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Comparison of brachial artery vasoreactivity in elite athletes and age-matched controls

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COMPARISON OF BRACHIAL ARTERY VASOREACTIVITY IN ELITE ATHLETES AND AGE-MATCHED CONTROLS

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ABSTRACT

The ability to distribute blood from areas with low demand to areas with high demand, such as occurs during exercise, is a critical function of the circulatory system. PURPOSE: To compare the resting diameter and vasoreactivity of the brachial artery in elite strength athletes to those of age-matched controls. We hypothesized that the brachial arteries of strength athletes would have larger diameters at rest, and show greater vasoreactivity in response to cuff occlusion and a cold pressor test than in untrained individuals. METHODS: Eight elite strength athletes (age, 23 ± 2 years) and ten age-matched controls (age, 22 ± 1 years) were studied. Using high-resolution ultrasonography, brachial diameter was assessed at rest and following 5 minutes of forearm occlusion (BAFMD) and a cold pressor test. RESULTS: The average resting brachial diameters of strength athletes (5.39 mm ± 1.51) was significantly larger than the diameters of the control group (3.73 mm ± 0.71). On average, strength athletes showed significantly greater vasodilation (BAFMD % Δ athletes, 8.21% ± 1.78; controls 5.69% ± 1.56) in response to cuff release and significantly greater vasoconstriction (CPT % Δ athletes, -2.95 ± 1.07; controls -1.20 ± 0.48) in response to the cold pressor test. The combined effect of vasodilation and vasoconstriction indicates a greater physiologic vascular operating range in the athletes (0.55 mm) compared to the controls (0.25 mm). CONCLUSIONS: This study reports significant differences in vascular responses to vasodilatory and constrictor stimuli of elite strength athletes and age-matched controls. These differential responses in the elite athletes suggest a well-adapted vasculature defined by a wide vascular operating range.

INTRODUCTION

In 1794, John Hunter stated that "blood goes where it is needed" (1794). John Hunter's intuition was often superb; even though he was left wondering how a system "knew" where flow was needed and how the right amount got to the right place. This most important concept remains critical and at the forefront of cardiovascular physiology today. Specifically, the body must be able to distribute blood from areas with low demand to areas with high demand, such as occurs during exercise, efficiently and quickly, to ensure adequate performance. Traditionally, the blood flow distribution that occurs with exercise is referred to as 'Functional Sympatholysis' and involves vasoconstriction and localized dilation to establish a path of least resistance for blood to follow. Arguably, those with the greatest ability to quickly and efficiently establish a path of least resistance for blood to travel to the working muscle have the highest cardiorespiratory capacity.

Over the past decade, the lab in the Department of Kinesiology at Louisiana State University has focused its research efforts on understanding the vasodilatory responsiveness under a variety of conditions and in several different populations (Arce 2008, Bahadir 2004, Credeur 2009, Dobrosielski 2007, Lestage 2006, Tisdell 2004, Welsch). Typically, younger, fitter, healthier individuals demonstrate greater vasodilatory responses than their counterparts. In addition, several previous studies have shown that localized exercise training protocols enhance vasodilation in large conduit arteries and contributes to greater regional blood flow responses. Although, the lab's focus has uniquely contributed to understanding how exercise can enhance vascular and physical function, the lab's agenda remained unilateral. Arguably the ability of a blood

vessel to dilate and constrict may provide important information about the vessel's "physiological" operating range. Such information may lead to a better understanding of the manner in which blood flow is distributed within the body during times of increased metabolic stress, such as with physical exercise.

A review of the critical factors that determines the vasoconstrictor/dilation properties of the vasculature is somewhat beyond the scope of this thesis. For a detailed explanation of vascular control the reader is referred to several exhaustive reviews available in the LSU ETD. In principle circulatory function is governed by: 1) the local tissue metabolic demand, 2) vascular resistance and 3) the blood pressure needed to maintain adequate perfusion of all organs (Rowell).

Briefly, vascular control is governed by two important factors, Poiseuille's and Ohm's Law. Poiseuille's Law states . . .

$$\underline{Q = \pi \, \Delta P \, r^4}$$

$$8 \, \eta \, l$$

. . . whereby, Q is blood flow, ΔP is the pressure difference over the vessel length, r is the radius of the vessel, l is the vessel length, and η is the viscosity of the blood. It is apparent that radius has the greatest effect upon blood flow considering the length of the vasculature and blood viscosity does not change transiently. Specifically, a 4-fold increase in vessel radius causes a 256-fold increase in blood flow (Guyton 2000).

The second factor is based on the derivative of Ohm's Law which states . . .

$$\Delta P = Q * R \ldots$$

. . . whereby, ΔP = the change in pressure across a system or vessel, Q = cardiac output or flow, and R = resistance to flow. Therefore, the most efficient way to increase pressure in a system or vessel is to increase flow by increasing cardiac output, increase resistance to flow through peripheral vasoconstriction, or both (Guyton 2000).

The major vascular control mechanisms responsible for central blood flow and pressure regulation include neurohormonal control, metabolic and intrinsic regulation and muscle afferents. Extrinsic neural control is directed toward maintaining blood pressure whereas local regulation of blood flow is directed toward meeting tissue requirements.

Neurohormonal controls include the baroreceptor reflex which responds to changes in arterial pressure; the hormone vasopressin in the medium-term (hours); and the renin-angiotensin-aldosterone axis in the long-term (days to weeks). The sympathetic branch of the autonomic nervous system is almost entirely responsible for nervous control of circulation. The sympathetic neurons release norepinephrine (therefore referred to as adrenergic), which binds to α receptors on the cell membrane, where a vasoconstrictor response is initiated. Local control is mediated via the myogenic reflex (the contraction of a blood vessel that takes place when intravascular pressure is elevated thus allowing for flow through the vessel to remain constant despite increases in arterial pressure), endothelial control (synthesis or release of vasoactive and thromboregulatory factors such as nitric oxide, endothelial derived hyperpolarizing factor, bradykinin, thromboxaine, endothelin and tissue-type plasminogen activator), and metabolic control [vasodilator substances (e.g. adenosine, adenine nucleotides, carbon dioxide, potassium, phosphate ions, lactate and Krebs cycle intermediates), which are released from active

muscle fibers, diffuse through the interstitial space to act on the smooth muscle cells of arterioles and increase blood flow].

The majority of studies that have examined the role of exercise on vascular function have typically focused on "aerobic" activities, and on vasodilatory properties. Much less research has examined the possible role of resistance training on vascular function. Given recent emphasis on including resistive exercises in a well-rounded program, more information about the role of resistance exercise and vascular health is warranted. Many studies have been conducted that indicate chronic heavy resistance training causes increases in muscle hypertrophy, strength, and bone density (Bravo et al., 1996, McDonagh and Davies, 1984). However, resistance training also improves insulin sensitivity (Ettinger et al. 1994), resting metabolic rate and neuromusclar recruitment. In regards to cardiovascular adaptations the overriding view still focuses on the potential hazards of resistance training, dating back to the 1967 study by Lind and McNichol. More recent studies by MacDougall et al. (1992), report that subjects performing heavy dynamic weightlifting exercises see extreme increases in arterial blood pressure. These increases in pressure are attributed to factors including a potent pressor response, an increase in heart rate and cardiac output, mechanical compression of blood vessels, the Valsalva maneuver (closing the glottis while contracting the expiratory chest and abdominal muscles), and the intensity of the effort. Interestingly, one might also argue that athletes who can handle enormous changes in blood pressure during their events must have a very well-adapted vasculature (MacDougall et al. 1992).

Study Purpose

The purpose of this study was to compare peripheral vascular responses to

vasodilatory and constrictor stimuli in elite strength athletes and age-matched controls during a single visit using high-resolution ultrasonography. Specifically, this study examined heart rate, blood pressure and blood flow responses at rest, following occlusion and a cold stimulus. While this study cannot state absolutely any differences seen are a direct result of adaptation to strength training, it is hypothesized that elite strength athletes will show greater resting blood flow and greater vasoreactivity in response to cuff occlusion and a cold pressor test than in untrained individuals. In addition, it is hypothesized that trained individuals will have increased lumen diameters at rest to account for the increased oxygen demands of greater lean muscle mass than untrained individuals. It is further hypothesized that a single conduit artery will show greater contraction and dilation in response to stimuli in trained individuals as a further means of meeting oxygen demands.

METHODOLOGY

Study Participants

All participants were given information about the risks and benefits of the study procedures and signed a participation consent approved by the host institution. Participants could not have any overt signs or symptoms of disease. The main inclusion criteria for the athletes were a nationally or internationally ranking in their respective sports; and sports that primarily required the implementation of high intensity, dynamic resistance training. The control subjects were recruited from current students enrolled in laboratory classes in the Department of Kinesiology at Louisiana State University. Participants were asked to refrain from alcohol for 24 hours, fast for 12 hours and refrain from intense training for 8 hours prior to participating.

Experimental Design

The study was a cross-sectional design comparing fitness profiles and vascular responses in elite strength athletes and age-matched controls. The experimental procedures consisted of basic cardiorespiratory and strength fitness and vascular measurements. The experimental procedures were conducted over the course of one visit. Vascular tests were conducted first, followed by the basic fitness tests.

Experimental Procedures

Ultrasound Measurements

All brachial artery imaging and analyses were conducted in accordance with the Guidelines set forth by the Brachial Artery Reactivity Task Force (Corretti et al. 2002). Brachial artery ultrasound measures (Toshiba Power Vision SSA-380A) were obtained with participants in the supine position using a 7.5-MHx linear array transducer prior to,

during and following five minutes of forearm occlusion. Prior to scanning, participants were instructed to fast and refrain from exercise and alcohol intake for 24 hours. Baseline ultrasound images were obtained after 10 minutes of supine rest. All images were obtained in the longitudinal view, approximately 4 cm proximal to the olecranon process, in the anterior/medial plane. Image depth was initially set at 4 cm gain; settings were adjusted to provide an optimal view of the anterior and posterior intimal surfaces of the artery and kept constant throughout. The participant's arm was immobilized and slightly supinated.

For this study the vasodilatory stimulus was induced through forearm occlusion consisting of inflation of a blood pressure cuff, positioned approximately 1 cm distal to the olecranon process, to 240 mm Hg for five minutes. Upon release of the occluding cuff, the reactive hyperemic response is thought to represent a trigger for vasodilation. The vasoconstrictor stimulus for this study was a cold pressor test consisting of a one-minute submersion of the right hand in ice water, with brachial constriction being monitored on the left forearm. After the one-minute submersion, the right hand was removed from the water.

Images for vessel diameter and velocity profiles were obtained at rest, and continuously from the final 30 seconds of occlusion until five minutes following the release of the blood pressure cuff and the cold pressor test. In addition, blood pressure and heart rate were monitored throughout the imaging process. All ultrasound images were recorded on compact discs for subsequent analysis.

Fitness Measurements

Height, weight and forearm circumference measurements were obtained on all participants. Following the occlusion and cold pressor tests, hand grip and step tests were performed by each participant. Hand grip was obtained using a hand grip dynamometer (Baseline Hydraulic Hand Dynamometer by Fabrication Enterprises Inc.). The dynamometer was held straight out at arm's length and the subject squeezed as hard as possible for several seconds and the highest value obtained was recorded. Each participant performed three trials with their left hand. Participants ended their visit by performing the 3-Minute YMCA step Test (ACSM's Guidelines For Exercise Testing And Prescription). This test was utilized in order to provide an unbiased fitness measure between the two groups. Subjects stepped onto and off of a 12-inch (30.5-cm) bench at a stepping rate of 24 steps·min^{-1}. At the end of three minutes of stepping, heart rate was taken immediately (within 5 seconds of the end of exercise), at one minute, two minutes and three minutes post-exercise so that heart rate recovery could be measured.

Data Analysis

The Brachial Imager Software (Medical Imaging Applications, LLC) was used to analyze the images. Arterial diameters were calculated as the mean distance between the anterior and posterior wall at the blood vessel interface, with the image in diastole, defined as the peak of the R wave on the ECG. Base diameter (BASE) was defined by the average of 30 seconds of data obtained after 10 minutes of resting conditions. Peak dilation (PEAK) and constriction (NADIR) were defined (by visual inspection of the arterial diameter curve) as the largest and smallest diameter following release of the occluding cuff and following the cold pressor test. Its value was calculated by the

average of 10 images (five seconds) surrounding this highest observable peak or nadir. BAFMD was defined as the absolute (mm) and percent change in vessel diameter from BASE to PEAK. Finally, the vascular operating range was calculated as the absolute change in diameter from peak dilation to peak constriction.

Statistical Analysis

All statistical analyses were performed using SPSS for Windows (version 16.0). Data are presented as mean ± standard deviation. To compare the brachial artery dimensions, flow velocity integrals, and fitness scores of elite strength athletes and age-matched controls a univariate analysis was used. To determine changes in brachial diameter before and after reactive hyperemia and the cold pressor test, a repeated measure ANOVA was used. To examine the heart rate recovery following the step test a second repeated measure ANOVA was used. An alpha level of $p<0.05$ was required for statistical significance.

RESULTS

Participant Characteristics

Eighteen adults (8 Men and 10 Women) between the ages of 19 and 26 years old participated in this study. Participant characteristics are presented in Table 3.1.

Table 3.1 Participant Characteristics

		Minimum	Maximum	Mean	SD
Age (yrs)	**Athletes**	**19**	**25**	**22.75**	**1.98**
	Controls	**20**	**26**	**22.30**	**1.42**
Height (in)	**Athletes**	**60**	**74**	**66.69**	**4.11**
	Controls	**60**	**74**	**66.4**	**4.14**
Weight (lb)	**Athletes**	**132**	**274**	**192.63***	**45.98**
	Controls	**108**	**205**	**146.1**	**33.49**
Forearm Circ. (cm)	**Athletes**	**25.7**	**32.8**	**29.61***	**2.92**
	Controls	**21.6**	**30.1**	**25.16**	**3.61**
SBP rest (mmHg)	**Athletes**	**110**	**150**	**123.71**	**12.93**
	Controls	**120**	**150**	**129.1**	**10.89**
DBP rest (mmHg)	**Athletes**	**70**	**84**	**78.86**	**5.52**
	Controls	**62**	**98**	**81.2**	**11.36**
HR rest (bpm)	**Athletes**	**55**	**66**	**61.5†**	**4.65**
	Controls	**50**	**85**	**71.56**	**10.94**

***p<0.05 vs. Controls; †p<0.10 vs. Controls**

Fitness Characteristics

Table 3.2 contains fitness characteristics for the average of the three grip strength trials and the heart rate measurements following the step test (ST HR). Upon further examination the decline in heart rate following the step test was not significantly different between the groups. The heart rate responses following the test are depicted in Figure 3.1.

Table 3.2 Fitness Characteristics

		Min	Max	Mean	SD
Grip Ave. (kg)	**Athletes**	**33.67**	**82**	**55.92***	**17.06**
	Controls	**24.33**	**63**	**36.77**	**14.49**
Peak Exercise HR (bpm)	**Athletes**	**100**	**140**	**123**	**16.25**
	Controls	**84**	**156**	**129.6**	**25.14**
HR 1 Min Recovery (bpm)	**Athletes**	**60**	**116**	**88**	**20.62**
	Controls	**52**	**136**	**97.6**	**26.14**
HR 2 Min Recovery (bpm)	**Athletes**	**56**	**108**	**79.5**	**18.69**
	Controls	**52**	**128**	**85.6**	**22.45**
HR 3 Min Recovery (bpm)	**Athletes**	**60**	**100**	**78**	**14.34**
	Controls	**56**	**124**	**81.6**	**21.08**

***p<0.05 vs. Controls**

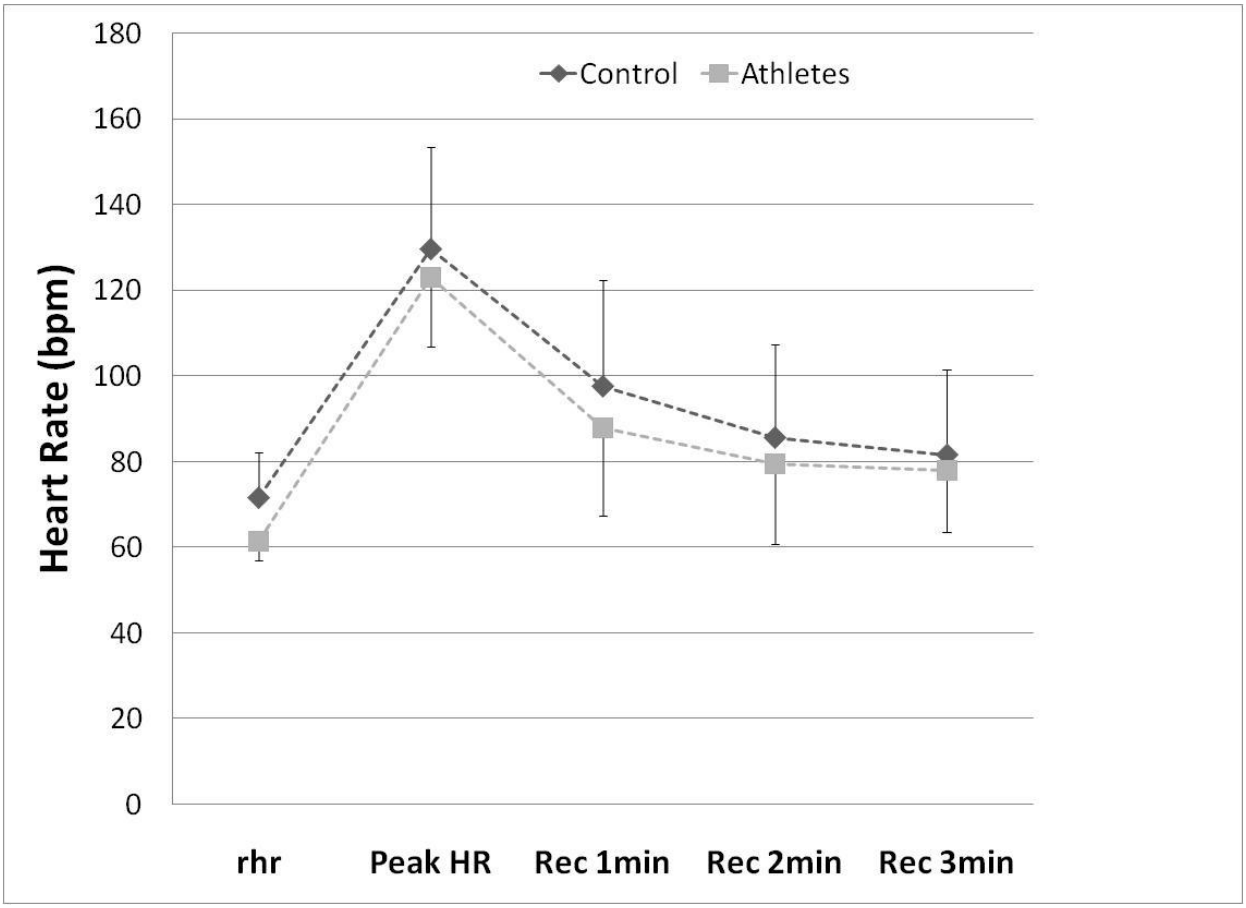


Figure 3.1. This graph displays the mean heart rates for the strength athletes and controls prior to and following the step test.

Vascular Responses

Vascular responses, presented in Table 3.3, include resting blood flow (RBF), resting diameter, reactive hyperemia diameter, the percent change in diameter from base to peak dilation (BAFMD % Δ), diameter at peak constriction (CPT Dia 60), and the percent change in diameter following the cold pressor test (CPT % Δ). Also, a vascular operating range was calculated the difference from peak dilation to peak constriction.

Table 3.3 Vascular Responses

		Minimum	Maximum	Mean	SD
RBF (mL·min-1)	**Athletes**	**10.8**	**31.46**	**16.7**	**6.49**
	Controls	**8.53**	**22.72**	**14.47**	**4.64**
Rest Dia (mm)	**Athletes**	**3.95**	**7.1**	**5.39***	**1.51**
	Controls	**3.14**	**5.05**	**3.73**	**0.71**
RH Dia (mm)	**Athletes**	**4.15**	**7.62**	**5.84***	**1.65**
	Controls	**3.32**	**5.32**	**3.94**	**0.75**
BAFMD % Δ	**Athletes**	**5.06**	**10.00**	**8.21***	**1.78**
	Controls	**2.84**	**7.32**	**5.69**	**1.56**
CPT Dia 60	**Athletes**	**3.80**	**6.95**	**5.29**	**1.52**
	Controls	**3.11**	**5.02**	**3.70**	**0.72**
CPT % Δ in Diameter	**Athletes**	**-1.70**	**-4.76**	**-2.95***	**1.07**
	Controls	**-0.49**	**-1.80**	**-1.20**	**0.48**
Vascular Operating Range (mm)	**Athletes**	**0.35**	**0.72**	**0.55***	**0.15**
	Controls	**0.11**	**0.32**	**0.25**	**0.78**

***p<0.05 vs. Controls; †p<0.10 vs. Controls**

Figure 3.2 shows brachial artery flow velocities (cm/sec) during reactive hyperemia and the cold pressor test for the strength athletes and controls.

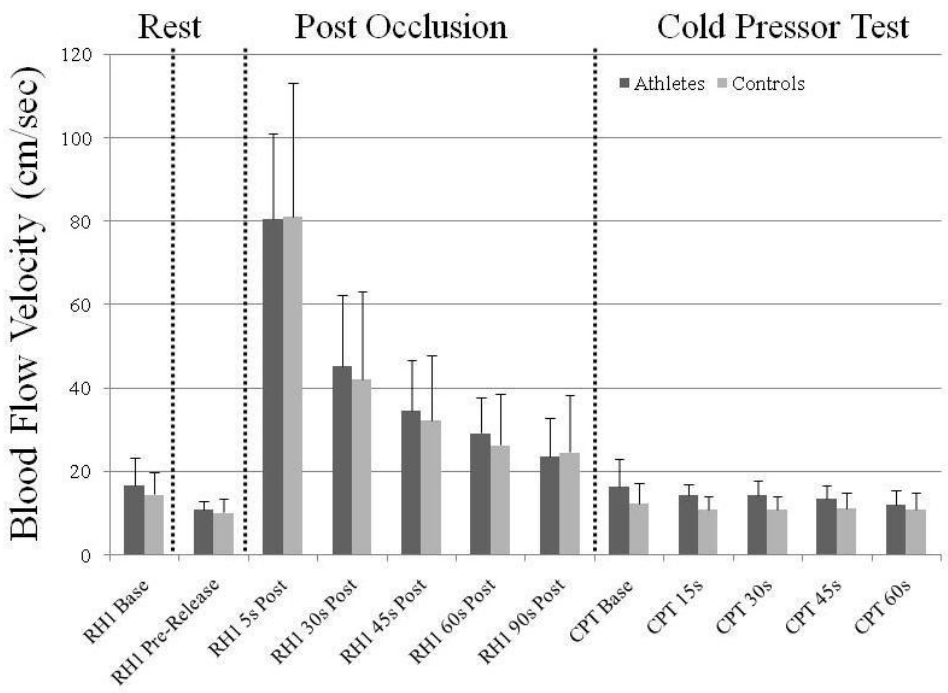


Figure 3.2. Shows brachial artery flow velocities (cm/sec) during reactive hyperemia and the cold pressor test for the strength athletes and controls

The mean percent change in the brachial artery diameter after cuff occlusion in strength athletes was 8.21 ± 1.78 %, and 5.69 ± 1.56 % in the control group. The mean percent change in the brachial artery diameter following the cold pressor test in the strength athletes was –2.95 ± 1.07 % compared to –1.20 ± 0.48 % in the control group. The percent changes in the brachial artery diameter are further depicted in Figure 3.3.

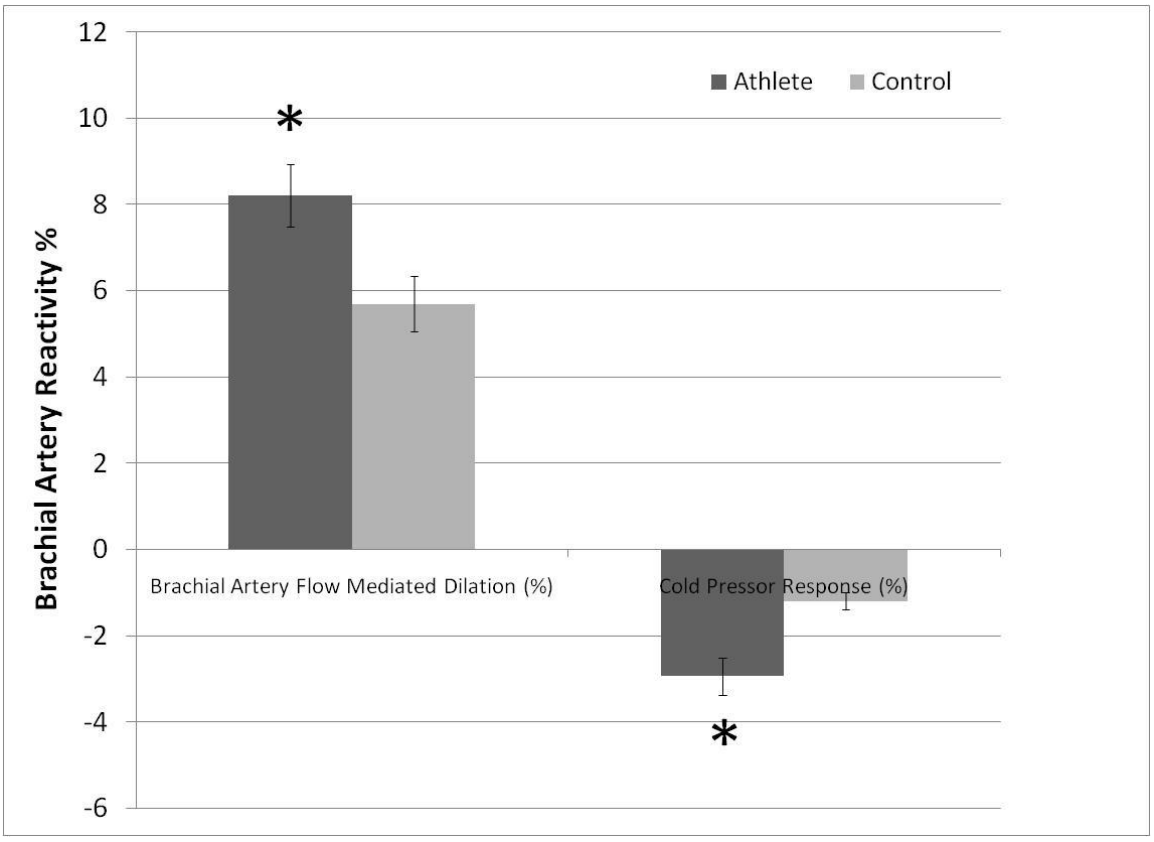


Figure 3.3. Percent change in (± standard error) brachial artery diameter following cuff occlusion and a cold pressor test in strength athletes and controls.

Given the above changes the vascular operating range (the absolute change in diameter from peak dilation to peak constriction) was significantly different ($p<0.001$) in the between the athletes (0.55mm) and controls (0.25mm). The vascular operating range for the athletes and controls is depicted in Figure 3.4.

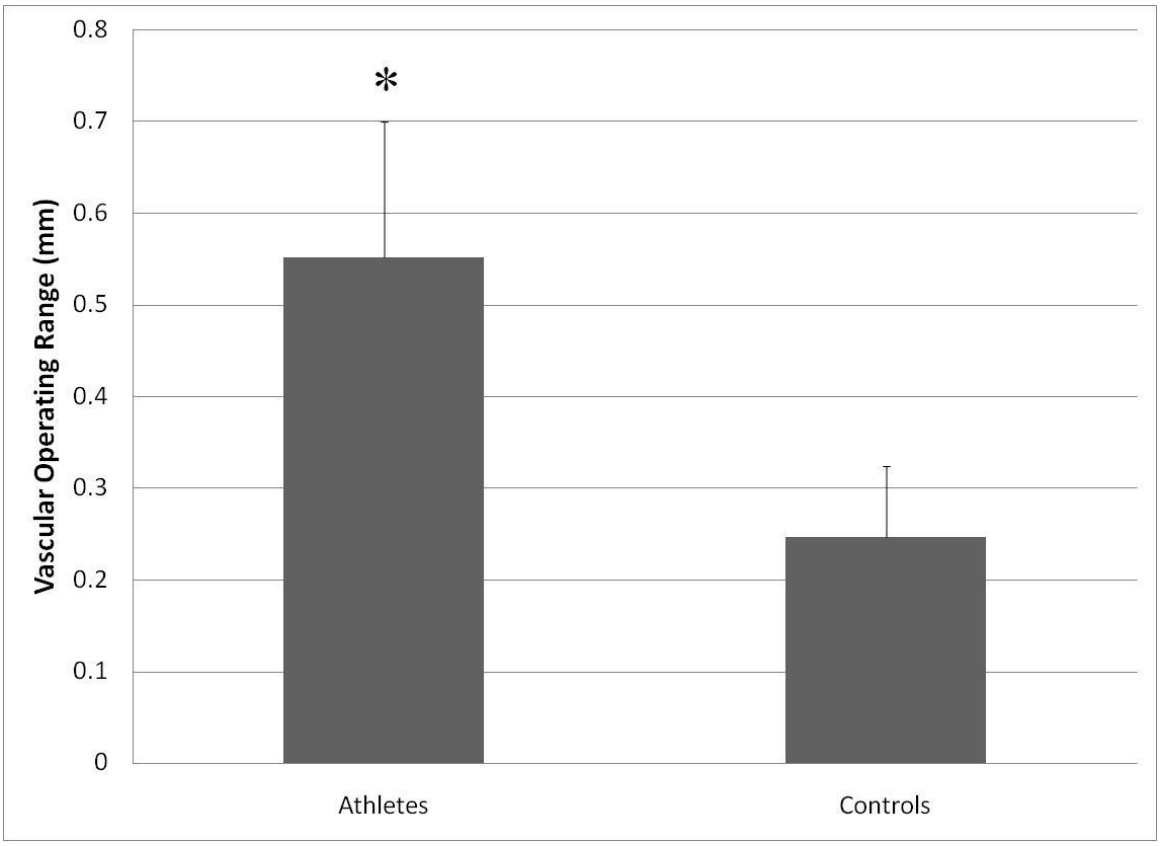


Figure 3.4. Vascular operating range (± standard error) for the brachial artery following cuff occlusion and a cold pressor test in strength athletes and controls.

DISCUSSION

This study examined the temporal pattern of brachial artery diameter and flow mediated dilation after five minutes of forearm occlusion and a one-minute cold pressor test. The unique finding of the study is that there are indeed significant differences between strength athletes and age-matched controls in vascular responses to both a vasodilatory and constrictor stimulus. The findings indicate the brachial artery of athletes have a significantly greater vascular operating range under physiologic conditions compared to non-athletes. This greater vascular operating range in the predominantly strength trained athletes compared to the controls is evident despite both groups having similar whole-body cardiorespiratory fitness measures.

Participant Characteristics

The strength athletes included in the study had significant training experience. In fact, the average training time for these individuals was ~6 years. All athletes were either nationally or internationally ranked in their respective sports. The athletes competed in Olympic weightlifting, power lifting or the hammer throw. All the athletes reported that the majority of their training consisted of heavy dynamic strength training with little or no emphasis on cardiovascular conditioning. The athletes reported frequent training sessions of 2-3 hours, 3-5 days/week. In contrast, the control subjects were selected from the student body at Louisiana State University. Their fitness profile indicates an average to above average fit group for the step test (Golding 1989) and handgrip strength (Sparling 1997).

Brachial Artery Size and Vasodilatory Response

The findings of this study indicate that the strength-athletes had significantly larger resting brachial diameters compared to the non-athletes. This finding is consistent with several other reports for rowers (Naylor 2006), elite strength athletes (Babaee 2007), and elite endurance athletes (Kasikcioglu 2005). Likely, larger conduit arteries in athletes may be due to greater amount of lean mass that the large conduit artery has to supply. A possible mechanism responsible for exercise training–mediated conduit vessel enlargement relates to the effect of episodic increases in flow and shear stress related to exercise training. A study by Langille and O'Donnell showed that a 70% ligation-mediated decrease in flow in rabbit carotid arteries resulted in a 21% decrease in artery diameter within 2 weeks. The smooth muscle relaxant papaverine did not attenuate the response; therefore, such reductions in diameter probably reflect a structural modification of the arterial wall rather than sustained contraction of smooth muscle. In addition, this change in vascular structure was dependent on the presence of the endothelium, indicating that changes in vessel structure secondary to long-term changes in flow may be dependent on the release of a labile factor from endothelial cells. These findings are consistent with the evolving hypothesis that exercise training induces structural enlargement of conduit vessels, which is dependent on shear stress–mediated nitric oxide (NO) release and may be an adaptive response that acts to mitigate the increases in wall stress brought about by repeated exercise bouts (Laughlin 2008).

Other reasons for arterial enlargement may be pressure or neurally related. For example, there is some belief that pressure overload during high intensity resistance training may also contribute to greater arterial dimensions (Babaee 2007).

In regards to the vasodilatory response, the findings of this study confirm previous reports from our lab (Allen 2002; Dobrosielski 2007) in such that the brachial diameter gradually increased from baseline to a peak and subsequently returned to baseline values, within approximately 60 seconds after release of the occluding cuff. The magnitude of BAFMD for the control group was in fact quite similar to Allen et al (Present study 5.7 ± 1.56 vs. Allen 6.20 ± 3.04), although a bit less than what was reported by Dobrosielski et al. (7.7 ± 3.5). Uniquely, the present study identifies that the elite strength athletes had a significantly greater BAFMD response compared to the controls. Typically, superior BAFMD is seen in elite endurance trained athletes. The belief is that the exercise induced modulation in shear stress triggers endothelium-dependent mechanisms to modify and contribute to a more favorable phenotypic expression of vasodilation. The fact that the strength athletes in the present study have superior BAFMD, despite having larger vessels is perhaps somewhat surprising. Usually the vasodilatory response in a larger vessel is less than that observed in a smaller vessel (Dobrosielski 2007). This makes physiologic sense in that a large vessel does not need to increase its diameter by much to see a significantly greater blood flow (Poiseuille's Law). Thus the fact that the elite athletes in this study did have a significantly greater BAFMD than controls may indicate a highly adaptive endothelium. Perhaps the manner in which the endothelium adapts in strength athletes is as follows: When performing heavy resistance training, blood flow is often drastically reduced or cut off completely by mechanical compression of blood vessels by the contracting skeletal muscle. As a lift ends, and the involved muscles relax, blood flow accelerates through the region thereby increasing the hemodynamic forces (i.e. shear stress) on the inner walls of blood vessels.

The surface endothelium of a blood vessel contains stretch receptors that react to elevations in wall shear by releasing vasoactive substances (e.g. nitric oxide (NO)), which cause smooth muscle relaxation and subsequent vasodilation (Thijssen 2005). While this study used the release of cuff occlusion to stimulate shear stress, long-term resistance training could contribute to vascular modifications secondary to elevated shear stress during the acute training sessions. These adaptations could take the form of an increase in the amount of stretch receptors on the endothelium, or possibly by an increase in the amount of NO released.

Cold Pressor Response

This study also validates previous findings (Lestage 2006) from this laboratory which examined the blood flow changes during reactive hyperemia and a cold stimulus. In that study average blood flow responses at rest and following calf occlusion, exercise, and a cold pressor test were as follows: resting arterial inflow, 2.27±1.06; post-occlusion peak blood flow, 19.42 ±6.37; post-exercise blood flow, 27.37±14.95; and blood flow following the cold pressor test, 1.53±0.89 ml/100ml/min. The flow responses following plantar flexion were significantly higher than reactive hyperemia, which were significantly greater than blood flow at rest, which were significantly greater than the flow response following the cold pressor test. Interestingly, there were significant associations between peak post-occlusion blood flow and post exercise blood flow (r^2=0.55), and between the change in blood flow following the cold pressor test and peak post-occlusion blood flow (r^2=0.25), and post exercise blood flow (r^2=0.37).

The physiological mechanism causing a decrease in blood flow following the cold pressor test is believed to be vasoconstriction, secondary to increased alpha adrenergic

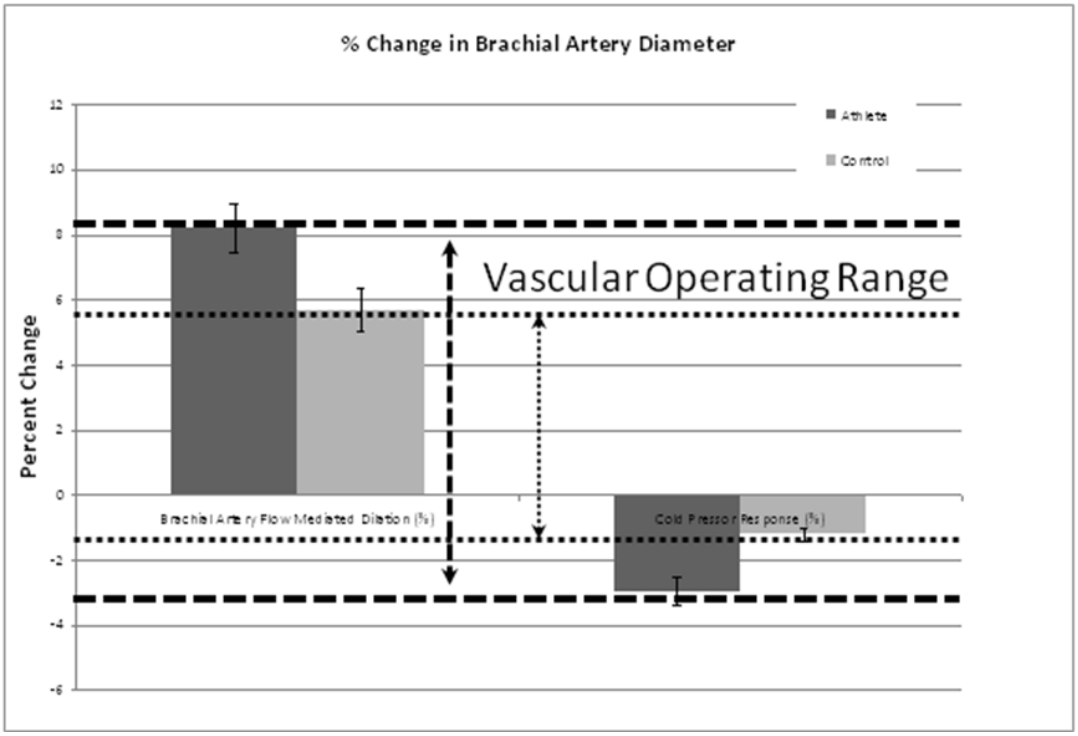
agonists (Stein 2000). Previous work has shown an increase in heart rate and blood pressure with a decrease in blood flow to the peripheral vasculature during a cold stimulus (Koch 2003; Dishman 2003; Lestage 2006). This indicates a change in central control factors. Kuniyoshi 2003 demonstrated that rapid changes in the autonomic nervous system during a cold stress increases the muscle sympathetic nerve activity in the body, which can then lead to vasoconstriction of blood vessels in the musculature. The blood flow responses by both the athletes and the controls follow this trend pointing to an increase in the autonomic nervous system during the cold stimulus.

The unique finding of the present study is that the strength athletes showed a significantly greater response to the cold pressor test than the controls by decreasing flow 2.95% in the brachial artery compared with only a 1.2% decrease in controls. Does this mean there are more receptors, more sensitivity, more NE and Epi, a combination of these factors, or some other as yet unidentified factors? Although this study's findings showed that there apparently is greater vasoconstriction in the brachial artery of the strength athletes, this study does not allow generalization of this phenomenon beyond this conduit artery. However, it does lead to a provocative speculation that would suggest that these athletes may have a superior ability to selectively vasoconstrict arteries to areas where demand for blood is low (e.g. gastrointestinal tract while exercising). The mechanisms responsible for the greater vasoconstriction seen in the athletes are again likely both CNS dependent and shear stress regulated via vasoactive molecules.

Vascular Operating Range

The combined effect of greater vasodilation and constriction allows interpretation of the conduit arteries vascular physiological operating range. This study uniquely

identifies that this operating range is quite different between the athletes and controls. In fact, the vascular operating range of athletes (0.55 mm) is twofold greater than controls (0.25 mm). Figure 4.1 diagrams the vascular operating ranges of the strength athletes versus the controls by showing the percent changes in brachial artery diameters from peak dilation due to forearm cuff occlusion to peak constriction via the cold pressor test.



The concept of the vascular operating range is unique, and therefore its meaning not completely clear at this point. However, if we reconsider John Hunter statement that "blood goes where it is needed" we must appreciate that distribution of blood flow in the body is a consequence of vasoconstrictor and dilatory properties. The fact, the present data indicates that the vascular operating range in strength athletes is much greater compared with age-matched controls may provide an initial glimpse about the vascular benefits obtained with strength training. For example, one could argue that greater vasoreactivity indicates a significant advantage for strength athletes to shunt blood where

they need it, when they need it. This in turn should yield better results in athletic performance. Again it is important to recognize that the higher vascular operating range in strength athletes may points toward yet another benefit of resistance training that has received little attention until now. While it is commonly accepted that resistance training causes muscle hypertrophy, increases bone density, and strengthens joints, endurance training is more closely associated with vascular benefits than strength training. Even though this study does not compare the relative vascular responses between endurance trained athletes and strength athletes, it is still clear there is a significant difference between strength trained individuals and untrained individuals.

Relevance

Most previous research has focused on only one property of the vasculature, using either a vasodilator or vasoconstrictor stimulus. The purpose of this study was to manipulate the vasculature using different stimuli to learn more about vasodilator and vasoconstrictor properties of the cardiovascular system. Using cuff occlusion to reach peak dilation and the cold pressor test to obtain peak constriction, a vascular operating range was calculated simulating normal physiological conditions. Also this study found significant differences in brachial artery size and responses between elite strength athletes and a control group. These differences are important because they suggest that heavy resistance training does affect the vasculature and blood flow distribution, a benefit previously downplayed or overlooked completely. This study will allow a better understanding of blood flow distribution, a combination of vasodilation and vasoconstriction, as it relates to performance. Also, it is probable that these results have clinical implications, for improved target treatments, as well.

Limitations

It is important to remain cautious regarding what interpretations are made from these findings due to inherent limitations of the study. Due to the difficult nature of locating elite strength athletes and insuring there is no conflict between participating in the study and their training regimes, the population size for the study was small. Further, since this study consisted of only one visit, it is recognized that longitudinal studies are needed to establish a cause and effect relationship between resistance training and the vascular responses seen here. The intention of the lab at Louisiana State University is to continue efforts along these lines to account for these shortcomings in future studies. However, the present observations contribute to the existing literature as it identifies several unique aspects that warrant further discussion and research.

This begs the question though of whether the athletes' blood vessels grew larger to accommodate muscle hypertrophy caused from strength training, or whether the athletes naturally had larger blood vessels to begin with, that then allowed the athletes to reach elite levels of strength.

CONCLUSIONS

The purpose of this study was to compare the peripheral vasculature of elite strength athletes and age-matched controls during a single visit using high-resolution ultrasonography. The unique findings of this study were that there were significant differences between the brachial arteries of strength athletes and controls, with athletes having significantly greater resting diameters and significantly greater vasodilation and vasoconstriction in response to occlusion and a cold stimulus. Also athletes showed a significantly greater increase in blood flow after occlusion and a significantly greater decrease in blood flow following CPT. All together, these responses resulted in elite strength athletes having a significantly greater vascular operating range than controls for everyday activities.

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