities outlined above, it is almost certain that this section of the flap will survive totally. If the connection between these two perforator angiosomes has been revealed by thermography to be a true anastomosis, it is likely that the angiosome territory of the next perforator will be captured safely as well (compare Figs. 2 and 3 in Part I).

In conclusion, we have shown that, when a flap is raised, there is no ingrowth or rearrangement of new vessels along the flap axis; rather, there is an increase in caliber of the existing vessel architecture from the base to the tip. This is caused by hyperplasia of all layers of the vessel walls, especially the choke vessels, commencing rapidly between 48 and 72 hours and complete by 7 days[15] (Fig. 3). All of this information has been obtained by studying the anatomy of the vasculature and observing how this has been changed by the physiologic events that have been inflicted on the tissue by the surgeon's scalpel.

If we ignore anatomy, does this mean that we go back to Gillies' era of designing flaps on a trial-and-error basis, especially as he often lamented that this was "a constant battle between blood supply and beauty"?[16] We hope not!

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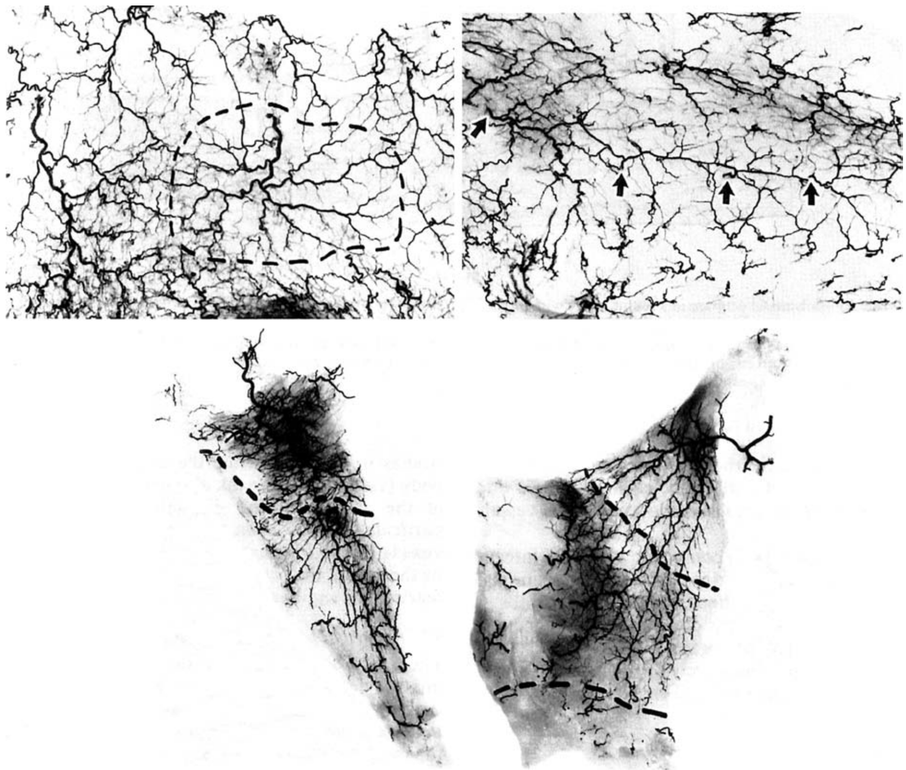

Fig. 1. A reproduction of Figure 12 and the text. [With permission from Taylor GI, Palmer JP. The vascular territories (angiosomes) of the body. *Br J Plast Surg*. 1987;40:113–141.] Radiographs of selected regions of the integument and muscles. (*Above*, *left*) *Dotted line* drawn around the perimeter of choke connecting vessels of a large acromiothoracic perforator to define its anatomical territory (compare with Figure 5 front view, left side of body). (*Above*, *right*) Chain-linked system of perforators anastomosing without change in caliber (*arrows*). They accompany the lateral cutaneous nerve of the arm (compare also with Figure 5 front view, right arm). (*Below*, *left*) Trapezius muscle with transverse cervical (*above*) and posterior intercostal (*below*) territories defined. (*Below*, *right*) Latissimus dorsi muscle with thoracodorsal (*above*), posterior intercostal (*center*), and lumbar territories (*below*) outlined, which again correspond to their respective angiosomes.

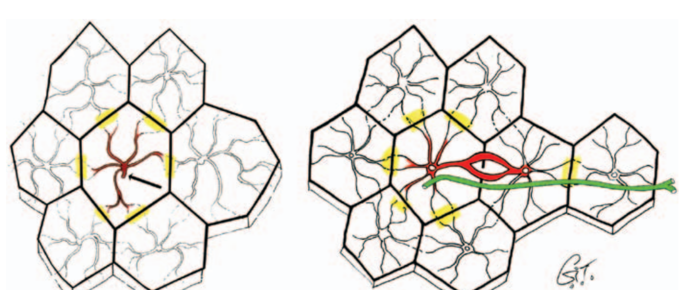

Fig. 2. Schematic diagram (*left*) of the clinical territory of a cutaneous perforator (*arrow*) when linked by choke vessels (*yellow*) to neighboring cutaneous angiosome perforators in any direction, and (*right*) additional angiosome territories captured in a particular axis when connected by a true anastomosis (*red*), especially if in parallel with a cutaneous nerve (*green*).

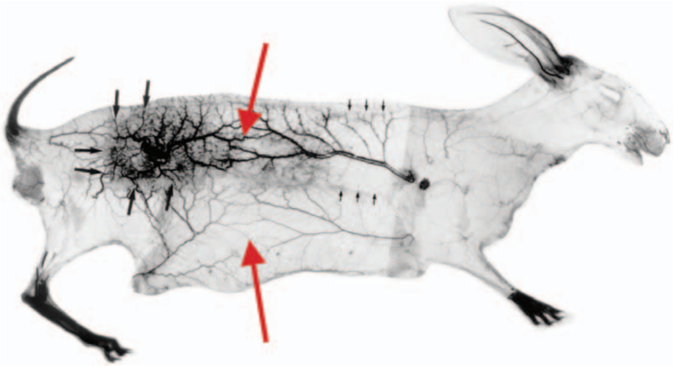

Fig. 3. Lead oxide arteriogram of a two-territory rabbit flank flap based at the axilla at 1 month. All vessels have enlarged along the flap compared with the parallel vasculature anteriorly, and the choke vessels connecting the two cutaneous angiosomes in the flap have enlarged to the size of the true anastomoses by comparison (*red arrows*). The difference in the caliber of vessels (*arrows*) crossing the flap margin, which appear to increase from the base to the tip, a possible response to a progressive decrease in oxygen tension. (Reprinted from Dhar S, Taylor GI. The delay phenomenon: The story unfolds. *Plast Reconstr Surg*. 1999;14:2079–2091.)

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