Muscle Architecture and its Relationship to Muscle Circulation

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Muscle contraction mechanically changes vessel geometry and consequently muscle circulation. Lengthening of muscle fiber straightens capillary tortuosity and further increase in muscle fiber length reduces blood flow by extending the capillary while reducing its diameter. The direction of capillary to the fiber long axis will modify the extent of capillary lumen diameter change due to lengthening muscle fiber. During passive stretching in human subjects, the relationship between changes in blood volume (determined by near-infrared spectroscopy: NIRS) and fascicle length differed among the three heads of triceps surae muscles with different fascicle length and pennation angle. Muscle thickness, muscle curvature, and muscle fascicle angle are potential determinants of intramuscular pressure during muscle contraction. Muscle circulation is impeded more in short bulging muscles with great curvature of fibers than in long slender muscles with less curvature. The different response of circulatory parameters across the synergist muscles in the calf and heterogeneity of muscle circulation in the same muscle were observed, partly due to the difference in the pennation angle of the muscle fascicle. The muscle architecture will influence venous outflow by changing the muscle pumping action. This is possibly an indirect way of modifying vasodilation due to difference in muscle architecture.

Keywords: pennation angle, muscle fiber length, blood volume

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1. Introduction

Blood flow during rhythmic exercise dramatically changes concomitant with muscle contraction and relaxation (Barcroft and Dornhorst 1949, Barnes 1986, Kagaya and Ogita 1992, Walløe and Wesche 1988). This mechanical disturbance of muscle circulation originates from the anatomical arrangements of muscle fibers and vessels.

Figure 1 shows a schematic illustration of artery and capillary alignment in relation to muscle fiber. The conduit artery (brachial artery, femoral artery, etc.) branches into the feeding artery, whose daughter arteries run into the muscles as terminal arterioles and capillaries (Figure 1A). When the muscle fibers are extended, the capillaries running parallel to the fiber long axis will be lengthened (Figure 1B), whereas those running in a perpendicular direction will be shortened (Figure 1A) (Nakao and Segal 1995, Poole, et al., 1997). When the muscle fibers are shortened, the opposite occurs. In addition, the muscles develop tension during muscle contractions and thereby the elevated intramuscular pressure compresses the capillaries between the fibers (Sjøgaard, et al., 1986, Sejersted, et al., 1984) (Figure 1B). This causes blood flow restriction or occlusion to the contracting muscles on the arterial side and expels blood from the muscle on the venous side (muscle pump) (Barnes 1986, Bonde-Petersen, et al., 1975, Gaskell 1877, Kagaya and Ogita 1992, Lind and McNicol 1967, Sadamoto, et al., 1983, Sjøgaard, et al., 1986.). Accordingly, changes in vessel geometry due to muscle lengthening/shortening and vessel compression due to developed muscle tension are two major factors which mechanically modify muscle circulation.

On the other hand, muscle architecture differs from muscle to muscle, (Kawakami, et al., 1998,
Maganaris, et al., 1998, Kanehisa, et al., 2003, Muraoka and Kagaya, 2003) and even within the same muscle (Maganaris, et al., 1998, Muramatsu, et al., 2002a). Thus the question arises as to how the difference in muscle architecture, such as fascicle curvature, fascicle angle, fascicle length, muscle thickness, etc., alters muscle circulation via changes in intramuscular pressure or muscle fiber geometry.

This paper reviews the effects of muscle architecture on muscle circulation and attributes these effects to the changes in capillary geometry and intramuscular pressure. The topics of this review include 1) the effect of muscle fiber length on muscle circulation in resting muscle, 2) the effects of muscle architecture on muscle circulation via intramuscular pressure change, 3) the heterogeneity of muscle circulation depending on muscle architecture, and 4) the effect of muscle architecture on the relationship between venous outflow and vasodilation.

2. Effect of muscle fiber length on muscle circulation

Results from animal studies indicated that the lengthening of muscle fiber reduced muscle blood flow at rest (Supinski, et al., 1986; Poole, et al., 1997) and during exercise (Supinski, et al., 1986). The reduction of blood flow was caused not only by the reduced capillary diameter (Welsh and Segal 1996, Poole, et al., 1997), which resulted in the augmented vessel resistance, but by the enhanced sympathetic nerve activity initiated by muscle lengthening (Welsh and Segal 1996).

However, the capillary diameter and muscle blood flow did not change linearly with increasing muscle fiber length (Supinski, et al., 1986; Poole, et al., 1997). Instead, it changed in a biphasic way (Figure 2, Poole, et al., 1997). The capillary lumen diameter of spinotrapezius muscle in rats decreased moderately up to sarcomere lengths of ~2.9 µm and steeply declined as it became longer. The authors suggested that these results were attributable to capillary tortuosity. The moderate increase in sarcomere lengths (up to 2.9 µm) did not influence muscle circulation, because it may only straighten capillary tortuosity, but further increases in sarcomere lengths reduced the capillary diameter and consequently blood flow.

Another effect of change in muscle fiber length is on the geometry of the vascular network in the
muscle. This effect will depend on the orientation of microvessels to the muscle fibers axis; whether the direction of the capillary long axis is identical or not to the long axis of muscle fiber. A sophisticated investigation by Nakao and Segal (1995) demonstrated how muscle length change would alter the geometry of arterioles and venules in the parallel-fibered retractor muscle of anesthetized male hamsters. When muscle fiber was lengthening, the capillary lumen changes differed between vessels running parallel to muscle fiber axis and those bifurcating away from the muscle fiber long axis (Figure 3, Nakao and Segal 1995). Accordingly if muscle fiber was lengthened, the former vessel (paralleled) lumens will become smaller, whereas the latter vessels could become larger. Therefore, the ratio of the number of capillaries running parallel to, and running away from the muscle fiber long axis

Figure 2  Relationship between sarcomere length and capillary lumen diameter in spinotrapezius sarcomere length in rat (Poole, et al., 1997).
The capillary lumen diameter decreased moderately up to sarcomere lengths of ~2.9 μm and steeply declined as it became longer than this.

Figure 3  Illustration of effect of muscle length on microvessel geometry. (Nakao and Segal 1995)
A schema of the effect of shortened (left, 80% of in vivo resting length) and lengthened (right, 130% of in vivo resting length) muscle fiber length on the vessel geometry and diameter (2 straight lines in the circles DN and DP) are presented. When muscle fiber is lengthened, the vessel oriented parallel to the muscle fiber long axis is lengthened and its diameter is reduced. In contrast, the vessel running in a perpendicular direction is shortened, or the tortuosity and bifurcation angle of the vessel become greater.
D: distance, L:length, A: angle, P:parallel to the muscle fiber, N: normal (perpendicular)
will have some influence on the sum of the vessel diameters in the whole muscle.

On human subjects, the different relationships between fascicle length and muscle circulation were reported in three heads of calf muscles (Yokozawa, et al., 2002). During passive stretching, Yokozawa, et al. (2002) studied fascicle length and muscle blood volume (total hemoglobin/myoglobin) using NIRS. They found that the blood volume decreased in two (medial gastrocnemius; MG and soleus muscle; SOL) of the three heads of calf muscles (Figure 4). However, in the lateral gastrocnemius muscle (LG), no blood volume reduction was detected with increasing muscle fascicle fiber length. The reason speculated is that the stretching used in the study was not enough to lengthen the long fascicle fibers in LG, which had the longest fascicle length of the three heads of gastrocnemius muscles (Kawakami, et al., 1998). Another possibility is that the capillaries in LG may be more tortuous than in MG and SOL and they might be not straightened during stretching used in the study.

3. Effects of muscle architecture on muscle circulation via intramuscular pressure change

This section will firstly deal with how muscle architecture, such as muscle thickness, muscle curvature, etc., modulates intramuscular pressure during muscle contraction, and secondly the difference in muscle circulation due to muscle architecture in synergist muscles.

3.1. Effects of muscle architecture on intramuscular pressure

A study by Sejersted, et al. (1984) demonstrated that contraction force and muscle layer thickness were determinants for intramuscular pressure. They recorded intramuscular fluid pressure in human vastus lateral muscles and showed that the intramuscular pressure increased with the increasing muscle contraction force and with the intramuscular distance (approximately this means the depth of the muscle tissue) from the point where the catheters penetrated the muscle fascia (Figure 5). They proposed the following formula for muscles with curvature, which showed that the pressure increased linearly with depth;

\[ P = P_0 + n \Delta h + s/r, \]

where \( P \) is the increment in tissue pressure, \( P_0 \) is the pressure just beneath the fascia, \( \Delta h \) is the thickness, \( s \) is stress and \( r \) is the radius curvature. The result suggests that the intramuscular fluid pressure increases more in short bulging muscles with great curvature of fibers than in long slender muscles with less curvature (Sejersted, et al., 1984, Van Leeuwen and Spoor 1992).

This suggestion was supported by a study by Naamani, et al. (1995) who showed that the inhibitory effect of muscle contraction on muscle circulation was greater in gastrocnemius muscles compared with diaphragm muscles. They explained this difference by different muscle structure; more cylindrical shaped muscle (gastrocnemius) and a muscle with a flat thin surface diaphragm.
Furthermore, in the study of human skeletal muscles using B-mode ultrasonography, the fascicle curvature of the medial gastrocnemius muscle was reported to be positively correlated to pennation angle and muscle thickness during isometric contraction (Muramatsu, et al., 2002b). Pennated muscle has another disadvantage in tension transfer from muscle fiber to the tendon. To induce a given tendon force at the site of the joint, muscle with a larger pennation angle is required to develop higher muscle tension (Gans, et al., 1987), leading to greater intramuscular pressure.

Accordingly, the shape of the muscle, indicated by the pennation angle of the fibers, muscle thickness, muscle fiber length etc., will be the important determinants of changes in intramuscular pressure due to muscle action and the consequent muscle circulatory alteration.

3.2. Muscle circulation difference due to muscle architecture in synergist muscles

Muscle architecture differs among the synergist muscles. Therefore, the effect of muscle action on muscle circulation was hypothesized to differ among muscles with different architecture.


To assess the effect of inhomogeneous muscle architecture on muscle circulation across synergistic muscles, the total hemoglobin was measured as an indicator of blood volume using near-infrared spectroscopy (NIRS) during static plantar flexion exercise in triceps surae muscles (Muraoka and Kagaya 2003). Blood volume decreased during static action in medial gastrocnemius (MG) and soleus (SOL) muscle and the reduction became greater with increasing force generation (%MVC) (Figure 6). However, in lateral gastrocnemius (LG), it did not change notably during static action at lower intensities and, to our surprise, it tended to increase during exercise at higher intensities.

As to muscle architecture, LG has the longest fascicle length, smallest fascicle angle (Kawakami, et al., 1998, Maganaris, et al., 1998, Muraoka and Kagaya 2003). This might lead to the smaller elevation in intramuscular pressure in LG and less blood volume reduction during muscle action. Another possibility for the different behavior in LG might be a lesser contribution to the force generation at the ankle joint. There has been no direct evidence for this, but the study by Kinugasa, et al., (2005)
showed that the activation of the muscles was less (35%) in LG compared to MG (46%). If this finding implies a smaller increase in force in LG than MG with an increase in contraction level, the absence of comparable decrease in blood volume in LG would be attributable to the lower resistance in the artery supplying to LG compared with MG.

However, this explanation was not applicable to different responses in blood volume between LG and SOL because the activation of the SOL was similar to the LG (Kinugasa, et al., 2005). Possibly, different responses in blood volume to a change in the muscle length (Yokozawa, et al., 2002) may be involved.

4. Heterogeneity of muscle architecture and circulation within the muscle

As generally accepted, an uneven distribution of muscle fibers rich in capillaries (type I) will cause inhomogeneous blood flow across the whole muscle. There are other aspects of inhomogeneous muscle circulation in the muscle. The first is inhomogeneous muscle architecture. The second is a mismatching of motor unit (MU) and microvascular unit (MVU), which came from a spatial consideration of motor unit and capillary alignment.

The finding that muscle architecture differed among different sites of the same muscle (Maganaris, et al., 1998, Muramatsu, et al., 2002a) will lead to the heterogeneity of muscle circulation or metabolism (Miura, et al., 2001). Miura, et al., (2001) showed that the distal portion of the medial gastrocnemius had larger changes in muscle oxygen saturation and blood volume (measured using NIRS) than the proximal portion had. To elucidate the relationship between muscle architecture and muscle circulation heterogeneity within the same muscles, Miura, et al., (2004) measured architectural properties, oxygen supply, and consumption index in the medial head of the gastrocnemius muscle in vivo using B-mode ultrasound and functional near infrared (NIR) imaging devices. They found that the changes in fascicle length and fascicle angle at the distal portion of the gastrocnemius muscle were greater than those at the proximal portion, and the muscle structural changes were closely related to the changes in the deoxygenated Hb and blood volume. The conclusion was that plantar flexion exercise produced regional differences in oxygenation status consistent with regional differences in muscle architecture.

Spatial considerations of muscle fibers at the single motor unit (MU) level and capillary alignment at the single microvascular unit (MVU) level are interesting from the viewpoint of heterogeneity of muscle circulation. MVU was defined as a terminal arteriole and the group of capillaries it supplies (Figure 7) (Emerson and Segal 1997). Each MVU was considerably shorter than the length of skeletal muscle fibers, which means the capillary branching from same MVU is located in a limited region. Furthermore, MVU was not precisely aligned along the muscle fiber(s) (Emerson and Segal 1997), and rather spread to neighboring muscle fiber(s). This finding implies that blood flow cannot selectively
increase to a specific muscle fiber or to a particular group of fibers, because whenever any capillary is perfused, all other capillaries in the same MVU are necessarily also perfused (Emerson and Segal 1997, Fuglevand and Segal 1997, Lo, et al., 2003). On the other hand, the muscle fibers belonging to the same MU are distributed diversely within the muscle. Accordingly, a spatial mismatching occurs between perfused capillaries and activated muscle fibers (Lo, et al., 2003) (Figure 8). A simulation study by Fuglevand and Segal (1997) demonstrated that the widespread dispersion of MU fibers facilitates complete capillary (MVU) perfusion of muscle at low level activity.

What remains to be shown is whether this spatial mismatching between muscle fiber of the same MU and capillaries of the same MVU differs among different muscles or persons of different physical characteristics.

5. Effect of muscle architecture on the relationship between venous outflow and vasodilation

The finding on blood volume changes in synergist muscles (Muraoka and Kagaya 2003) led us to hypothesize that muscle architecture modifies the muscle pumping effect on circulation. Muraoka and Kagaya (2003) demonstrated that the reduction of blood volume during static plantar flexion was larger in MG and smaller in LG, and the former muscle had a shorter fascicle length and larger fascicle angle than the latter muscle. However, this hypothesis on human subjects has been tested using muscle blood volume changes and no studies directly determined the blood outflow (venous flow) from the muscle due to muscle pump.

Our recent study challenged to estimate the venous outflow at the site of brachial vein when a handgrip exercise was performed (Kagaya, et al.,
Figure 9 illustrates the spectrum of the venous flow velocity in brachial vein at the onset of isometric muscle contraction. The venous blood flow velocity was accelerated immediately after initiation of muscle contraction, but thereafter it gradually decreased despite the muscle still contracting at a given force. Therefore, the muscle pump effect was enforced at the very beginning of each contraction.

At the arterial side, the blood flow in the conduit artery was inhibited during the first cardiac cycle at the beginning of isometric muscle contraction when the muscle starts shortening (Kagaya, et al., 2001). This is consistent with a study by Rogers, et al., (1997), which showed the arterial blood flow was inhibited during concentric contraction (shortening) phase. During this phase, the muscle tension was increasing (inhibitory factor), with the muscle fiber shortening (dilatory factor). After the 2nd cardiac cycle, the arterial blood flow was augmented and thereafter remained at a constant level (Kagaya, et al., 2001). Only limited knowledge was obtained as to the coordination of venous and arterial blood flow during exercise.

The next topic we are interested in is the effect of muscle architecture on the relationship between arterial and venous blood flow. However, no evidence has accumulated yet on this topic. Before elucidating this question, the muscle pump effect on vasodilation should be determined, because the results of recent studies are conflicting concerning the effects of muscle pump on vasodilation.

Several studies showed that the muscle pump increased venous emptying and increased the pressure gradient between the artery and vein across the muscle (Shiotani, et al., 2002, Tschakovsky and Hughson 2000, Tschakovsky and Sheriff, 2004). Shiotani, et al., (2002) reported that the muscle pump-dependent venous pressure drop has a potential to increase leg blood flow at least threefold during upright (not in supine position) exercise via the increase in leg perfusion pressure and/or reflex vasodilation in the leg. In contrast, Hamann, et al., (2003) showed that muscle contraction did not give any effect on the maximally vasodilated vasculature by infusing adenosine. Similarly Valic, et al., (2005) concluded in a study using anesthetized dogs that the muscle pump is not a major contributor to the hyperemic response to skeletal muscle contraction.

Considering the effect of muscle structure on intramuscular pressure and muscle blood volume (Muraoka and Kagaya 2003), it is reasonable to suppose that muscle architecture plays an important role in determining venous outflow as well as arterial inflow. Accordingly the balance between arterial blood flow and venous blood flow could be modified due to the difference in muscle architecture. However more information is needed on this topic.

6. Summary

This paper reviews the effects of muscle architecture on muscle circulation with special reference to intramuscular pressure to the vessels and capillary geometry. The subjects discussed in this study were summarized as follows:

1) The lengthening of muscle fiber straightens capillary tortuosity, and further increase in muscle fiber length reduces blood flow by...
extending the capillary while reducing its diameter. The direction of the capillary to the fiber long axis will modify the effect of muscle fiber lengthening on the capillary lumen diameter. These relations between the muscle fiber length and capillary geometry may produce heterogeneity in blood circulation across synergistic muscles at rest and during passive stretching.

2) The muscle architecture, such as muscle thickness, muscle curvature, muscle fascicle angle, etc., will change intramuscular pressure, which increases more in short bulging muscle with great curvature of fibers than in long slender muscles with less curvature. These factors of muscle architecture lead to heterogeneous responses in blood circulation across synergistic muscles during contractions.

3) Heterogeneity of muscle circulation was indicated at the distal and proximal portion of the calf muscle. This difference is partly attributable to the difference in the pennation angle of the muscle fascicle. The mismatching/coordination of microvascular unit and motor unit will be an interesting subject to study.

4) Muscle architecture influences venous outflow by changing the muscle pumping action. However, only limited information is available on venous blood flow directly measured on human subjects. The effect of muscle architecture on the relationship between venous outflow and arterial inflow remains to be studied.

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References
Miura H, McCuly K, Hong L, Nioka S, Chance B. (2001). Regional difference of muscle oxygen saturation and blood volume during exercise determined by near infrared imaging

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