Effect of Different Time Durations of Static Stretching of the Calf Muscle on Vascular Response in Popliteal Artery

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Abstract: Background: Precise matching of blood flow and metabolism is required for virtually all living tissues, this is especially important for skeletal muscle. Static stretch was thought to restore blood flow to the muscle and interrupt the pain spasm cycle. Purpose: To determine the effect of optimal time duration of passive static stretch of calf muscle on blood flow volume and cross sectional area of popliteal artery. Method: The study was done on sixty healthy male volunteer subjects aged from 20 to 25 years old. They were randomized into three groups (A, B, and C). Doppler ultrasound was used to measure the blood flow volume and cross sectional area of popliteal artery. 15, 30, and 60 seconds of static stretching of calf muscle for group A, B, and C respectively. Results: There was significant increase of blood flow volume and cross sectional area of popliteal artery as p= 0.004 and 0.011 respectively among three groups. Group B that holding stretch for 30 seconds was significantly increased blood flow volume (FV) in L/min and cross sectional area (CSA) in cm2 of popliteal artery compared to other two groups (P < 0.05). Conclusion: The findings of this study suggested that using thirty seconds of static stretching for calf muscle was the most effective time duration to increase blood flow volume and cross sectional area of popliteal artery.


Key words: Static stretch; cardiovascular response to stretch; mechanoreceptors of muscle

1. Introduction

Stretching is important because it is believed to provide many physical benefits, including improved flexibility, improved muscle or athletic performance, improved running economy (decreased energy expenditure at a given speed), injury prevention, promotion of healing, and possibly decreased delayed-onset muscle soreness. Although evidence to support these beliefs is limited, stretching appears in widespread (Feland et al., 2001). Muscle stretching is commonly used in rehabilitation programs and sports activities to increase the range of motion (ROM) of a joint and, although still controversial, to prevent muscle injury (Kato et al., 2010; Lois et al., 2009). Static stretching is performed by placing muscles at their greatest possible length and holding that position for a period of time. Although documentation exists that static, ballistic and proprioceptive neuromuscular facilitation (PNF) techniques will increase the flexibility of muscle, we believe that the most common method is the static stretch (Bandy et al., 1997). Static stretching exercises are a common part of the warm-up routine of several athletes and physical activity practitioners in an attempt to improve performance and reduce the risk of injuries (Wilson et al., 2010). Despite its widespread use and some evidence of its effectiveness, an explanation as to why this stretch-based rehabilitation method may be effective is lacking (De Deyne, 2001). Within skeletal muscle, a functional microvascular bed is necessary for the provision of an adequate supply of O2 and other nutrients, as well as for removal of metabolic waste products (Padilla et al., 2006).

Precise matching of blood flow and metabolism is required for virtually all living tissues. This is especially important for skeletal muscle, where metabolism can vary considerably from periods of inactivity to periods of repeated contractions. Physical activity requires adequate blood flow to support the contractile activity of muscle fibers (Jackson et al., 2010; Clifford and Hellsten, 2004). Also the extracellular matrix (ECM) in relation to both muscle and tendon is extensively filled with blood vessels, to provide the contracting muscle with oxygen and substrate for energy production, and to ensure an efflux from musculature of combustion products (Kjaer, 2004). Stretching the
Muscle was thought to restore blood flow to the muscle and interrupt the pain spasm-pain cycle. The muscle spasm theory of muscle soreness has since been discredited, but the practice of stretching persists (Bobbert et al., 1986).

Mechanical stress caused by flexibility training (static stretching) can affect hemodynamic responses, particularly the heart rate (HR) (Farinatti et al., 2011). Passive stretch of the triceps surae muscle for 1-2 min by dorsiflexion of the foot caused a significant but slight increase in HR of 4-5 beats/min with no significant changes in mean arterial blood pressure (MAP) and forearm blood flow in humans (Matsukawa and Nakamoto, 2008). Another study reported that Passive stretch increased heart rate with a short period, that 1 min of sustained static triceps surae (calf muscle) stretch increased heart rate (Cui et al., 2006; Hayes et al., 2005).

The purpose of this study was to determine the effect of optimal time duration (15, 30, 60 seconds) of passive static stretch on blood flow volume and cross sectional area of popliteal artery, as measured by Doppler ultrasound.

2. Material and Methods

Study Design was a randomized controlled trial, performed over the period from April to June 2012 at the physiotherapy clinic of College of Applied Medical Sciences - Salman bin Abdullaziz University, Saudi Arabia.

Subjects were sixty healthy male volunteer subjects aged from 20 to 25 years old and heart rate (HR) at rest, 66 ± 9 BPM; and systolic blood pressure (SBP) at rest, 113 ± 10 mmHg. Subjects were selected and randomly assigned into three groups (A, B, and C) of equal number. They met the following inclusive criteria, which are 20 to 25 years old and non athletic subject. Exclusion criteria consisted of any pathology of hip, knee, and ankle joints, inflammatory diseases of spinal column or spinal disorders (rheumatoid disease, anklosing spondylitis, spodylolisthesis), diabetic neuropathy, intermittent claudication, any cardiac and vascular abnormality, smoker subject, and obese subject.

Outcome Measures were performed before and after passive static stretching intervention of calf muscle. Baseline demographic variables include name, age, height, weight, heart rate and systolic blood pressure were carried out. Two criteria were used to evaluate the outcome in intent to treat analysis. First, measurement was (Flow volume FV in L/min of popliteal artery) and the second measurement was cross sectional area (CSA in cm² of popliteal artery).

Instrumentation: Ultrasound scanning was performed using a high resolution digital Hitachi Hi Vision 6500 Ultrasound Machine that uses a Windows XP-based operating system, fitted with EUP-L53S 5-10 MHz. Linear array transducers. The knee joint was examined by the Technical Guidelines of the European Society of Musculoskeletal Radiology (ESSR) (Ian et al., 2012), posterior knee approach used to check popliteal neurovascular bundle.

Stretching intervention: Passive static calf stretching was done by physiotherapist before and after measurement of flow volume and cross sectional area of popliteal artery. Subject was lying supine and non dominant leg was chosen to be stretched. Therapist kept subject's knee extended supported by proximal hand of therapist. The static stretching protocol requires that the stretch be performed in a slow, gradual manner and held at end-range just before the point that causes discomfort to the patient (Farinatti et al., 2011). Stretching time (duration of hold) was 15 sec, 30 sec, and 60 sec for group A, B, and C respectively for one time using digital stopwatch to count.

3. Results

All data reported in the results were mean (± SD). Paired t-test was applied within each group for flow volume (FV) in L/min and cross sectional area (CSA) in cm² of popliteal artery. Two outcome measures of three groups were analyzed by ANOVA. A Tukey post hoc test was used to determine exactly where significant differences occurred if F-ratios were significant. A value of P < 0.05 was considered statistically significant.

There were no significant differences between patient characteristics (age, weight, height, resting heart rate, and systolic blood pressure) in three groups of the study thus the patients' selection was homogenous. Group (A) the mean age, height, and weight of the subjects was 22.95 ± 1.19 years, 73.7 ± 11.71kg, respectively. For group (B) the mean age, height, and weight of the subjects was 23 ± 1.25 years, 171.75 ± 6.56 cm, and 73.5 ± 13.87kg, respectively. For group (C) the mean age, height, and weight of the subjects was 22.7 ± 1.41 years, 172.20 ± 5.89 cm, and 74.55 ± 14.22kg, respectively. The mean changes in FV in L/min and CSA in cm² of popliteal artery for each group pre and post static stretch of calf muscle and for three groups pre and post static stretch of calf muscle were summarized in Table 1 and 2. The effect of different duration of static stretching of calf muscle within each group revealed that there were significant differences in mean changes for all measurements pre and post static stretch of calf muscle for each group (P < 0.05). Analysis of variance showed significant differences between the three groups, indicating that
the duration of stretch employed during the study significantly affected the flow volume and cross sectional area of popliteal artery. Post hoc Tukey’s HSD tests on the results showed that the (group B) holding stretches for 30 seconds more significantly increased flow volume (FV) in L/min and cross sectional area (CSA) in cm² of popliteal artery when compared with two other groups (P < 0.05). There was no significant difference observed between (group A) that holding stretches for 15 seconds and (group C) that holding stretches for 60 seconds as shown in Figure 1 and Figure 2.

Table 1. Flow volume (FV) in L/min of popliteal artery for three groups

<table>
<thead>
<tr>
<th></th>
<th>Group A Mean±SD</th>
<th>Group B Mean±SD</th>
<th>Group C Mean±SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre</td>
<td>0.0225 ±0.0137</td>
<td>0.027 ±0.0217</td>
<td>0.031 ±0.0125</td>
<td>.274</td>
</tr>
<tr>
<td>Post</td>
<td>0.039 ±0.0265</td>
<td>0.07 ±0.0411</td>
<td>0.0445 ±0.0182</td>
<td>.004</td>
</tr>
<tr>
<td>P-value</td>
<td>.004</td>
<td>.0001</td>
<td>.002</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Cross sectional area (CSA) in cm² of popliteal artery for three groups

<table>
<thead>
<tr>
<th></th>
<th>Group A Mean±SD</th>
<th>Group B Mean±SD</th>
<th>Group C Mean±SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre</td>
<td>0.1825 ±0.066</td>
<td>0.1795 ±0.0507</td>
<td>0.2185 ±0.0665</td>
<td>.995</td>
</tr>
<tr>
<td>Post</td>
<td>0.2235 ±0.072</td>
<td>0.2775 ±0.029</td>
<td>0.231 ±0.0662</td>
<td>.011</td>
</tr>
<tr>
<td>P-value</td>
<td>.0004</td>
<td>.0001</td>
<td>.0005</td>
<td></td>
</tr>
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</table>

Figure 1. Difference of mean values of FV in L/min of the popliteal artery pre and post static stretch of calf muscle for three groups.

Figure 2. Difference of mean values of CSA in cm² of the popliteal artery pre and post static stretch of calf muscle for three groups.

4. Discussion

The study was carried out to determine the most appropriate time of static stretch of calf muscle for improving blood flow in popliteal artery and increasing cross sectional area of popliteal artery. The muscle afferents that cause the cardiovascular responses are the smaller myelinated (group III) and unmyelinated (group IV) afferents. The group III afferents are mainly stimulated by mechanical stimuli like stretch, contraction or pressure (therefore termed mechanoreceptors) and respond abruptly when a muscle contracts. The majority of the group IV afferents is stimulated by metabolic or chemical products of contraction such as potassium, bradykinin and inorganic phosphate, and can therefore be termed metaboreceptors. However, both groups of afferents include receptors that are polymodal, responding to both mechanical and chemical stimuli (Gladwell and Coote, 2002).

Passive mechanical stretch of skeletal muscle causes stimulation of muscle mechanoreceptors, which increases heart rate (HR), arterial blood pressure (AP), and plasma epinephrine in anesthetized or decerebrate cats and rats. Direct assessment of autonomic outflows has revealed that passive muscle stretch increases cardiac and renal sympathetic nerve activities and decreases cardiac parasympathetic nerve activity, suggesting a possible role of the muscle mechanosensitive reflex in autonomic regulation of the cardiovascular adaptation during voluntary exercise (Matsukawa and Nakamoto, 2008).

The muscle mechanosensitive reflex may augment the sympathetic outflow controlling other
organs, because it has been reported that renal sympathetic outflow is increased by stimulating muscle mechanoreceptors during static contraction and passive static stretch in anesthetized cats. Passive static stretch of muscle predominantly stimulates mechanoreceptors but not metaboreceptors, because there are no changes in the blood, passive static stretch of muscle excites most group III muscle afferents but fails to activate the majority of group IV muscle afferents (Murata and Matsukawa, 2001). Ken Tokizawa et al. (2004) stated that the mechanical muscle deformation by triceps surae stretch may have activated mechanoreceptor in blood vessel. The mechanical distention of the peripheral blood vessel via increasing blood volume activates group III and group IV muscle afferents. Additionally, triceps surae stretch may have activated mechanoreceptor in skin and joint. It has been shown that, in simultaneous muscle stretching and contraction (which is typical of the static stretching method, due to the muscle spindle reflex), type III fibers and metaboreceptor activation may induce vagal inhibition and baroreflex stimulation and contribute to an increase in the overall cardiovascular response (Farinatti et al., 2011).

Previous study revealed that increase in heart rate (HR) at exercise onset is mainly determined by different afferent mechanisms to those determining the pressor response. The results strongly suggest that part of the effect on HR is mediated by muscle mechanoreceptors since we showed for the first time that stimulation of these receptors by sustained passive static muscle stretch selectively decreases parasympathetic activity and increases HR. This effect is due to the small group III mechanoreceptors in the muscle reflexly inhibiting cardiac vagal neurones (Gladwell and Coote, 2002).

The results of present study showed that 30 seconds of passive stretching was the most effective time in increasing blood flow and cross sectional area of popliteal artery, this comes with agreement to previous study findings that cardiac vagal outflow is decreased throughout passive stretch of the hind limb or the triceps surae muscle whereas cardiac sympathetic outflow is increased only at the first short duration of 90 seconds (Murata and Matsukawa, 2001). The findings of present study were in consistent with study done revealed that mechanically sensitive muscle afferents respond transiently to stimulation. Furthermore, the tension in the muscle elicited by stretch is higher at onset and then fades. This means that, along with a tendency for a decline in mechanically sensitive muscle afferent activation, the mechanical stimulation also declines after the onset of stretch. The progressive increase in diastolic blood pressure (DBP) during Stretch, when HR had returned to resting levels, suggests that sympathetic activation is increasing and this may indicate the sensitization of some afferents with polymodal characteristics over time (Fisher et al., 2005).

Short duration of static stretch produced an obvious effect on increasing blood flow through increasing heart rate than long duration of stretching. This finding was clarified in previous study that demonstrated static stretch of resting human calf muscle caused an immediate increase in HR. They suggested that this was caused by activation of a population of mechanoreceptive afferents, which they termed tentonoreceptors. There was evidence that the initial HR rise during stretch is mediated by immediate vagal inhibition (Drew et al., 2005).

A variety of ion channels sensitive to mechanical stimuli has been identified to serve many physiological functions, such as mechanosensation in specialized sensory cells and local control of blood flow and cellular volume in nonsensory cells. Mechanical perturbation (static exercises or passive static stretch) influences mechanosensitive ion channels in their conductive state, leading to a change in membrane potential and triggering Ca$^{2+}$ entry with a subsequent cascade of intracellular reactions (Matsukawa et al., 2007). Also, within 1 min of evoked static contraction or mechanical stretch of the muscle increased arterial plasma adrenaline and NA (noradrenaline), thus Stimulation of muscle mechanoreceptors can augment adrenal preganglionic sympathetic nerve activity (adsNA), that plasma catecholamines (plasma epinephrine) is increased by mechanical stretch of skeletal muscle (Tsuchimochi et al., 2010). These plasma catecholamines were produced by short to moderate duration of muscle stretch that used in present study produced inhibition of conducted vasodilatation of artery during muscle stretch. This was consistent with study reported that the short exercise periods and relatively small muscle masses recruited by stretching protocols represented mild vascular dilation with small exercise volume that was not capable of inducing the hypotensive response (Farinatti et al., 2011).

In addition to findings of present study that revealed 30 seconds of muscle stretch was the most efficient time to produce increasing in blood flow volume than 60 seconds, evidence may referred to strong effect of short duration of static stretch on mechanoreceptors than plasma catecholamines. This could be explained in previous study reported that passive muscle stretch for 1 to 10 minutes can attenuate conducted vasodilatation by activating $\mu$-adrenoreceptors on feed arteries through noradrenaline released from perivascular sympathetic nerves. This autonomic feedback mechanism can
restrict muscle blood flow during passive stretch (Haug et al., 2003).

Thirty seconds of muscle stretching seemed to be effective time for static stretching either for increasing range of motion (ROM) and increasing blood flow volume of feeding arteries. (Bandy et al., 1997) suggested that 30 seconds duration was an effective amount of time to sustain a hamstring muscle stretch in order to increase ROM. No increase in flexibility occurred when the duration of stretching was increased from 30 to 60 seconds or when the frequency of stretching was increased from one to three times per day.

5. Conclusion

The findings of this study suggested that using thirty seconds of static stretching for calf muscle was the most effective time duration to increase blood flow volume and cross sectional area of popliteal artery which supplied the calf muscle. Using thirty seconds of static stretching is recommended in clinics or sport medicine in case of warming up or rehabilitating a desired muscle(s).

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