CARDIOVASCULAR RESPONSES IN

RUNNERS, POWER ATHLETES, BODYBUILDERS,

AND HEALTHY SEDENTARY SUBJECTS

Thomas Leslie Babits
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CARDIOVASCULAR RESPONSES IN

RUNNERS, POWER ATHLETES, BODYBUILDERS,

AND HEALTHY SEDENTARY SUBJECTS

By

Thomas Leslie Babits

A Thesis Submitted in Conformity with the Requirements for the Degree of Master of Science Graduate Department of Community Health University of Toronto

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Abstract

Across a wide range of health and fitness, a linear relationship exists between Gmax and VO2peak. Gmax values have not been obtained for those elite athletes whose performance is dependant on reaching their highest possible VO2max, or in bodybuilders.

VO2max, Gmax, and MAP, at rest and following calf ergometry at submaximal and maximal workloads, in age and sex matched elite middle distance runners (ER), body builders (BB), power athletes (PA) and healthy sedentary subjects (HS) was compared.

Grest was significantly higher in BB than in all other groups, while Gmax was higher in BB and ER than PA and HS. A strong linear relationship was found in BB (r = 0.94, p < 0.002) between Gmax and Grest, but not in any of the other groups. A strong linear relationship was found, across the non-resistance trained groups (ER, HS) (r = 0.84, p < 0.0001).

This study shows that within the vasculature of trained muscle, adaptive processes involved in body building are qualitatively similar to those found in ER, and different from those resulting from a traditional strength training model.
In memory of my Grandparents

a different time.....a different place
Acknowledgments

This thesis could not have been prepared without the help of many colleagues, family, and friends.

I would like to thank all the members of my thesis examination committee; Dr. L. Goodman, Dr. B. Goode, and Dr. S. Thomas, who worked on my behalf on such short notice. I greatly respect the care taken in suggesting improvements, which have clearly strengthened the work.

I was fortunate to be accepted by Dr. Jack Goodman following my project proposal and a disappointing initial experience at graduate school. As my supervisor, he patiently handled my occasional overzealousness, and helped to shape the thesis. Special thanks are extended to Dr. Mike Plyley, who not only helped with the development of the ideas and writing of the thesis, but years ago, rekindled my fascination with the potential of the human body, and brought reason to the world of science.

Instrumental in the completion of this thesis was the encouragement and feedback of my fellow graduate students. I would especially like to thank Rich Evans, and Dr. Russ Hepple. Their support was truly invaluable. Russ in particular has greatly influenced the ideas in the thesis.

This thesis would not have been possible without the support of my family, especially my parents, George and Kathy Babits. For nearly thirty years they have devoted themselves to ensuring I receive a balanced and meaningful education, and that I strive for excellence in the areas I chose. This work represents your efforts as much as it does mine. Thank you.

Finally, I would like to thank my most patient and understanding wife, Trisha. The thesis has been a part of our entire married life together, and with all the changes in life that have occurred over the past three years, her love and support was instrumental in its completion. Another dream attained...thanks!
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Chapter 1
INTRODUCTION

1.1 Purpose

The purpose of this study was to characterize maximum oxygen uptake, skeletal muscle blood flow and blood pressure responses at rest and following both submaximal and maximal calf ergometry in athletes who have undergone one of two forms of resistance training (power training or bodybuilding) and endurance training, compared to healthy untrained individuals.

1.2 Rationale

It has been clearly demonstrated that across a wide range of health and fitness, a strong linear relationship exists between the maximal vascular conductance (G max) of the calf, and maximal oxygen uptake (VO₂max) (Reading, 1993, Snell, 1987). However, for high caliber athletes, in which athletic performance is dependent on the development of the greatest possible maximum oxygen uptake, maximal lower leg vascular conductance may reflect the structural maximum for the system. Therefore, the relationship between maximum oxygen uptake and lower leg muscle vascular conductance might well deviate from the observed linear relationship.
established among sedentary or mildly active and 'endurance trained' individuals (Reading et al., 1993; Snell, 1987).

It is well known that many practitioners of traditional strength training models, such as long, triple, and high jumpers, develop increases in muscle bulk and strength (Hakkinen et al., 1981; MacDougall et al., 1980; Young et al., 1983). Runners too, typically model their resistance training regimens on such programs. There is evidence that the adaptive processes involved in bodybuilding not only lead to increases in muscle bulk and strength, but may also promote significant increases in aerobic function through adaptive changes within the muscle tissue (Shantz, 1982; Tesch and Larsson, 1982; Tesch et al, 1989, Bell and Jacobs, 1990). Additionally, the volume of high force muscular contractions performed during bodybuilding elicits high blood flows (Walloe and Wesch, 1987), which may result in a high G max.

There is a paucity of data describing peripheral blood flow and training, especially concerning the relationship of muscle blood flow to maximum oxygen uptake in specific training groups. Therefore, this study will provide:

1) a more complete understanding of the contribution of maximal vascular conductance to maximum oxygen uptake;

2) an examination of the effects of the different models of training on the cardiovascular responses to exercise;

3) evidence suggesting the optimal method of resistance training for endurance athletes.
1.3 Specific Objectives

The objectives of the present study were: (1) to compare healthy, untrained individuals with sex matched, elite runners, body builders and power athletes with regard to maximum oxygen uptake (VO\textsubscript{2}max), skeletal muscle blood flow, and vascular conductance at rest, immediately following sub-maximal exercise, and immediately following exercise to exhaustion, and (2) to examine the relationship between maximal aerobic power and vascular conductance in these groups.

1.4 Hypotheses

1) Body builders will demonstrate higher maximal vascular conductance and lower blood pressure than power athletes, elite runners, and healthy sedentary subjects in response to maximal calf ergometry.

2) Elite runners will demonstrate a higher maximal oxygen consumption, and body builders will demonstrate a lower maximal oxygen consumption than would be predicted by maximal vascular conductance in accordance to the relationship established by healthy sedentary and endurance trained individuals.
Chapter 2
RELATED LITERATURE

2.1 Maximal Aerobic Power

2.1.1 Background

One of the classic debates in the field of exercise physiology has been the question of which component(s) of the oxygen transport system limit(s) maximal oxygen uptake (VO$_2$ max). The variables involved in the oxygen transport and utilization system can be grossly separated into central and peripheral components: the central components are generally considered to be those components associated with the heart, lungs, and large vessels, while the peripheral components are those which are involved with control of blood flow, diffusion, and metabolism in the active muscle tissue. The major focus of research in this area has been on oxygen uptake during an acute bout of maximal exercise (Saltin and Strange, 1992).

The cardiovascular correlate of Ohm's Law states that the flow (cardiac output) is equal to the pressure (mean arterial) divided by the resistance (total peripheral). Since the circulatory system is a closed loop system, the so-called central and peripheral components are intricately linked, i.e. they are interdependent. It is due to this simple fact that an answer to the question of which factor(s) limit(s) VO$_2$ max is so elusive. The more important question for the general population, and the crucial question for the competitive distance runner concerns what limits the degree of improvement in VO$_2$ max? While the answer to this question may not be as simple as the central vs. peripheral argument, it does represent a way of
systematically investigating the adaptive changes with training. Is an improvement in VO\textsubscript{2} max limited by the capacity of the heart to generate the cardiac output required to provide sufficient blood flow to the exercising tissues? Or conversely, does a limitation in the peripheral vasculature to accept freely the blood flow into the exercising tissues result in a ceiling for VO\textsubscript{2} max improvement? A series of most interesting observations, and a potential answer to this question, can be found in the little understood cardiovascular adaptations that occur with body building training.

2.1.2 Limits to Increases in Maximal Aerobic Power

Healthy individuals of any age can expect an increase of 5 to 15% in VO\textsubscript{2}max with training (Daniels et al., 1978b). Smith and O'Donnell (1984) demonstrated an increase in VO\textsubscript{2}max of 14% following 36 weeks of running training. Interestingly, most of the improvement occurred within the first 12 weeks, and all of the change had occurred by 24 weeks. Most exercise physiology textbooks describe a maximum of 5 to 20% improvement in VO\textsubscript{2}max following endurance training irrespective of training frequency or the length of the training period. (Brooks and Fahey, 1985; Guyton, 1991; Noakes, 1991). However, since the typical VO\textsubscript{2}max value for a young untrained male is 40 to 45 ml (kg min\textsuperscript{-1}) (Macnab et al., 1969; Staron et al., 1983; Simoneau et al., 1985), a 5 to 15% improvement would not be adequate to achieve the high values measured in elite athletes (table 2.2).

Since most training studies are conducted over weeks or months, whereas elite athletes will have often been engaged in training for many years or even decades, this analysis may be somewhat simplistic. In addition, it is very difficult for training studies to duplicate the extreme training intensity to which a high
performance athlete would be committed. Indeed, there is some evidence to suggest that with high-intensity and a long-term training commitment (>2 years), improvements of up to 50% can be achieved (Saltin et al., 1977). Although changes in VO$_2$\text{max} among established elite runners are usually less on a percentage-basis than in previously sedentary subjects, Martin and Coe (1991) anecdotally reported observing an increase in VO$_2$\text{max} from 4695 to 5525 ml (min)$^{-1}$ (+18%), in an elite middle distance runner over a seven month period.

Table 2.1 considers a hypothetical training effect based on various degrees of improvement in VO$_2$\text{max} in a healthy male, weighing 83.3 kg, with an initial VO$_2$\text{max} of 45 ml (kg min)$^{-1}$, and a body fat percentage of 19% (figures based on mean data from Staron et al., 1983):

<table>
<thead>
<tr>
<th>VO$_2$\text{max Improvement}</th>
<th>VO$_2$\text{max (ml (kg min)$^{-1}$)}</th>
<th>VO$_2$\text{max (ml (kg min)$^{-1}$)*}</th>
</tr>
</thead>
<tbody>
<tr>
<td>10%</td>
<td>49.5</td>
<td>54.5</td>
</tr>
<tr>
<td>20%</td>
<td>54.0</td>
<td>59.4</td>
</tr>
<tr>
<td>40%</td>
<td>63.0</td>
<td>69.3</td>
</tr>
</tbody>
</table>

* with a body fat correction of -10%

Clearly, an improvement of 20% in VO$_2$\text{max} would not be sufficient for the average male to develop an aerobic power comparable to that found in elite athletes; however, an improvement in the order of 40% is more likely necessary.

It would seem that there is a limit to the extent that VO$_2$\text{max} can be improved with training, and that the values seen in elite athletes may be partly because of
genetics, and partly due to intensive training over a period of years. A genetic endowment for a high VO₂max may manifest itself as a response to training. In other words, some people may respond to training to a greater degree than most others.

In conclusion, although the evidence is mixed, there is a distinct possibility that with appropriate training over an extended period, some people may be able to increase their VO₂max from that indicative of a normal, healthy, sedentary individual (45-50 ml (kg min)⁻¹) to the 70 ml (kg min)⁻¹ range representative of elite runners.

2.1.3 Maximal Aerobic Power and Testing Modality

Maximal aerobic power values are usually higher when larger muscle masses are used, (McArdle and Magel, 1970; Pechar et al., 1974; Pannier et al., 1980; Shephard et al., 1988). Subsequently, VO₂max values reported using cycle ergometry tend to be 7-11% lower than those reported using a treadmill (McArdle and Magel, 1970; Pechar et al., 1974; Shephard 1977; Pannier et al., 1980; Shephard et al., 1988). Typically, in studies such as these, the subjects tended to be from a non-highly trained population. Withers et al. (1981) have shown that elite runners score higher (p<0.05) on a treadmill (4,600 ml min⁻¹) than on a cycle ergometer (4,190 ml min⁻¹) VO₂max test. In contrast, elite cyclists scored higher (not statistically significant) on a cycle ergometer (4,520 ml min⁻¹) than on the treadmill (4,340 ml min⁻¹) (Withers et al., 1981). However, Hagberg et al., found competitive cyclists had VO₂max values 4% higher using cycle ergometry than with treadmill running (1978). A recent review of the VO₂max testing of runners found
graded motorized treadmill running to be the most reliable and reproducible method for the determination of VO$_2$max in runners (McConnell, 1988).

The difference between VO$_2$max scores on a cycle ergometer compared to treadmill running in untrained subjects is probably due to a muscular limitation in the quadriceps muscles. This muscle limitation to VO$_2$max is revealed through higher levels of perceived exertion, and higher arterial blood lactates two minutes after any given submaximal workload, when compared to either treadmill running or step testing, (Shephard, 1977). Indeed, a positive correlation has been found between quadriceps strength and performance on a cycle ergometer VO$_2$max test (MacNab and Conger, 1966).

Coyle et al. (1988) were able to demonstrate the sensitivity of test modality to specific adaptations in a study of a group of cyclists with similar VO$_2$max scores. Despite similar mitochondrial enzyme activity and oxidative ability, those cyclists who had lower lactate thresholds during cycle ergometry had similar lactate thresholds during treadmill running up a 10% grade when compared to cyclists with high lactate thresholds during cycle ergometry. This evidence indicates that even among trained cyclists, those with more years of training had a far superior cellular capability for aerobic performance without eliciting large amounts of blood lactate, but only when cycling.

Two conclusions can be drawn about the specificity of VO$_2$max testing:

1) Even though cycle ergometry involves two leg performance, it seems to involve a smaller muscle mass than treadmill running (Shephard et al., 1988), and is limited mainly by the quadriceps muscles, except among elite cyclists (see (2) below).

2) When testing elite athlete populations, it is critical to test athletes in a modality which involves motor recruitment patterns similar to those used in training
to obtain a "true" maximal value and to ensure that the limitation to the test is similar for all subjects.

2.1.4 Maximal Aerobic Power and Running Performance

The correlation between VO$_2$max and running performance ranges from 0.08 to 0.91 (McConnell, 1988). While the relationship may be stronger across a broad range of aerobic fitness, within a group of elite runners, the range of VO$_2$max values is quite small, and therefore, VO$_2$max is not a good predictor of performance. Within the group of 26 elite runners listed in table 2.2, there is no correlation between performance and VO$_2$max (Noakes, 1991). In a study by Costill et al. (1976b), some of the greatest runners at the time were investigated to find out what physiological mechanism(s) was responsible for their outstanding endurance performances. They concluded that there was no correlation between VO$_2$max and performance; however, despite these findings, there is strong evidence that VO$_2$max is very important to running performance, and in fact, the relationship may not be a causal one, but specific to the performance duration. This evidence is outlined in the remainder of this section.

Performance prediction

Running economy (RE), or what is often called efficiency, is the amount of oxygen required for an individual to maintain a specific submaximal running velocity (Daniels, 1974). It has been suggested that RE is a very good predictor of running performance. It has been shown in several studies that the more accomplished the runner, the better the running economy (Noakes, 1988). Although great middle and long distance running performance may require an individual to
have a VO$_2$max of 70 ml kg$^{-1}$ min$^{-1}$ or higher, among those with VO$_2$max values of such magnitude, performance is best predicted by running economy.

**Table 2.2 Maximum Oxygen Consumption (VO$_2$max) Values in Elite Endurance Athletes (from Noakes 1991)**

<table>
<thead>
<tr>
<th>Athlete</th>
<th>VO$_2$max value (ml min$^{-1}$ kg$^{-1}$)</th>
<th>Major Performance</th>
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</thead>
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<tr>
<td>Dave Bedford</td>
<td>85.0</td>
<td>10 km WR 1973</td>
</tr>
<tr>
<td>Steve Prefontaine</td>
<td>84.4</td>
<td>1 mile 3:54.6</td>
</tr>
<tr>
<td>Gary Tuttle</td>
<td>82.7</td>
<td>2:17 marathon</td>
</tr>
<tr>
<td>Kip Keino</td>
<td>82.0</td>
<td>2 km WR 1965</td>
</tr>
<tr>
<td>Don Lash</td>
<td>81.5</td>
<td>2 mile WR 1937</td>
</tr>
<tr>
<td>Craig Virgin</td>
<td>81.1</td>
<td>2:10:26 marathon</td>
</tr>
<tr>
<td>Jim Ryun</td>
<td>81.0</td>
<td>1 mile WR 1967</td>
</tr>
<tr>
<td>Steve Scott</td>
<td>80.1</td>
<td>1 mile 3:37.69</td>
</tr>
<tr>
<td>Bill Rodgers</td>
<td>78.5</td>
<td>2:09:27 marathon</td>
</tr>
<tr>
<td>Matthews Temane</td>
<td>78.0</td>
<td>21.1 km WR 1987</td>
</tr>
<tr>
<td>Don Kardong</td>
<td>77.4</td>
<td>2:11:15 marathon</td>
</tr>
<tr>
<td>Tom O'Reilly</td>
<td>77.0</td>
<td>927 km in a 6 day race</td>
</tr>
<tr>
<td>John Landy</td>
<td>76.6</td>
<td>1 mile WR 1954</td>
</tr>
<tr>
<td>Alberto Salazar</td>
<td>76.0</td>
<td>Marathon WR 1981</td>
</tr>
<tr>
<td>Johnny Halberstadt</td>
<td>74.4</td>
<td>2:11:44 marathon</td>
</tr>
<tr>
<td>Amby Burfoot</td>
<td>74.3</td>
<td>2:14:28 marathon</td>
</tr>
<tr>
<td>Cavin Woodard</td>
<td>74.2</td>
<td>48-160 km WR 1975</td>
</tr>
<tr>
<td>Kenny Moore</td>
<td>74.2</td>
<td>2:11:36 marathon</td>
</tr>
<tr>
<td>Bruce Fordyce</td>
<td>73.3</td>
<td>80 km WR 1983</td>
</tr>
<tr>
<td>Grete Waitz</td>
<td>73.0</td>
<td>marathon WR 1980</td>
</tr>
<tr>
<td>Buddy Edelen</td>
<td>73.0</td>
<td>marathon WR 1963</td>
</tr>
<tr>
<td>Peter Snell</td>
<td>72.3</td>
<td>1 mile WR 1964</td>
</tr>
<tr>
<td>Zithulele Sinqe</td>
<td>72.0</td>
<td>2:08:05 marathon</td>
</tr>
<tr>
<td>Frank Shorter</td>
<td>71.3</td>
<td>2:10:30 marathon</td>
</tr>
<tr>
<td>Willie Mtolo</td>
<td>70.3</td>
<td>2:08:15 marathon</td>
</tr>
<tr>
<td>Derek Clayton</td>
<td>69.7</td>
<td>marathon WR 1969</td>
</tr>
</tbody>
</table>
The onset of plasma lactate accumulation (OPLA) has been shown to be highly correlated to maximum running velocity over long distances (Noakes, 1991). OPLA is related to the highest steady state percentage of VO₂max that can be tolerated. When this critical intensity is surpassed blood lactate begins to accumulate in an exponential manner (Farrell et al., 1979). Coyle et al. (1988) elucidated the factors responsible for a high lactate threshold (LT). The two factors most strongly related to the LT were years of experience \( (r = 0.75; p < 0.01) \) and percent type I muscle fibers \( (r = 0.55; p < 0.05) \). Running economy can be combined with LT as a powerful predictor of endurance running performance through the measurement of running velocity \( (v) \) corresponding to OPLA (Farrell et al., 1979) or the onset of blood lactate (OBLA) (Sjödin and Jacobs, 1981). Sjödin et al. (1982) showed that the metabolic changes brought about by the introduction of a twenty minute treadmill run at a blood lactate of 4 mmol l⁻¹ into the training program of well trained male distance runners resulted in an increase in \( v_{OBLA} \). Although there was no statistical increase in VO₂max following the \( v_{OBLA} \) training, all but two of the subjects exhibited an increase \( (2.35 \text{ ml kg}^{-1} \text{ min}^{-1}) \). It is interesting to note that the two runners who did not demonstrate an increase in VO₂max were the only two 800 m runners in the group; the others specialized in longer distances (Sjödin et al., 1982). Subsequently, other investigators have also dissociated improvements in LT or \( v_{OBLA} \) and VO₂max (Denis et al., 1982; Yoshida et al., 1982; Smith et al., 1984; Gaesser et al., 1984; Hurley et al., 1984; Gaesser et al., 1986; Gaesser and Poole 1988).

The Role of Muscle Power

Scrimgeour et al. (1986) found that the maximal horizontal running speed during a level running treadmill VO₂max test was the best predictor of running performance \( (r = 0.72) \), even for ultramarathon running. While high lactate
thresholds are strongly associated with a high percentage of slow twitch fibres (Coyle et al., 1988), high VO$_2$max scores require high power outputs (Jones and McCartney, 1986), possibly making the two incompatible (La Fontaine et al., 1981; Noakes, 1991). If this were the case, then athletes competing in short duration, high power output events, where there is little concern with steady state aerobic metabolism, and therefore LT, may actually achieve higher VO$_2$max scores. In fact, Costill et al. (1976a) reported that middle distance runners have a lower percentage slow twitch fibres than distance runners, and that 800 m performance is inversely related to the percent slow twitch area (this may explain Sjödin’s (1982) finding of no increase in VO$_{BLA}$ in 800 m runners after VO$_{BLA}$ training). More directly, it has been shown that elite middle distance runners (1,500 to 10,000 meters) had VO$_2$max scores 5 ml kg$^{-1}$ min$^{-1}$ greater than elite marathoners (Pollock, 1977); the marathoners however were 2 ml kg$^{-1}$ min$^{-1}$ more efficient at 19.3 km/hr.

It has been reported that integrated electromyographic activity (IEMG) during incremental exercise is highly correlated ($r = 0.92$, $p<0.001$) to the blood anaerobic threshold (Nagata et al. 1981). It may well be possible that elite middle distance running requires the development of the type IIa fibers. It is quite logical that the type IIa fibers have the potential to perform at higher force outputs for sufficient duration to be conducive to successful middle distance running performance, and yielding higher VO$_2$max values. Specifically, type IIa fibres are capable of being both highly oxidative and highly glycolytic (Essen et al., 1975; Holloszy et al., 1982; Faulkner et al., 1986), and therefore, would have the capability of producing much higher power outputs than type I fibers for a much longer time than type IIb fibers (Faulkner et al., 1986). Jones and McCartney (1986) have demonstrated a strong linear relationship between VO$_2$peak and total
work done in a 30 second maximal power test. In addition, Martin and Coe (1991) have suggested that by using four repeated VO$_{2\text{max}}$ tests, one can generate a "mean sustainable VO$_{2\text{max}}$" and that the duration of the plateau is an important indicator of anaerobic work tolerance. Therefore, it seems quite convincing that within a group of elite runners, VO$_{2\text{max}}$ may in part reflect an interplay of muscle power and oxidative factors.

It has been shown that lipolysis is inhibited by elevated blood lactate levels (Boyd et al., 1974). Conversely, PFK may be inhibited by endurance training. When ATP, CP, and citrate levels are high, PFK is inhibited and FFA is utilized as an energy source, (Gollnick and Hermansen 1973). Therefore, it seems possible that training the type I fiber capability only would be advantageous for the marathoner due to the glycogen sparing effect of increased lipolysis. However, this could limit glycolytic activity, and therefore limit VO$_{2\text{max}}$ and middle distance performance. In fact, it may well be that VO$_{2\text{max}}$ is related to a specific power output. In other words, if the race distance is too short, then the factors limiting performance may have more to do with anaerobic power and capacity. On the other hand, if the race is too long, the race performance may be more dependant on running economy and LT.

Interestingly, in a study of elite middle distance runners (Lacour et al. 1989), only the 3,000 meter specialists showed a correlation between VO$_{2\text{max}}$ and race performance. Meanwhile Yoshida et al. (1990), from their assessment of the relative contributions of VO$_{2\text{max}}$, VOBLA, vLT, RE, and anaerobic peak and mean power in collegiate female distance runners (800 m, 1,500 m, 3,000 m), revealed that only anaerobic peak and mean power (Wingate anaerobic test) were significantly related to 800 m running performance. VO$_{2\text{max}}$, on the other hand, was a good
predictor of 3,000 m performance ($r = 0.67$, $p < 0.01$), but not significantly related to 800 m and 1,500 m performance.

Svendenhag and Sjödin (1984) measured the VO$_2$max of track athletes from the Swedish national team. They found that those athletes who participate in the 5,000 m and/or 10,000 m events had the highest values of aerobic power followed by the 1,500 m and 5,000 m runners, and then the marathoners. The marathoners were, however, higher than the 800 m and/or 1,500 m runners.

Three conclusions can be made from the foregoing:

1. VO$_2$max is an important measure related to running performance in that it provides an upper limit for aerobic metabolism from which fractional measures corresponding to racing durations can be effectively utilized. In this way, it is a valuable tool for assessing running potential.

2. Additionally, it seems possible that the contractile properties of the muscles may be responsible for setting the limit of oxygen utilization. For short middle distance running performances (1,500 m and shorter), the maximal contribution of anaerobic power and capacity from the type II muscle fibers is critical, whereas for distance running performance, the contribution of the type I fibers dominate. This may explain why elite middle distance running performance (especially 3,000 m to 10,000 m) is more more directly linked to VO$_2$max.

3. For the individual elite runner, the relative (corrected for body weight) VO$_2$max score is most important as it represents an excellent measure of fitness. Any change in relative VO$_2$max over time in an individual is likely to be reflected in a change in running performance potential. It would obviously be to a runner’s benefit to attempt to increase VO$_2$max over time, thereby upwardly adjusting any performance prediction equations, and more importantly, the performances themselves.
Therefore elite middle distance runners are most likely to represent a physiological model of optimized VO\textsubscript{2}max.

2.1.5 Interactions with Resistance Training

Resistance training is well known to lead to increases in strength and muscle power (Schmidtbleicher, 1992). Many endurance athletes, including elite runners, have utilized modest resistance training programs, both at times of injury and as a regular part of their training program, in the belief that strength gains would help prevent injury and/or improve performance. Since runners are very concerned about keeping their body mass as low as possible (rationale as demonstrated in table 2.1, and explained in 2.1.4), one of the most significant reasons not to engage in an ambitious resistance training programs has been the fear of muscle hypertrophy and the resulting weight gain which would ensue.

Concurrent strength and endurance training in previously untrained individuals seems to impede strength development, but not VO\textsubscript{2}max or short term endurance (Hickson, 1980; Dudley and Djamil, 1985; Hunter et al., 1987). In contrast, when endurance trained individuals added strength training programs to their overall training regimens, no impedance to strength development was observed (Hunter et al., 1987), and endurance performance was improved (Hickson et al., 1988).

Sale et al. (1990), used a single leg model in previously untrained males and females, to examine strength and endurance training interactions. Strength training, in addition to endurance training, resulted in the same improvement in 1RM as strength training alone, and resulted in a greater improvement in muscular endurance than endurance training alone or strength training alone. A slightly greater thigh
girth was also found among the strength + endurance training group. It is of interest to point out that the resistance training protocol was similar to a bodybuilding type (6 sets of 15-20 repetitions maximum with 2 minute rests between sets).

Nelson et al. (1990) found that concurrent strength and endurance training resulted in a similar degree of hypertrophy as seen with strength training alone. Additionally, the strength gains from concurrent training was found to be similar to those who only strength trained (Bell et al., 1990). Thus, both studies indicate that concurrent endurance training does not impede gains in strength.

While differing resistance training protocols make it difficult to draw conclusions, the evidence suggests that (i) the addition of strength training to the program of an endurance athlete would increase strength, muscular endurance, and muscle girth, and (ii) that the effect on VO2max remains unclear, although probably neutral.
2.2 Muscle Blood Flow

2.2.1 Blood Flow Regulation

Although muscle comprises approximately 40% of body mass, it receives approximately 20-21% (1,260 ml) of cardiac output at rest (Vander et al., 1985; McArdle et al., 1991). During maximal exercise however, active muscles can receive up to 88% (26,400 ml) of cardiac output. Within a muscle tissue, blood flow can increase by a factor of up to 30 times or more. This shift in the distribution of cardiac output is accomplished through a complex regulatory system involving neurogenic, humoral, and local control mechanisms (Rowell, 1986; Guyton, 1991, pp. 185-198).

According to Ohm's Law, blood flow is determined by the relationship of the pressure gradient and the resistance:

\[
Q = \frac{(P1 - P2)}{R}
\]

where \( Q \) = flow, \( (P1 - P2) \) = change in pressure, \( R \) = resistance

Specifically, Ohm's Law states that the blood flow is directly proportional to the pressure difference, and inversely proportional to the resistance.

Vascular conductance is a measure of blood flow through a vessel for a given pressure difference. In this way, it is the inverse of resistance:
Conductance = 1 / Resistance

As TPR cannot be directly measured, it must be calculated as the quotient MAP / Q.

Factors influencing vascular conductance

The three factors which affect the conductance of a given vessel or set of vessels include:
(i) the degree of laminar vs turbulent flow, (ii) the blood viscosity, and (iii) the diameter of the vessel(s).

Laminar and turbulent flow:

Flow in the arterioles is laminar (streamlined or linear). The molecules in the center of the vessel flow more quickly than do the molecules closer to the vessel walls. This is termed a 'parabolic velocity' flow profile. In contrast, under certain conditions, such as when flowing blood passes a rough surface, or makes a sharp turn, the flow becomes turbulent. In turbulent flow, some of the molecules will be flowing crosswise (inward and outward), thereby causing eddy currents. Within eddy currents, some molecules will actually travel opposite the direction of flow, and therefore, produce an increase in the overall friction and an increase in resistance to the flow. Although rough surfaces are not normally present inside the arteries, thrombotic plaques may be a contributing factor to the increased TPR seen in people with atherosclerosis.
Turbulence has been found to increase in direct proportion to the velocity of blood flow, and the diameter of the blood vessel (Guyton, 1991). In response to exercise, arteriolar diameter is increased to accommodate an increased flow, and the velocity in any given vessel is increased due to a greater driving pressure gradient. These factors contribute to an increase in resistance with rising blood flow.

Viscosity:

The viscosity of blood is constant under normal conditions. Changes in hematocrit have the most significant effect on blood viscosity. This is because layers of suspended red blood cells 'caress' each other as they move down the vessel, and thereby cause friction. Factors which affect hematocrit include pathological states, such as anemia and polycythemia, and transient states, such as dehydration. Under normal conditions, there does not seem to be any difference in blood viscosity between endurance trained individuals and healthy untrained individuals (Wood et al., 1991).

Vessel Calibre:

The diameter of the vessel is perhaps the most important factor affecting resistance as it is capable of rapid change, thereby altering the flow of blood to a tissue. It is in this way that the blood flow is matched to metabolic need.

The effect of even a small change in diameter greatly influences flow because vascular conductance increases with the fourth power of vessel radius. Two thirds of TPR is provided by the arterioles. Since these vessels are capable of a four-fold dilation, and based on the fourth power relationship of vessel radius and flow described by Poiseuille's law, arterioles could increase flow by a factor of 256. Obviously arterioles play the major role in controlling blood flow.

There are other important structures downstream from the arterioles which play an important role in regulating blood flow to tissues and individual cells. Metarterioles
and precapillary sphincters control the flow of blood into and within a capillary network in such a way that under normal conditions, blood does not flow continuously through a given capillary. This is termed 'vasomotion'.

**Overall control of blood flow**

The mechanical factors which effect blood flow have been discussed above, however, the factors which regulate the degree of vasoconstriction in the arterioles, and the pattern of vasoconstriction in metarterioles and precapillary sphincters are complex. It is the complex interaction of all mechanisms that allows for the precise matching of blood supply to cell needs. A system controlled centrally would be unable to provide for this precise matching. There are several recent reviews of blood flow control mechanisms which are quite comprehensive (Granger et al., 1988; Reading 1990; Shephard and Plyley, 1992). The key regulating factors are of neurogenic, humoral, and local origins.

**Neural Control**

It is well known that neural control of blood flow involves both sympathetic and parasympathetic branches of the autonomic nervous system. Arterial baroreceptors and chemoreceptors constantly monitor arterial blood pressure and blood volume (Guyton, 1991, pg. 198). In addition, ergoreceptors in skeletal muscle also send afferent nerve traffic to the brain stem, where they are processed, and adjustments to the levels of activity of the sympathetic and parasympathetic nerves is made (Shepherd, 1987).

Sympathetic outflow acts on the cardiovascular system through two basic types of adrenoreceptors. They are classified as the α and β-adrenergic. The α-adrenergic receptors are present on the sympathetic resistance and capacitance vessels, and their activation results in the contraction of the smooth muscle tissue surrounding these vessels. At rest, the smooth muscle in a vessel wall is constantly slightly contracted.
as it receives a steady discharge from the sympathetic fibers. This is called 'basal tone'. Vasodilatation can be achieved by reducing the sympathetic activity.

β-adrenergic receptors are classified as either β₁- and β₂. When β2 receptors are activated, there is a reduction of intra-cellular calcium, disengagement of intracellular proteins, and therefore vasodilatation via a relaxation of the smooth muscle tissue (Meisher, 1982).

**Hormonal Factors**

Hormones are released into the systemic circulation in response to a variety of stimuli including changes in blood pressure, the two most active agents during exercise being adrenalin and noradrenalin. They are released from the adrenal medulla as a result of sympathetic discharge. Noradrenalin acts as a vasoconstrictor by acting on the μ-receptors in the vessel wall. Adrenalin can act on either the μ-receptors, in which case the vessels vasoconstrict, or on the β₂-receptors causing vasodilatation. The vasculature of most tissues in the body are supplied with μ-receptors (cerebral tissue is a notable exception), while only the smooth muscle of vessels found in skeletal muscle, bronchi, and cardiac muscle, contain β₂-receptors (Shephard and Plyley, 1992).

**Local Control**

Dilatation of the resistance vessels occurs immediately with the contraction of muscle (Corcondilas et al., 1964), and is proportional to the strength of the contraction (Walloe and Wesche, 1988). The precise mechanism(s) that are responsible for the adjustments in dilatation are controversial (Granger et al., 1988), however they include: increases in hydrogen ion concentration, potassium, inorganic phosphate, and osmolarity, as well as various prostaglandin's, adenosine
nucleotides, and adenosine (Granger et al., 1988). The factors which initiate vasodilatation may be different than the factors that maintain it since arteries upstream from the dilating arterioles also relax, suggesting a possible neural component.

2.2.2 Blood Flow to Active Muscle

During endurance exercise, neural control mechanisms act to maintain mean blood pressure. As exercise becomes progressively more intense, a progressive elevation of mean blood pressure is achieved. This is accomplished by a decrease in flow to inactive skeletal muscle tissue, a decrease in blood flow to the viscera (Clausen, 1976). Flow to active muscles increases through the effects of local control mechanisms such as decreased oxygen partial pressure, heat, and leakage of potassium ions, which are responsible for the dilatation of the resistance vessels (arterioles) (Granger et al., 1988).

During exercise with a small muscle mass, it has been shown that there is a relatively larger increase in heart rate than with a large active muscle mass, and that at an equal work output, there is a more pronounced rise in sympathetic tone (Astrand et al., 1965). Similarly, it has been shown that arm work leads to a greater increase in total peripheral resistance than with leg work, possibly caused by vasoconstriction of the relatively larger arteriolar bed in the legs (Bevegard and Shepherd, 1966).

Clausen (1976) showed that maximal oxygen intake is inversely related to systemic vascular resistance. More recently, a relationship established between maximal oxygen uptake and maximal vascular conductance of the calf muscle (Snell
et al., 1987, Reading et al., 1992). Therefore the degree to which vasodilatation can be induced in the skeletal muscle vasculature is associated with maximal oxygen intake. It is unknown whether there is a functional or a structural difference in the skeletal muscle vasculature between endurance trained and sedentary subjects. Additionally, work by Shephard et al. (1988) has supplied strong evidence that as active muscle mass increases, the limitation of oxygen uptake moves from a peripheral to a central dependency. It seems that there is a threshold amount of active muscle mass that if not achieved, will lead to local muscle factors resulting in fatigue. If this threshold is achieved, then the limitation for oxygen consumption will be more dependent on the central delivery of oxygen.

2.2.3 Measurement of Skeletal Muscle Blood Flow

Blood flow is usually expressed in units of ml of flow per 100 ml of muscle volume. Normal resting values for blood flow tend to be in the range of 2-4 ml min$^{-1}$ 100 ml$^{-1}$ of muscle volume. Vascular conductance is calculated by normalizing the blood flow by the driving pressure. Normal resting vascular conductance values are very close to 0.04 ml min$^{-1}$ 100 ml$^{-1}$ mmHg$^{-1}$.

Measurement of blood flow has recently been extensively reviewed (Bernstein 1985; Reading, 1990; Hutchison, 1994). There are a variety of methods available which measure directly, or reflect, the rate of blood flow through muscle (Clausen, 1971; Anderson et al., 1983). Venous occlusion plethsmography has been shown to be a reliable non-invasive technique for measuring resting and post-exercise blood flows on small limbs, such as the calf (Snell et al., 1987; Weber et al., 1988; Hepple, 1992; Reading et al., 1993). The basic principle of the technique is that
when the thigh cuff is inflated, venous blood is prevented from flowing out of the calf while arterial blood continues to flow in, producing a volume change in the size of the calf in proportion to the arterial blood inflow. The use of a mercury or gallium-indium strain gauge sensor to detect the changes in the volume of a limb has further improved the application of venous occlusion plethsmography (Hughson, 1988). Vascular conductance can be calculated using this technique by dividing the blood flow by the simultaneously obtained mean arterial blood pressure (MAP).

One significant limitation of strain gauge venous occlusion plethsmography concerns the interpretation of values achieved immediately following submaximal exercise. Walloe and Wesch (1988) studied the magnitude and time course of adjustments in blood flow during and after quadriceps exercise, using a rapid pulsed doppler ultrasound technique. Their data revealed an inverse exponential decline in blood flow immediately following exercise, with a 30% reduction after 5 seconds, and 50% after 10 seconds. Therefore, the post exercise measurement is not truly reflective of the actual blood flow during exercise, but it still provides valuable information, providing that the time between the cessation of exercise and the flow measurement is consistent.

The one exception to this limitation is when maximal ischemic exercise is performed. Ischemic conditions are created by inflating the proximal (thigh) cuff to occlude blood flow to the calf (>220mm Hg). Maximal exercise is defined as an inability of the subject to continue to contract the calf (usually limited by discomfort or pain). Under such conditions, reactive hyperemia ensues, and maximal vasodilation is achieved (Snell et al., 1987). Conway (1966) has shown that an intra-arterial infusion of ATP yields the same blood flow as reactive hyperemia. Additionally, following maximal ischemic exercise, the blood flow values remains elevated for several minutes; in fact, it may even rise during the first 20-30 seconds
following the ischemic episode (Snell et al., 1987). Since vascular conductance is determined by the luminal size of the arterioles, precapillary sphincters, and metarterioles to a lesser extent, changes in vascular conductance in response to exercise reflect the change in the cross-sectional area of the arteriolar bed. Therefore, strain gauge plethsmography, using the maximal ischemic exercise technique, can provide an accurate reflection of the maximum capacity for vascular conductance (G max) under these controlled conditions.
2.3 Vascular Conductance and Whole Body Oxygen Consumption

2.3.1 Background

Andersen and Saltin (1985) have shown that VO$_2$ max is limited by the capacity of the heart to pump blood. They used a constant infusion thermodilution technique to measure quadriceps blood flow during several graded exercise loads, including peak flow during maximal exercise. Their findings of a mean blood flow of 248 ml min$^{-1}$ 100ml$^{-1}$ muscle mass would require a cardiac output of over 41 L min$^{-1}$, assuming an active muscle mass of 15 kg and shunting a flow of 4 L min$^{-1}$ to the non-exercising tissues (theoretical argument based on Rowell, 1988). A maximum blood flow of these proportions clearly indicates that the ability of the muscle to accept such a large blood flow when a large muscle mass is active as would be the case during running, is far in excess of the ability of the heart to pump such large quantities of blood. This conclusion is logically deduced as well, since it has long been known that maximal cardiac output is closely linked to VO$_2$ max (Jones, 1988). Although the prevailing view of the scientific community is that central processes are limiting, there is ample evidence to suggest that a more complex interplay of central and peripheral factors occurs than previously realized (Green and Patla, 1992).
2.3.2 The Possibility of a Blood Flow Related Peripheral Limitation to Improvement in VO₂ max

As would be expected, elements representing the central and peripheral contributions are evident in the VO₂ equation from the Fick equation (fig. 2.3.1), in which cardiac output (Q) defines the total transport capacity of the system, a central component, and the arterio- mixed venous oxygen difference, (a-VO₂ diff.), i.e. the extraction, represents the peripheral component. Assuming normal respiratory function, O₂ transport is dependent upon myocardial function, blood volume, hematocrit and O₂ binding capacity. The extraction is determined by the balance between the intracellular oxygen utilization (metabolic rate) and the oxygen availability, which is a function of local muscle blood flow, capillary blood volume, and diffusion. Consequently, mean transit time of blood through the capillaries, is negatively correlated with VO₂ max (Kayar et al., 1994).

While blood flow and mean transit time are closely related (both are, in part, dependent upon the design characteristics of the microcirculatory bed), mean transit time is determined primarily by the density of capillary supply (Saltin, 1985), and blood flow in the microcirculation is dependent on the balance between the pressure head and the ability of the arterioles to dilate (Ohm's law at the local level). The size of the arteriolar bed is reflected numerically by the conductance, i.e. the amount of blood flow when normalized for the driving pressure.

Figure 2.3.1

\[ \text{VO}_2\max = \text{Cardiac Output (MBP ÷ TPR)} \times (\text{a-VO}_2\text{ content}) \]
If the ability of the heart to deliver blood to the exercising tissues, i.e. Q, actually limits VO$_2$ max, and not the ability of the peripheral vasculature to accept the blood freely (as demonstrated by Andersen and Saltin (1985)), then we would not expect any significant changes in the peripheral vasculature after endurance training. However, significant changes in the peripheral circulation do occur as evidenced by an increase in the number of capillaries in the muscle tissues which has been well documented as a commonly occurring adaptation to endurance training (Hudlicka et al., 1992). Much less is known about the changes which occur with regard to arteriolar structure and function. Clausen (1976) published a review paper in which he compiled 125 values from the various human studies in which VO$_2$ max, mean blood pressure (MBP) and Q had been determined at maximum exercise. From the data, he plotted the VO$_2$ max data against calculated values for total peripheral resistance (where TPR = MBP ÷ Q); the relationship was found to be hyperbolic in nature (fig. 2.3.2), and the regression equation determined was $y = 11.8 / x^{0.72}$ ($r = 0.87$, $p < 0.001$). Clausen also plotted the mean values for a number of subject groups from the various studies, including studies involving exercise or bed rest interventions which are known to alter VO$_2$ max. The derived regression line described above fit these data quite well (fig. 2.3.2).
Figure 2.3.1
Total Peripheral resistance at VO₂max
Maximal oxygen consumption (VO₂max) in relation to total peripheral resistance (TPR) at VO₂max (MBPmax / Qmax) based on VO₂max, Qmax and MBPmax values from the literature. The left-sided panel displays 125 individual values. Subjects included top trained young athletes, well trained middle aged athletes, young and middle aged healthy men and women, and patients with CAD and patients with essential hypertension. Subjects with the highest VO₂max values had the lowest TPR values, while subjects with the lowest VO₂max values had the highest TPR values.
The right-sided panel gives group mean values from intervention studies where changes in VO₂max were induced by training or bed rest, by beta receptor blockade, or different types of exercise. Any intervention that changed VO₂max, changed it in accordance with the regression line $y = 11.8 / x^{0.72}$ ($r = 0.87$, $p < 0.001$) derived from the 125 individual values shown on the left-sided panel. (Clausen, 1976).
The relationship between VO$_2$ max and TPR is significant for several reasons. It clearly shows that a decrease in TPR is accompanied by an increase in VO$_2$ max. Furthermore, in the well-trained athletes (upper left corner of the graph), a small decrease in TPR corresponds to a large change in VO$_2$ max. Since vasoconstriction in the non-exercising tissues during maximal exercise is not significantly modified by training (Clausen, 1976; 1977), a decrease in TPR at maximum exercise can only be achieved through alterations in arteriolar function in the exercising muscles. Any decrease in resistance, therefore, must be due to a functional increase in the size of the arteriolar bed, attained either by increasing the number of arterioles and/or by developing a greater degree of dilation in existing arterioles.

2.3.3 The Link Between Muscle Blood Flow and Maximal Aerobic Power

More recently, a strong relationship has been established between VO$_2$ max and lower leg maximal vascular conductance (G max) in well trained and untrained men (Snell, 1987), and in male endurance trained and untrained men, and congestive heart failure patients (Reading et al., 1993). These studies have shown that across a wide range of fitness, higher levels of VO$_2$ max are associated with higher levels of maximal vascular conductance in the lower leg, suggestive that an increase in G max as a result of endurance training produces a concomitant increase in VO$_2$ max.

The endurance trained subjects in the study by Reading et al. (1993) were defined as those participating in a regular physical activity program - not elite by any standard. The VO$_2$ max of these subjects was 48 ml kg$^{-1}$ min$^{-1}$ as measured on a cycle ergometer, and the mean G max was 0.65 ml min$^{-1}$ 100 ml$^{-1}$ tissue mmHg$^{-1}$ pressure. This mean G max was only slightly less than that found by Snell (1987).
5.2 Subject Characteristics

As can be seen in table 4.1.2, the athlete groups studied were distinct in how they trained for their sport. The power group, consisting of track and field jumpers and decathletes, were chosen because of the high priority they place on fitness (personal correspondence with the University of Toronto Jumps coach: Carl Georgevski). These athletes likely present a better model for a cardiovascular study than those athletes usually used to represent a power trained muscle, such as throwers and Olympic and power lifters. Such athletes are not concerned about propelling their own body weight, and therefore are less concerned about maintaining a low fat body mass and overall fitness. Furthermore, in the power group utilized in this study, the volume of supplementary aerobic activity and running, albeit mostly short distance, contrasts well with the body builders who avoided such activities. Therefore, any aerobic adaptations seen in the body builders which are greater than those seen in the power group could be attributed directly to the mode of resistance training employed.

The modes of resistance training employed are markedly different between the two resistance trained groups. Although the volume of training was not quantified, table 4.1.2 clearly shows that a much larger number of repetitions are performed by body builders than by power athletes. The total time spent resistance training was similar in the two groups. Additional leg work was done by the power athletes through plyometric training.
2.3.4 Significance to the Elite Endurance Athlete

If a structural / functional limit to G max exists, then it would be expected that those athletes for whom the highest possible VO$_2$ max is crucial for success will lie closest to that limit (Rowell, 1988). The VO$_2$ max - G max relationship for the range involving the sedentary and "trained" individuals has been established, it is unclear as to the nature of the relationship in the elite, endurance athlete. Clearly, Rowell (1988) would predict that the relationship of VO$_2$ max and G max in the elite endurance athlete should have a much greater slope than that observed with the sedentary and "trained" individuals described by Snell (1987) and Reading et al. (1993). A greater slope for the VO$_2$ max - G max relationship in the elite endurance athlete would be suggestive of a leveling off of the structural / functional vascular adaptation, and likely, a perfusion limitation.
2.4 Training Methods and Related Adaptations in Elite Athletes

2.4.1 Elite Runners

Training

In general, any runner whose primary competitive event is 800 m or longer has traditionally been viewed as an endurance athlete, yet the length of competitive distances in running races exceeds the marathon, a race of 42.2 km. In fact, the metabolic demands and muscular limitations presented by these activities are quite different, and so is the training program in which these athletes participate. As was clarified in the review of VO₂max and running performance, the middle distance events (1,500m - 10,000m) seem to be more directly dependent on VO₂max. This is intuitive, based on the type of training employed, since middle distance runners spend a significant portion of their overall training volume at high velocities using various forms of interval training. This intensity element is a higher priority than absolute volume of training, which is the hallmark of the marathon competitor. Consequently, a greater portion of training volume is spent by middle distance runners at intensities at or even above VO₂max, as measured by heart rate (Janssen, 1987; Martin and Coe, 1991; Noakes, 1991).

The training year of a competitive middle distance runner is highly periodized. Approximately 60% of the year is in the preparatory phase of training, and is characterized by a relatively higher volume and lower intensity training. Although training programs vary, many runners continue to engage in interval training, utilizing sessions which are more volume-based with work to rest ratios rarely exceeding 1.5:1, usually in the range of 2:1 or 3:1. Such sessions may be performed twice per week. A third 'hard' day is included either in the form of a
long run, a 'tempo' run, or a race. The relative intensity of 'recovery' runs (often called 'easy' runs) on alternate days is highest during this period.

As the competitive season approaches, there is a gradual priority shift from volume to intensity. In general, 'tempo' runs become shorter in length, and races are introduced until they occur with a frequency of at least once every week (not always at distance of specialization). At this point, the training programs of the various distance specialists begins to diverge, but the difference in training between the specialists is primarily one of volume vs. intensity. The 800, 1,500, and even 3,000 m runners devote a great deal of their hard training sessions to developing anaerobic power and capacity interspersed on alternate days with recovery runs. A typical set may take the runners through a velocity range of below race velocity, usually at or near VO$_2$max (a 'warm up set': e.g. 4 X 800 m with 1:15 min. rest), to repeated runs near maximal velocity for the given distance (e.g. 8 X 200 m with rests ascending from 1 min. to 5 min.). Since such training probably recruits FT fibers repeatedly, it is likely that this training stimulus is responsible for the inverse relationship between 800 m performance and ST% (Saltin et al., 1976a).

At the longer end of the competitive spectrum, 10,000 m runners taper their volume of training more slowly, and only deal with high intensity (anaerobic) training through races or through a high intensity repetition at the end of a training session (e.g. 6 X 800 m with 1:00 min. rest + 2 X 800 m with 3:00 min. rest).

Adaptations

Adaptations to running training have been discussed in section 2.1. It seems reasonable that fiber type specific adaptations occur depending on training stimulus combined with genetic differences and may account for the differences seen in VO$_2$max among elite athletes. If the factors that lead to VO$_2$max are viewed as a series of interdependent resistances, then it would be logical that physical training
designed to improve $\text{VO}_2\text{max}$ would lead to a 'fine tuning' of the entire system, limited by the most unchangeable resistance.

Structures which are adaptable would not be maintained with a structural capacity greater than the functional demands placed on it. Therefore, they would likely appear to limit $\text{VO}_2\text{max}$. Unadaptable structures on the other hand, would exist with excess capacity in order to be able to accommodate any potential adaptation in aerobic capacity (Lindstedt et al., 1988).

### 2.4.2 Bodybuilders

**Training: the bodybuilding method**

The primary objectives of body building are to increase muscle mass, to produce symmetry, and to produce definition so that individual muscles can be visually separated from each other (Tesch, 1992). Within the body building community, there are a variety of training strategies, which have evolved over the years based on the experiences of successful competitors. This is, in part, because science has not yet been able to identify clearly a specific resistance training protocol which is optimal for muscle hypertrophy (Tesch et al., 1989). In general, bodybuilders tend to see their training as "breaking down" the muscle. During recovery between training sessions, they believe that the muscle protein is being "rebuilt", and that due to supercompensation, a larger muscle will be developed. As a result, much of the current emphasis in body building has revolved around supplementing recovery nutritionally with the aid of extensive vitamin and amino acid suplementation.

It is well documented that forceful contractions, especially eccentric contractions, result in delayed muscle soreness; analysis of biopsies provides evidence to support
that actual muscle damage to contractile proteins occurs (Abraham, 1977; Tiidus and Ianuzzo, 1983; Friden et al., 1983). Based on the "break down and build up theory", it seems logical to many bodybuilders that the greater the "break down", provided that there is adequate recovery, the greater the muscle growth. Subsequently, bodybuilders typically employ training protocols which utilize submaximal contractions that lead to concentric contraction failure between 6 and 12 repetitions. Each set lasts approximately 30 seconds, followed by a 60 second rest, i.e. a work to rest ratio of 1:2. This effort is repeated 3 or more times for any given exercise. Additional exercises activating the same muscle group are then performed, so that in 30 minutes, approximately 20 sets are performed on one muscle group (Tesch and Larsson, 1982; Dudley, 1988).

Adaptations

Bodybuilders do not show greater increases in systolic or diastolic blood pressure than untrained controls when performing resistance exercise with high relative or absolute weights (Fleck and Dean, 1987). In fact, resting blood pressure in bodybuilders has been reported to be the same or lower than in untrained controls, and showed a much lower rate pressure product (Colliander and Tesch, 1988; Urhausen et al., 1989).

Very little is known about the circulatory adaptations which result from chronic body building training. This is in large part, due to the problem in categorizing resistance training. It is now recognized that the morphological changes which occur as a result of resistance training vary greatly depending on the load used, the number of repetitions per set, the number of sets, and the length of the rest period between repetitions and sets. As these training variables are manipulated to produce different training regimens, several distinctive levels of adaptive stress can be applied to the muscle. Most training studies have employed a "standard" resistance training
protocol both for simplicity and/or consistency with the existing literature on resistance training (Vogel, 1988).

However, the type of resistance training performed by bodybuilders is thought to result in skeletal muscle structural adaptations which are similar to those seen following endurance training (Tesch and Larsson, 1982). During graded cycle ergometry, bodybuilders have been shown to exhibit a lower heart rate and systolic blood pressure for any given workload when compared to students (Tesch, 1992). This is a response similar to that found in endurance trained subjects, and indicates an enhanced capacity for aerobic work.

From the limited data available on the adaptive response of skeletal muscle to body building training regimens, one can only speculate on the circulatory adaptations at the level of the muscle. Again, it must be emphasized that to bring about alterations in the flow capacity of the muscle, i.e. to bring about adaptations in arteriolar structure / function, the training involved in body building must adequately stress the cardiovascular system.

It has been shown that bodybuilders have an increase in the number of capillaries per muscle fiber (Shantz, 1982; Tesch and Larsson, 1982; Tesch et al., 1984; Bell and Jacobs, 1990), and an increase in the oxidative capability of the fast twitch fibers (Tesch et al, 1989). The same study showed that bodybuilders had a greater citrate synthase activity within the slow twitch muscle fibers. These studies are supported by Ingjer's endurance training study in which it was shown that the capillary supply to a given fiber is more closely related to its mitochondrial content than to its fiber type (Ingjer, 1979). This study shows that a high percentage of slow twitch fibres, as found in elite endurance athletes, would not be necessary for a muscle to be highly capillarized. These changes by themselves do not necessarily indicate an increased capacity for blood flow. It has also been reported that
recovery from fatiguing exercise, similar to that employed by bodybuilders, is strongly related to the muscle's respiratory capacity and to the capillary blood supply (Tesch and Wright, 1983; Ivy et al., 1982). In addition, significant findings were reported by Walloe and Wesch (1988), who demonstrated that blood flow through a muscle is directly related to the tension of the contraction, and that it was increased when the relaxation phase was shorter than the contraction phase, and that near maximal flow rates were achieved within a few contractions. These findings implicate the high repetition, multiple set, low rest training regimen employed by bodybuilders as being extremely stressful on the peripheral vasculature. However, it is unknown whether bodybuilders exhibit high peripheral blood flows and high G max values.

2.4.3 Power Athletes

Training: the classical strength method

The primary objective of power training is for the neuromuscular system to be able to produce the greatest possible force within a given time period. The relationship between maximal force and movement speed is expressed as a force-time curve. In a general sense, as the external load decreases, a movement can be performed more quickly, and the relative contribution of maximal strength diminishes. In this scenario, the rate of force generation is the predominant factor (Schmidtbleicher, in Komi (ed.), 1992).

In track and field competition, high, long, and triple jumpers, as well as decathletes, are primarily concerned about moving their own body weight, a mass which is far less than their 1 repetition maximum (1RM) load. Since most forms of
resistance training result in some degree of hypertrophy, the jumper, in contrast to the thrower, cannot afford to increase muscle mass without positively adjusting the whole body force time curve.

In contrast to bodybuilders, power athletes use resistance training as a means of improving their relative power (work / time / body mass). The resistance training model utilized by jumpers is modeled on the program followed by olympic lifters. This program involves few repetitions with fairly high loads, with full recovery between sets. The emphasis is on exercises for the legs, such as squats and lunges, but also include the snatch, clean and jerk, and some basic exercises for the upper body. This training is heavily supplemented by plyometrics, medicine ball work, and performing the jump activity itself. Jumping rarely exceeds three days per week, and a maximum of twenty jumps are performed within one hour. Therefore, an average of one maximal contraction is performed every three minutes.

In contrast to the jumpers described above, it should be noted that since the lifters are training for an event that is more directly dependent on their 1RM, they use resistance training as the primary mode of training (Garhammer and Takano, 1992). The one significant difference may be that there is a greater degree of hypertrophy. So called "pure" power training of the legs, such as jumping, results in smaller increases in maximum strength and muscle hypertrophy, but has a greater effect on the rate of force development than seen with classical strength training (Häkkinen and Komi, 1986). Maximal strength and power are not separate entities however, as maximal strength provides the basis for power development. It is for this reason that jumpers utilize both "pure" power training as well as maximal strength training.

Adaptations

There is ample evidence to support a detrimental effect of power lifting training on systemic cardiovascular parameters. Heavy resistance training seems to subject
the athletes to more extreme blood pressures, with values greater than 300 mmHg being reported (MacDougall et al., 1985; Fleck and Dean, 1987). The adaptive response to such high vascular pressures include increases in left ventricular wall thickness and cardiac mass (Fleck, 1988), and may occur at the expense of heart volume (Longhurst et al., 1980; Brown et al., 1983; Morganroth, 1975).

In addition, this form of training typically results in muscle hypertrophy (Hakkinen et al., 1981; MacDougall et al., 1980; Young et al., 1983), primarily of the fast twitch fiber types (Hakkinen et al., 1981; Young et al., 1983). Fast twitch fibers are associated with lower leg blood flows (Frisk-Holmberg M. et al., 1981). Since there is no capillary neoformation during this form of training, the capillary density decreases, resulting in a greater diffusion distance (Tesch et al., 1984). There is also a concomitant reduction in the skeletal muscle mitochondrial fractions (MacDougall et al., 1980; Luthi et al., 1986). These adaptations indicate a decrease in aerobic power. Longitudinal studies utilizing a similar type of training as that employed by power lifters have reported either no change (Hickson 1980; Hickson et al., 1980; Staron et al., 1984), or a decrease (Gettman and Pollock, 1981), in VO\textsubscript{2} max. Thus, the cardiovascular and cellular adaptations which have been reported for power lifting indicate a reduced aerobic capacity. If this is the case, then one would expect that this type of training might result in a decreased G max in relation to VO\textsubscript{2} max.
2.4.4 A Comparison of Bodybuilding to Power Training

In a typical bodybuilding training sessions the oxygen consumption will rise to no more than 50% of maximum (Seals and Hagberg, 1984). One can speculate that resistance training, utilizing fewer sets and repetitions, as well as a longer rest period, would result in a relative oxygen consumption which is lower than that seen with bodybuilding. Previous reports indicating a lack of improvement in VO$_2$max in response to resistance training have suggested that this is due to the lack of an adequate central circulatory stress, as reflected by the low relative rate of oxygen consumption (Dudley, 1988). However, during graded cycle ergometry, bodybuilders demonstrated the same or slightly lower blood lactates at each workload when compared to untrained controls. Weight lifters and power lifters, on the other hand, had higher blood lactates for any given workload (Tesch, 1987). These findings may, in part, be explained by evidence of selective hypertrophy of fast twitch fibers in response to traditional resistance training programs (Prince et al., 1976; Hakkinen et al., 1981; Tesch and Larsson, 1982; Young et al., 1983). In contrast, bodybuilding seems to result in the hypertrophy of both fiber types (Tesch et al., 1989), probably due to the exhaustive 'endurance' nature of body building. Such training is likely to recruit all fiber types.

In one case, Tesch and Larsson (1982) examined the muscle fibers of three elite body builders and found no hypertrophy of either fast twitch or slow twitch fibers. Additionally, the bodybuilders were found to have a very low percentage of FTb fibers (45% for the vastus lateralis and 3% for the deltoid). As the authors speculated that their peculiar finding were indicative of hyperplasia, it is unfortunate that they did not measure capillary density and diffusion distance.
Hakkinen and colleagues (1984) have studied the neuromuscular, anaerobic, and aerobic performance characteristics of bodybuilders and compared them to those of power lifters. It was shown that there was no difference in fiber type distribution and isometric force production per unit body weight, but there were significant differences in time to 30%, 60%, and 90% force production. They also found a significant difference in VO₂ max values with 42 and 51 ml min⁻¹ kg⁻¹ for the power lifters and bodybuilders, respectively. These data indicate that despite no differences in fiber type distribution and isometric strength, the bodybuilders had an elevated aerobic capacity and greater power. Tesch et al. (1989) found a greater citrate synthase activity in the slow twitch fibers of bodybuilders than in power lifters, and a greater myokinase activity in the fast twitch fibers. It is interesting to note that the citrate synthase activity was lower in the bodybuilders than it was in the untrained subjects. Furthermore, it appears that the bodybuilders, unlike the power lifters, have developed an increased VO₂ max compared to that of sedentary individuals (Hakkinen et al., 1984; Shephard et al., 1984).

There is a paucity of data on the circulatory adaptations to resistance training in general, and body building in particular. A recent review of cardiovascular adaptations to resistance training (Fleck, 1988) concluded with a call for more research to identify the nature of these adaptations with consideration of the caliber and type of athlete (i.e. bodybuilder, power athlete). There does not seem to be any published data on muscle blood flow in resistance trained subjects.
2.4.5 Conclusions

The inferential evidence from the review presented above suggests that there may be significant increases in peak skeletal muscle blood flow and conductance as a result of the type of training a body builder would engage in. Power athletes would contrast well with body builders since they represent a model of traditional resistance training methods. Furthermore, track and field jumpers and decathletes would be an ideal power athlete group for a study utilizing calf ergometry to assess skeletal muscle blood flow and treadmill VO2max testing, since their training focuses on improving power (work / time) relative to body mass, specifically in muscles involved in running (e.g. plantar flexors).

Elite runners provide a calf muscle model of the most highly endurance trained. From the relationship established between Gmax and VO2max, it could be postulated that this group would have the highest Gmax values. If a peripheral perfusion limitation to VO2max exists, then the relationship between Gmax and VO2max in elite runners should display a steeper slope than in untrained and less well trained groups.
Chapter 3

Methods

The VO2 max and G max of 37 male subjects consisting of 7 body builders, 10 elite runners, 11 power athletes, and 9 sedentary subjects was examined. All testing was done at the University of Toronto Lifestyle Laboratory. All subjects were free from medication. None of the athletes had been exercising for twenty four hours prior to the lab tests.

3.1 Subject Selection

Age was not included in the selection criteria as the priority was to obtain the highest caliber athletes possible. It was expected that any variation in age will not be so great as to be a confounding factor.

All subjects signed the informed consent approved by the Office of Research Services at the University of Toronto (appendix 1), and were asked to complete a standard physical activity readiness questionnaire (PAR-Q) prior to the testing. All athletes were asked to complete a detailed performance and training questionnaire (appendix 2).
3.1.1 Healthy Sedentary

All healthy sedentary (HS) subjects were recruited from the general university community. All were free of medication, and had not been engaged in prior regular physical activity or training programs for at least two years. Regular physical activity was defined as any exercise program which elevated heart rate above 60% of maximum, and/or involved any form of resistance training, and required three sessions a week.

3.1.2 Elite Runners

Elite middle distance runners (ER) were recruited from local running clubs. Inclusion criteria were that they must have been training for competition for at least 2 years, and racing competitively at distances between 800 m and 10,000 m at a national level.

3.1.3 Power Athletes

Power athletes (PA) were recruited from the University of Toronto Track and Field Club. Inclusion criteria were that they must have been training for competition for at least 2 years, and competing at national and/or international competitions. PA subjects were considered as jumpers (long, triple, high), or decathletes.
3.1.4 Bodybuilders

Body Builders (BB) were recruited from local bodybuilding clubs. Inclusion criteria were that they must have been training for over two years in preparation for competition. No attempt was made to screen subjects for steroid use.

3.2 Maximal Aerobic Power Testing

All laboratory testing was performed on one day within a two hour period. Maximal aerobic testing followed blood flow testing on a calf ergometer.

VO\textsubscript{2} max was obtained through open circuit spirometry during an incremental exercise test to exhaustion on a motor-driven treadmill. Following establishment of a comfortable treadmill speed, the slope was increased by 2% every 2 minutes for 8 minutes followed by 1% increases in slope every 1 min. until the subject became exhausted. Test termination was determined by subject exhaustion, and the RER (> 1.15), achievement of an age predicted maximum heart rate, and a plateau in VO\textsubscript{2} (an increase of less than 1 ml kg\textsuperscript{-1} min\textsuperscript{-1} with an increase in work load of 1%) were used as criteria to verify that a maximal value was attained (McConnell, 1988).

Expired gases were collected every 15 seconds in a mixing chamber. The fraction of expired oxygen (F\textsubscript{E}O\textsubscript{2}) was determined using an oxygen analyser with a zirconium dioxide electro-chemical cell (Amtek) which is heated to precisely 700 °C. The fraction of carbon dioxide (F\textsubscript{E}CO\textsubscript{2}) was determined by a fast response carbon dioxide analyser (Jaeger) using non-dispersive infrared absorption. A heated
sampling line insures that the correct amount of gas sample, with its original water vapour content, is drawn from the mixing chamber.

Ventilation measurements were performed by inspired air flowing through a turbine flow-meter cartridge (PK Morgan). In this device, air spins a helical impeller supported on cushion-mounted sapphire jewel bearings. Four infrared light beams cross the flowmeter bore in the path of the impeller. These light beams are sequentially interrupted as the impeller rotates and produce four trains of digital pulses. Flow rate and total volume can be determined by processing these pulses. The turbine blade has a very small mass (0.005 grams), and therefore, any changes in air direction or velocity were detected.

Calibration:

The gas analysers were turned on at least 30 minutes prior to each calibration to allow the cell to heat and equilibrate. Analysers were calibrated with known oxygen and carbon dioxide concentrations immediately before and following each test. During the calibration procedure, the time delay between the volume signal and the exhaled gas transport to the analyser is determined and introduced to the program. Volume calibration was accomplished using a 3-liter calibrated syringe prior to and after each test.

All cardiorespiratory data were collected and analyzed by a semi-automated metabolic cart (PK Morgan) with on-line computer.
3.3 Strain Gauge Venous Occlusion Plethysmography

3.3.1 Blood Flow Determination

Blood flow to the calf was measured using venous occlusion strain gauge plethysmography (SGP) as described previously by this laboratory (Reading et al., 1992, 1993). Subjects were placed in a supine position with the right foot (or jumping foot) on the calf ergometer pedal, with the calf elevated approximately 25 centimeters. Shoulder restraints were adjusted to position the lower leg to slightly less than horizontal in order to facilitate venous return. Subjects remained in this position for 15 to 20 minutes to allow for acclimatization and SGP setup. A blood pressure cuff ("exclusion cuff") was placed around the ankle and inflated to a pressure of 220 mmHg to prevent blood from circulating in and out of the foot. A second cuff (occlusion), placed around the thigh just above the knee, was rapidly inflated to 60 mmHg, and the change in volume of the leg was measured over the first seven seconds of data collection via an indium-gallium strain gauge (Medisonics Model SPG16) placed around the greatest circumference of the right calf (or jumping leg), and was applied with a tension of approximately 10 gm in order to ensure skin contact around the entire calf. The strain gauge was electrically calibrated with a square wave deflection of 1% prior to each data acquisition session. The indium-gallium strain gauge technique has been previously shown to be reliable (Reading, 1990).

Systemic blood pressure and heart rate was estimated from recorded beat to beat signals during the strain gauge measurement using a Finapress 2300 automated blood pressure monitor (Ohmeda). The Finapress utilizes the Penaz method of non-invasively measuring the complete arterial waveform at the fingertip. It is based on
the concept that if an externally applied pressure is equal to the arterial pressure, there will be a zero transmural pressure and the arteries will not change in size. Therefore the blood volume in those arteries will not change, and this constant blood volume will become the set point for a servo loop. As the blood volume varies from the set point, the servo loop is driven to increase or decrease the pressure of the finger cuff. The cuff pressure is measured using an electronic pressure transducer, and the resulting signal is displayed as the arterial pressure.

The Finepress uses two methods for setting and maintaining the set point. The first is called the 'servo start-up adjustment'. It consists of stepping the cuff pressure through various pressures and measuring the magnitude and shape of the waveform at each level. If the pressure is below diastolic pressure, the resulting waveform is small because the arterial wall is constantly under tension. If the cuff pressure is above systolic pressure, the arterial wall will be collapsed, and there will be virtually no pulsatile component. Somewhere between these two pressures will be a point where the resulting pulsation will be relatively large because the arterial wall experiences both compression and tension. The computer measures and interprets these waveforms, and selects the initial set point. Once the set point is established, the second method, called the servo-self adjustment, is used. It involves the same decision by the computer (to slightly adjust the pressure based on the waveform), but on a single beat basis (every 10 to 70 beats, depending on the magnitude of the last adjustment).

All data collected as part of the calf blood flow protocol was obtained through an automated on-line collection and processing system using a WATSMART data acquisition unit and custom written software.
3.3.2 Blood Flow Data Analysis

Blood flow (BF) data analysis was facilitated by a custom written analysis program with samples taken at 100 Hz. A blood flow curve was displayed on the monitor which visually represented the increasing and decreasing calf volume (inflow and outflow phase during thigh cuff inflation and deflation). The software calculated the slope from two points selected at the region of the curve where there appeared to be the fastest increase in calf volume. Any artifact from the thigh cuff when it was turned on could be easily seen and avoided during the selection of the points for slope calculation. Systolic and diastolic blood pressures, mean arterial blood pressure (MAP), and heart rate were calculated simultaneously. Conductance calculations were made by the software as the blood flow normalized for simultaneous MAP, where $G = BF / MAP$. Systolic and diastolic blood pressures, MAP and HR were also calculated using the above method for the entire 45 second period.

3.3.3 Calf Exercise Protocol

Calf blood flow was measured prior to the maximum oxygen consumption testing. The blood flow measurement technique was performed three times: while the subject was at rest, after submaximal exercise, and after maximal ischemic exercise, using a specially designed calf ergometer which provides resistance to plantar flexion (Reading et al, 1992). The exercise protocol used consisted of continuous, rhythmical, plantar flexion exercise on the calf ergometer. Blood flow was assessed immediately upon cessation of the exercise.
Submaximal exercise was performed immediately following the resting data acquisition. Work through the movement of a 5 kilogram load moved 15 cm by continuous, rhythmical, plantar flexion at a frequency of once per second for a duration of 2 min.

The maximal exercise protocol was designed to elicit a maximal ischemic response. It was performed approximately 5 to 10 min. following submaximal exercise data acquisition. Maximal blood flow following ischemic exercise was accomplished through the inflation of a blood pressure cuff around the thigh (>220 mmHg) to prevent flow to the calf. With the thigh cuff still inflated, the subject rested for 2 min., followed by vigorous, rhythmical, plantar flexion exercise at an initial cadence of 60 rpm, with a 30 kg load, and continuing to volitional fatigue.

3.3 Statistical Analysis:

All data were analyzed using a one-way ANOVA to compare responses among the four groups. A post-hoc test (Duncan's multiple range) was used to identify differences between specific subgroups following the ANOVA. Within each group, linear regression analysis (least squares method) and simple correlation coefficients were used to examine the relationship between the following variables: VO$_2$ max and G max, VO$_2$max and BFmax, Gmax and Grest, and Gmax and years of training. Statistical significance was determined as $p<0.05$. 
Chapter 4

RESULTS

4.1 Subject Characteristics

4.1.1 Basic Characteristics

Subject characteristics are listed in table 4.1.1. Although no attempt was made to age-match the four groups, all groups were similar in age. Significant differences in body mass were found between BB and ER compared to HS or PA. As would be expected ER had a lower body mass than all other groups, while PA were taller than all other groups. The events which track and field athletes considered their primary events are also listed.

4.1.2 Athlete Groups Training Characteristics

Table 4.2 is a training summary of the athlete groups. All athletes utilized a periodized training program. Each athlete was asked to report typical training patterns over the past year. None of the athletes indicated a lapse in training all year.

All athletes except for five runners participated in resistance training on a regular basis. The six runners who did participate in resistance training reported that resistance training was never undertaken during competitive phases of their training.
In addition, the runners did not tend to participate in additional aerobic activities. PA on the other hand tended to participate in a variety of different aerobic activities.

<table>
<thead>
<tr>
<th>Table 4.1.1 Subject Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Values are means ± SEM</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>HS</th>
<th>ER</th>
<th>PA</th>
<th>BB</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>8</td>
<td>11</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>Age (years)</td>
<td>25.1±1.3</td>
<td>24.8±1.4</td>
<td>23.0±1.1</td>
<td>26.9±1.5</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>78.5±3.1ᵇ</td>
<td>64.9±0.99⁰</td>
<td>79.6±2.8</td>
<td>89.6±3.2ᵃᵇᶜ</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>173.6±3.7ᶜ</td>
<td>178.8±1.2ᶜ</td>
<td>186.8±2.1</td>
<td>173.8±2.3ᶜ</td>
</tr>
<tr>
<td>Competitive Event</td>
<td>N/A</td>
<td>800m (1)</td>
<td>Decathlon (3)</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td>1.5-5km (4)</td>
<td>Triple Jump (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5-10km (6)</td>
<td>Long Jump (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>High Jump (4)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\[ a=p<0.05 \text{ (vs. HS)}, \quad b=p<0.05 \text{ (vs. ER)}, \quad c=p<0.05 \text{ (vs. PA)} \]

While PA participated in an extensive running program, none of it was endurance oriented. The decathletes trained the same way as the jumpers, but tended to spend more total time training when calculated on a weekly basis.
### Table 4.1.2 Training Pattern Summary

Values are means ± SEM

<table>
<thead>
<tr>
<th></th>
<th>ER</th>
<th>PA</th>
<th>BB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Years of Training</td>
<td>9.1±1.6</td>
<td>7.9±1.0</td>
<td>4.4±1.6</td>
</tr>
<tr>
<td>Running</td>
<td>11/11</td>
<td>11/11</td>
<td>1/7</td>
</tr>
<tr>
<td></td>
<td>Mean Volume = 85 km/wk</td>
<td>High Intensity Intermittent (40m - 300m) /Continuous (warm-up only)</td>
<td>10-15 min</td>
</tr>
<tr>
<td></td>
<td>Intermittent and Continuous</td>
<td>Continuous for warm-up</td>
<td>2 d/wk</td>
</tr>
<tr>
<td></td>
<td>5-7 d/wk</td>
<td>4-5 d/wk</td>
<td></td>
</tr>
<tr>
<td>Other Aerobic Activities</td>
<td>Cycling - 3/11</td>
<td>Pool Running - 2/11</td>
<td>Cycling - 7/7</td>
</tr>
<tr>
<td></td>
<td>Pool Running - 1/11</td>
<td>Cycling - 1/11</td>
<td>Stairmaster - 1/7</td>
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<td></td>
<td>Swimming - 1/11</td>
<td>Basketball - 3/11</td>
<td>15-60min</td>
</tr>
<tr>
<td></td>
<td>Basketball - 1/11</td>
<td>Wrestling - 1/11</td>
<td><strong>always easy</strong></td>
</tr>
<tr>
<td></td>
<td>Hockey - 1/11</td>
<td>Bobsledding - 1/11</td>
<td></td>
</tr>
<tr>
<td>Resistance Training</td>
<td>6/11</td>
<td>11/11</td>
<td>7/7</td>
</tr>
<tr>
<td></td>
<td>10-15 reps</td>
<td>1-10 reps</td>
<td>6-100 reps</td>
</tr>
<tr>
<td></td>
<td>1-3 sets</td>
<td>2-6 sets</td>
<td>3-5 sets /exercise</td>
</tr>
<tr>
<td></td>
<td>2-3 d/wk</td>
<td>1.5-4hrs /session</td>
<td>2-4 exercises /muscle</td>
</tr>
<tr>
<td></td>
<td>0.5 -2 hr /session</td>
<td>2-6 d/wk</td>
<td>1-3hr /session</td>
</tr>
<tr>
<td></td>
<td>(+ extensive plyometrics)</td>
<td></td>
<td>4-7 d/wk</td>
</tr>
</tbody>
</table>

Numbers indicate the number of subjects who participated in the named activity.
4.2 Blood Pressure and Heart Rate Responses to Calf Ergometry

Tables containing heart rate and blood pressure data at rest, post submaximal exercise, and post maximal ischemic calf ergometry can be found in the appendices. Post submaximal measures were made 2 to 32 seconds following calf ergometry with a mass of 5kg and a repetition rate of 60 per minute. Post maximal measures were made 5 to 35 seconds following calf ergometry with a 10kg mass and a self determined repetition rate, terminated by volitional fatigue.

4.2.1 Resting Measures

At rest, there was a tendency for all groups to show a higher systolic and a lower diastolic blood pressure than the 120mmHg and 80mmHg normally expected for this age group.

HS tended to have the highest mean arterial blood pressure values and BB the lowest. Body builders tended to have a lower mean arterial pressure although this difference was found not to be significant (figure 4.2.2). Significant differences were observed for heart rate between ER and all other groups (p < 0.01), which is consistent with their high volume of chronic endurance training (figure 4.2.1).
4.2.2 Post Submaximal Calf Ergometry Measures

ER had a significantly lower heart rate than all other groups (p < 0.01). HS tended to have the highest mean arterial pressure. In contrast, BB exhibited the lowest MAP.

4.2.3 Post Maximal Ischemic Calf Ergometry Measures

While ER demonstrated a lower heart rate than all other groups, the differences were not statistically significant. BB had a lower MAP following the maximal ischemic calf ergometry protocol (p < 0.05) when compared to each of PA, ER and HS. The MAP among PA, ER, and HS was very similar (figure 4.2.2).

4.3 Maximal Aerobic Power

According to the criteria, all subjects demonstrated a maximum effort. Table 4.3.1 depicts the data obtained from the VO$_2$max testing. A significant difference (p < 0.001) was found between HS and ER in absolute VO$_2$max (i.e. ml min$^{-1}$). When VO$_2$max, was expressed per kilogram body mass, ER was found to be significantly different from all other groups (p < 0.0001). None of the other groups were found to be significantly different from each other.
Maximal heart rates at VO$_2$max were not recorded for BB due to technical difficulties with the heart rate monitor. ER and PA showed lower heart rates at VO$_2$max than HS ($p<0.05$), but were not different from each other.
Figure 4.2.1
30 Second Average Heart Rate Following Calf Ergometry

a=p<0.05 (vs. HS), b=p<0.05 (vs. ER), c=p<0.05 (vs. PA)
Figure 4.2.2
30 Second Average Mean Arterial Pressure Following Calf Ergometry

a=p<0.05 (vs. HS), b=p<0.05 (vs. ER), c=p<0.05 (vs. PA)
## Table 4.3.1 Maximal Oxygen Consumption Test Results

Values are means ± SEM

<table>
<thead>
<tr>
<th></th>
<th>HS</th>
<th>ER</th>
<th>PA</th>
<th>BB</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>8</td>
<td>11</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>VO₂ (ml (min⁻¹))</td>
<td>3531±173</td>
<td>4606±98a</td>
<td>4061±226</td>
<td>3971.±233</td>
</tr>
<tr>
<td>VO₂ (ml (kg min⁻¹))</td>
<td>45.15±1.86</td>
<td>71.04±1.16a</td>
<td>50.74±1.57b</td>
<td>44.73±3.24b</td>
</tr>
<tr>
<td>HR (b min)</td>
<td>192±2</td>
<td>183±2a</td>
<td>185±1a</td>
<td>N/A</td>
</tr>
<tr>
<td>RER (VCO₂ / VO₂)</td>
<td>1.263±0.020</td>
<td>1.151±0.025a</td>
<td>1.220±0.018a</td>
<td>1.126±0.010a</td>
</tr>
</tbody>
</table>

a=p<0.05 (vs. HS), b=p<0.05 (vs. ER), c=p<0.05 (vs. PA)
4.4 Blood Flow and Vascular Conductance

Tables containing blood flow and conductance measures at rest, and following both submaximal and maximal ischemic calf ergometry can be found in the appendices.

4.4.1 Resting Blood Flow and Vascular Conductance

Figures 4.4.1 and 4.4.3 depict resting blood flow and vascular conductance observations at rest. BB had a significantly higher blood flow (4.7±0.41 ml (min 100 ml)^{-1}) than HS (3.6±0.24 ml (min 100 ml)^{-1}, p<0.05), ER (3.2±0.17 ml (min 100 ml)^{-1}, p<0.001) and PA (3.5±0.28 ml (min 100 ml)^{-1}, p<0.05). Resting vascular conductance in BB (0.059±0.003 ml (min 100 ml mmHg)^{-1}) was found to be significantly higher than HS (0.036±0.003 ml (min 100 ml mmHg)^{-1}, p<0.001), ER (0.030±0.002 ml (min 100 ml mmHg)^{-1}, p<0.001), and PA (0.036±0.003 ml (min 100 ml mmHg)^{-1}, p<0.01). HS, ER and PA were not significantly different from each other in either blood flow or vascular conductance.

4.4.2 Blood Flow and Vascular Conductance in Response to Submaximal Calf Ergometry

Submaximal blood flow and vascular conductance were not significantly different between groups.
4.4.3 Blood Flow and Vascular Conductance in Response to Maximal Ischemic Calf Ergometry

Figures 4.4.2 and 4.4.4 depict blood flow and vascular conductance observations following maximal ischemic calf ergometry. HS exhibited the lowest values for maximal blood flow (54.5 ± 3.00 ml (min 100 ml)^{-1}) and Gmax (0.456 ± 0.022 ml (min 100 ml mmHg)^{-1}). ER had a significantly higher maximal blood flow (87.1 ± 5.28 ml (min 100 ml)^{-1}, p < 0.001) and Gmax (0.721 ± 0.038 ml (min 100 ml mmHg)^{-1}, p < 0.0001) than HS, and PA (68.5 ± 3.90 ml (min 100 ml)^{-1} and 0.570 ± 0.034 ml (min 100 ml mmHg)^{-1}) respectively, p < 0.05). PA had a significantly higher Gmax than HS (p < 0.05). BB had a significantly higher maximal blood flow (89.1 ± 6.49 ml (min 100 ml)^{-1}, p < 0.01) and Gmax (0.807 ± 0.054 ml (min 100 ml mmHg)^{-1}, p < 0.001) than HS, and PA (p < 0.05) for both blood flow and Gmax (p < 0.01). ER and BB were not significantly different from each other although the Gmax for BB tended to be higher.

4.4.4 Resting Vascular Conductance and Maximal Vascular Conductance

Linear regression analysis revealed a strong relationship in BB between resting vascular conductance and Gmax (r = 0.94, p < 0.002) (Fig. 4.4.6), as well as between resting BFrest and BFmax (Fig. 4.4.7). There was no relationship between resting vascular conductance and Gmax, or BFrest and BFmax in either the HS, PA, or ER (Fig. 4.4.5, Fig. 4.4.6, and Fig. 4.4.7).
Figure 4.4.1
Blood Flow at Rest
a=p<0.05 (vs. HS), b=p<0.05 (vs. ER), c=p<0.05 (vs. PA)

Figure 4.4.2
Maximal Blood Flow
a=p<0.05 (vs. HS), b=p<0.05 (vs. ER), c=p<0.05 (vs. PA)
Figure 4.4.3
Vascular Conductance at Rest
a = p < 0.05 (vs. HS), b = p < 0.05 (vs. ER), c = p < 0.05 (vs. PA)

Figure 4.4.4
Maximal Vascular Conductance
a = p < 0.05 (vs. HS), b = p < 0.05 (vs. ER), c = p < 0.05 (vs. PA)
Figure 4.4.5
Gmax vs. Grest in Elite Runners and Power Athletes
Figure 4.4.6
Gmax vs. Grest in Body Builders and Healthy Sedentary Subjects
Figure 4.4.7
BFmax vs. BFrest in Body Builders and Healthy Sedentary Subjects

\[ r = 0.93, p < 0.003 \]
Figure 4.4.8
Years of Training vs. Gmax in Body Builders

$r = 0.80, r < 0.04$
Figure 4.4.9

Years of Training vs. Gmax in Elite Runners
4.5 Vascular Conductance vs. Aerobic Power

Figure 4.5.1 depicts the relationship between maximal blood flow and $VO_2^{\text{max}}$. Inter-group analysis revealed a non-significant relationship. The relationship between $G_{\text{max}}$ and $VO_2^{\text{max}}$ was slightly stronger, but was also not significant (figure 4.5.2). However, a strong positive linear relationship between $VO_2^{\text{max}}$ and $G_{\text{max}}$ was found ($r=0.84, p<0.0001$) when the non-resistance trained groups were analysed separately.

One BB subject had a $VO_2^{\text{max}}$ value of 58.3 ml (min kg)$^{-1}$ which is over 30% higher than the mean for BB. A strong negative relationship was found between $VO_2^{\text{max}}$ and $G_{\text{max}}$ ($r=-0.88, p<0.03$) within the BB group when this one outlier was removed ($n=6$).
Figure 4.5.1
Maximal Blood Flow and Maximal Oxygen Consumption
Figure 4.5.2
Gmax and Maximal Oxygen Consumption

$r = 0.30, p < 0.42$
Chapter V
DISCUSSION

5.1 Major Findings

The major findings of this project are:

1. Blood pressure at rest, and in response to submaximal and maximal calf ergometry, was less pronounced in body builders than in power athletes, elite runners, and healthy sedentary subjects.

2. Maximal aerobic power in elite runners was significantly higher than in the healthy sedentary or resistance trained groups. None of the other three groups were different from each other.

3. Resting vascular conductance was greater in body builders than in any of the other groups, none of which were different from each other.

4. Maximal vascular conductance was greater in body builders and elite runners than in power athletes or healthy sedentary subjects. Power athletes had a greater maximal vascular conductance than healthy sedentary subjects.

5. According to the established relationship between VO₂max and Gmax, elite runners had a higher VO₂max than would have been predicted by Gmax. Conversely, body builders had a lower VO₂max than would have predicted by Gmax.
5.2 Subject Characteristics

As can be seen in table 4.1.2, the athlete groups studied were distinct in how they trained for their sport. The power group, consisting of track and field jumpers and decathletes, were chosen because of the high priority they place on fitness (personal correspondence with the University of Toronto Jumps coach: Carl Georgievski). These athletes likely present a better model for a cardiovascular study than those athletes usually used to represent a power trained muscle, such as throwers and Olympic and power lifters. Such athletes are not concerned about propelling their own body weight, and therefore are less concerned about maintaining a low fat body mass and overall fitness. Furthermore, in the power group utilized in this study, the volume of supplementary aerobic activity and running, albeit mostly short distance, contrasts well with the body builders who avoided such activities. Therefore, any aerobic adaptations seen in the body builders which are greater than those seen in the power group could be attributed directly to the mode of resistance training employed.

The modes of resistance training employed are markedly different between the two resistance trained groups. Although the volume of training was not quantified, table 4.1.2 clearly shows that a much larger number of repetitions are performed by body builders than by power athletes. The total time spent resistance training was similar in the two groups. Additional leg work was done by the power athletes through plyometric training.
5.3 Blood Pressure and Heart Rate Responses to Calf Ergometry

Most studies have shown that highly strength trained athletes exhibit average or lower than average systolic and diastolic blood pressure (Fleck, 1988; Fleck et al., 1989; Goldberg, 1989; Stone et al., 1990). Training studies have supported this observation as a training adaptation (Fleck, 1988; Goldberg, 1989; Stone et al., 1991). This study has shown a consistent tendency toward a lower blood pressure both at rest and in response to calf ergometry in the bodybuilders compared to any of the other groups. This tendency was strongest following maximal ischemic calf ergometry (figure 4.2.3). In fact, the increase in mean arterial pressure in the bodybuilders (from $82.7\pm 2.7$ mmHg to $103.3\pm 4.5$ mmHg) was only $20.6\pm 3.6$ mmHg. Power athletes, on the otherhand, exhibited an increase from $88.6\pm 2.6$ mmHg to $118.9\pm 2.8$ mmHg, an increase of $30.3\pm 2.7$ mmHg. The similar response of mean arterial pressure to the maximal calf exercise protocol among healthy sedentary, elite runners, and power athletes demonstrates a pressor response which is different from that of the body builders, and suggests that bodybuilding training results in alterations in the pressor response to such exercise. These findings support those of Fleck and Dean (1987) in which lower pressor responses were demonstrated by bodybuilders during exercises consisting of dumb-bell overhead press and one legged knee extension than was seen in untrained controls or in novice strength trained subjects.

A reduced mean arterial pressure, especially if achieved primarily through changes in diastolic blood pressure, would indicate a decreased risk of pressure induced cardiac hypertrophy. Indeed, the enlarged left ventricular mass seen in bodybuilders is due not only to an increase in diastolic posterior left ventricular wall
thickness (PWTd) and intraventricular septum wall thickness (IVSd), as in power athletes, but also because of an increased chamber size (Deligiannis et al., 1988).

Unfortunately, in the present study the work done during the maximal exercise bout was not quantified, and therefore, the categorical nature of the workloads does not allow for a statistical analysis between groups. Indeed Tesch (1992) showed that during graded cycle ergometry, bodybuilders have a lower heart rate and systolic blood pressure for any given workload when compared to students (Tesch, 1992). Because the submaximal workloads were standardized, their finding may be explained by a lower relative workload in the bodybuilder group. Stronger muscle fibers would result in a lower portion of those fibers (therefore, a lower number of motor units) firing in order to produce a given amount of force. This would likely trigger a lower pressor response than in subjects with weaker fibers.

There are two reasons that it is unlikely that a lower relative workload is the cause of the observed lower pressor response in the body builders in this study. Firstly, it can be anecdotally reported that during the maximal ischemic protocol, more work was done by the body builders than any other group, since more resistive weight was required to fatigue those subjects within a reasonable time. Secondly, each subject continued until volitional fatigue, therefore providing a maximal effort immediately prior to the cessation of ergometry. The results are also supported by others who found a decreased RPP following strength training in previously untrained college aged males (Stone et al., 1991), indicating a possible hypotensive effect of resistance training.
5.4 Maximal Aerobic Power

The VO2max found in the healthy sedentary group (45.2±1.86 ml (min kg)-1) was within the range expected as found by others (48±8 ml (min kg)-1)(Shephard, 1992).

The VO2max results for the elite runners and power athletes were also as expected. As was extensively outlined in Section 2.1, it was expected that elite middle distance runners would have a much higher VO2max than all other groups. Therefore, the result of 71 ml (min kg)-1 compares well to the range outlined in the literature for such athletes (70-75 ml (min kg)-1) (Neumann, 1988). The power athletes' value of 50.7±1.57 ml (min kg)-1 compares well to the range found in the literature for power track and field athletes (48-52 ml (min kg)-1 for sprinters, 50-55 ml (min kg)-1 for long jumpers, and 60-65 ml (min kg)-1 for multi-event athletes) (Neumann, 1988).

The VO2max found in the body builders in this study is slightly lower than would have been expected from the literature (44.7±3.7 ml (min kg)-1 in this study compared to 51 ml (min kg)-1 found Hakkinen and colleagues (1984). A possible explanation for this difference is that the body builders in this study were all in the preparatory phase (build-up) period of training and were concerned only with building muscle mass and not a low body fat. They therefore tended to have a relatively (to competitive season) high fat weight.
5.5 Vascular Conductance

5.5.1 Sources of Error

There are five potential sources of error with the data acquisition system used to quantitate resting and post exercise blood flow:

1. fitting of the strain gauge around the calf
2. assumption that the calf is cylindrical
3. temperature sensitivity of the strain gauge
4. varying limb compartment size
5. finapress monitor
6. unblinded experimenter

1. The strain gauge must be applied with a slight (approximately 5-15 gram) stretch so as to ensure contact with the appendage. This stretch was applied and full contact was always achieved. Furthermore, the gauge tubing length should be matched as closely as possible to the calf girth. Since calf girths vary among individuals, a systematic error may exist. Calf girths from this study are supplied in appendix 8.

2. In a cylinder, the cross-section is uniformly circular. Therefore, any change in the circumference is directly proportional to the change in cylinder volume. Therefore, the theory of strain gauge plethsmography is dependent on the assumption that the calf is a cylinder (Sumner, 1985). Any change in strain gauge resistance (circumference of the calf) is directly proportional to the change in volume, provided that the circumference of the limb is the same as the length of the strain
gauge. The cross section of the calf may not be truly circular, therefore, a direct relationship must involve a systematic error depending on the deviation in shape from that of a cylinder.

3. The gallium indium in rubber strain gauge has a slight temperature sensitivity of 0.056% per °C (Holanson et al., 1975). This means that an increase in temperature of 10 °C would result in an error 0.56%. The temperature in the laboratory was carefully monitored and remained at 19-21 °C. Increases in core temperature were likely minimal due to the small muscle nature of the exercise, the short duration of the exercise, and the rest interval between the exercise bouts. Any skin temperature effects on the strain gauge were minimized due to the low temperature sensitivity of the gallium indium strain gauge used.

4. The calf compartment size may change with the unequal expansion of different parts of the calf. The skin overlying the muscular compartment of the calf will obviously expand more than the skin overlying the tibia. It has been found that unequal expansion of the limb does not affect the accuracy of the results (Sumner, 1985).

5. A potential inaccuracy in the blood pressure measurement using the Finapress monitor. The fingertip is an extremity and is used in resistance training. Subjects were acclimated, and room temperature was consistent, therefore temperature was an unlikely source of error. The thickness of the skin may also contribute to error in blood pressure measurement. The resistance trained trained groups in particular may have had thicker skin due to the heavy work done with their hands. Finger tip skin thickness was not measured. The hand was held in a relaxed manner at the level of the heart in order to ensure no variance in elevation or intra-muscular pressure.
6. In the present study, the experimenter was unblinded when blood flow analysis was performed. Therefore, the possibility exists that data acquisition and analysis was biased despite the best efforts of the experimenter. Strict attention to the measurement criteria was used when determining the slope selection.

5.5.2 Resting Vascular Conductance

One surprising and significant finding of this study was that of a significantly (p < 0.001) higher resting vascular conductance in the body builders compared to all other groups.

5.5.3 Response to Submaximal Calf Ergometry

The submaximal exercise load did not reveal any significant differences between groups. There are two possible reasons for these findings:

1. Precise control of the workload was not possible, as some individuals exhibited difficulty in maintaining the steady rhythmic contraction frequency set by the metronome. Furthermore, despite our criteria of a 15cm deflection of the load, some subjects exceeded this criteria by a greater or lesser degree. Combined with the expected intersubject variability, and a small but finite, variation from data analysis, all groups exhibited a high standard deviation in blood flow and conductance.

2. Post exercise blood flow data is likely dependent on more than the supply of oxygen; other local vaso-regulatory factors, such as the clearance of metabolic
waste, are also of importance. It may be expected that any training induced cellular improvements in muscle function would result in a decreased submaximal blood flow. However, such adaptations may be achieved concomittent with an improvement in the vasodilatory response to exercise. Such changes would not be revealed with the methods employed in this study. An examination of the processes involved in recovery from such submaximal exercise bouts would have to be necessary to distinguish between these possibilities.

5.5.4 Response to Maximal Ischemic Calf Ergometry

Maximal vascular conductance was significantly higher in the elite runners compared to the power athletes and healthy sedentary subjects (elite runners vs healthy sedentary; p<0.0001, elite runners vs power athletes, p<0.05). These findings were expected based on previous studies (Snell et al., 1987; Reading et al., 1993); however, the high value of Gmax (0.807±0.054 ml/min/100ml/mmHg) found in the body builders was also significantly higher than that of the healthy sedentary and power athletes (body builders vs healthy sedentary, p<0.001, body builders vs power athletes, p<0.05). These findings clearly demonstrate that a high peripheral vascular blood flow can be achieved not only through endurance training (Snell et al., 1987; Reading et al., 1993), but also through chronic resistance training.

The lower peripheral vascular conductance in the power athletes (0.570±0.034 ml/min/100ml/mmHg) suggests that the increase in Gmax for the body builders occurred as a result of the specific form of resistance training used by these athletes. Track and field jumpers and decathletes were originally chosen to represent a power
trained group because of the high degree of fitness required by their comprehensive training program. Additionally, because these athletes train primarily for jumping power, and therefore perform extensive daily exercises involving the calf, it was expected that some enhancement of calf muscle function would be reflected in the peripheral blood flow measures. It was therefore, not surprising that the power athletes had a significantly higher maximal vascular conductance than the healthy sedentary subjects (p<0.05), and exhibited a tendency for a higher blood flow (68.5±3.9 vs 54.5±3.0).

Despite almost identical blood flows (87.1±5.3 for the elite runners vs 89.1±6.5 for the body builders), the body builders exhibited a higher (also not significant) conductance (0.72±0.04 for the elite runners vs 0.81±0.05 for the body builders). In this study, a rather limited sample (n=7) of body builders was evaluated, and therefore, it is possible that a larger number of subjects might have yielded a significant difference. Furthermore, three of the seven body builder subjects were level I/II competitors (i.e. municipal/regional), and therefore, had a relatively shorter training history. Interestingly, these subjects also exhibited the lowest maximal vascular conductance values. Figure 4.4.8 clearly shows that a strong linear relationship (r=0.80) exists between the number of years of training and maximal vascular conductance. The relationship between the years of experience and Gmax demonstrates an association between training and Gmax among the body builders, but not the elite runners (figure 4.4.9). This indicates that if a structural enlargement of arteriolar structure can occur in response to training, then it would more likely be reflected in body builders with a long training history.
5.6 The Possibility of a Structural Increase in the Cross-Sectional Area of the Arteriolar Bed

5.6.1 The Conductance - Mean Arterial Pressure Relationship

In the body builders examined in this study, there was a tendency for lower blood pressures at rest. Previously, low levels of resting blood pressure have been reported among strength trained individuals. Possible explanations offered include a decreased body fat, a decreased level of body salt, and alterations in the sympathoadrenal drive (Fleck, 1988; Goldberg, 1989; Stone et al., 1991). This study provides data on blood pressure at rest and following both submaximal and maximal exercise of a small muscle mass, as well as corresponding vascular conductance data. Perhaps an alternative interpretation for the lower blood pressure seen in the body builders can be used to interpret the observations in the current investigation.

Vanhees et al. (1992) found a decreased arm muscle blood flow (resting brachial artery blood flow), despite an unchanged cardiac output, after running and cycling endurance training in previously untrained subjects. In addition, an earlier investigation reported a 36% higher resting blood flow ($p<0.001$) in the brachial artery of wheelchair confined paraplegics compared to age matched able bodied controls (Shenberger et al., 1990). Furthermore, an increased resting muscle blood flow, particularly in the fast twitch red muscle fibers, has been found in rats following training (Armstrong and Laughkin, 1984). Therefore, it would seem possible that after training, cardiac output at rest may be redistributed in favour of the trained muscle tissues. Since bodybuilding involves a concerted effort to
develop all skeletal muscles, it may be presumed that there would be a hyperkinetic blood flow at rest to all muscle tissues, potentially resulting in an elevated cardiac output. Although cardiac output was not measured in the present study, it has been shown both in literature cited above and through the observed data on the resting heart rate of the body builders in this study, that it is unlikely that cardiac output increased by an order of 36%.

Vanhees et al. (1992) did not present a hypothesis for the mechanism involved in the redistribution of cardiac output. One possible answer lies with the sympathetic nervous system. Although it is clear that physical training decreases sympathetic activity in hypertensive subjects (Tanji, 1992), many studies have shown an increase in resting epinephrine levels of healthy subjects following physical training (Kjaer et al., 1984; Lehmann et al., 1984; Kjaer et al., 1985; Svendahag et al., 1985). Additionally, end organs have specific independent thresholds for response to circulating hormones (Tanji, 1992). In the unique case of body builders, an increase in sympathetic tone throughout the body may act as a balance to an increased structural size of the arteriolar bed in trained muscle in an attempt to maintain TPR at constant levels, with cardiac output remaining at near normal levels. One major implication of this theory is that resting blood flow may be in excess of metabolic demands. Such a situation is plausible considering vasomotion, which allows for more or less blood flow, depending not only on demand, but also supply.

The lower blood pressure may reflect a practical compromise in bodybuilders between further increases in sympathetic tone and increases in the functional size of peripheral skeletal arterioles at rest. In the face of a reduced TPR, a decrease in mean arterial pressure would maintain cardiac output according to Ohm's Law. Consistent with this premise, a significantly higher vascular conductance was seen at
rest in the body builders compared to the other groups (p<0.001). The three other groups were not different from each other. The fact that body builders train all muscle groups equally suggests that the vascular conductance measured in the calf is probably representative of the other muscle tissues in this subject group. Furthermore, the body builders did not participate in an extensive aerobic conditioning program which could otherwise have been implicated in an increased calf muscle vascular conductance relative to the other muscles.

Arteriolar neoformation has been well documented in animals (Hudlicka et al., 1992), so it seems likely that it could occur in humans as well. The possibility that an increased arteriolar structure is responsible for a decreased resting TPR in body builders is indirectly supported by the findings of Egan et al. (1987), who implicated structural changes in the arterioles as the reason for an increased vascular reactivity in mildly hypertensive individuals. It was found that normotensives and hypertensives required the same amount of an intra-arterial infusion of norepinephrine to obtain a 30% increase in forearm vascular resistance, indicating that μ-receptor sensitivity was not different between the two groups. Greater resistance at maximum vascular contraction was also seen in the hypertensive subjects despite no difference in μ-receptor sensitivity. The authors speculated that this was probably due to a smaller vascular lumen prior to the vasoconstrictive intervention. Based on this evidence, it could be hypothesized that a hypotensive effect would be seen in subjects with a very large arteriolar structure, assuming μ-receptor sensitivity was not significantly modified. Therefore, a decreased blood pressure could also reflect a larger arteriolar bed irrespective of changes in sympathetic stimulation.

The present study corroborates Reading et al. (1993) and Snell et al. (1987) in that a higher Gmax and VO2max was observed in the elite runners than in healthy
sedentary subjects, in the presence of similar blood pressures. However, the body builders exhibited a G max equal to that of the elite runners, with no positive correlation to VO₂max, and a lower mean arterial pressure response to maximal ischemic calf ergometry. This suggests that a structural increase in the cross sectional size of the arteriolar bed is an adaptation to chronic bodybuilding training.

Since the tendency for turbulent blood flow increases in direct proportion to the velocity of flow, as well as the size of the vessel, a structural adaptation may be preferential to a functional one in response to training stimuli. In other words, if maximal or near maximal blood flow is regularly elicited, it may well be advantageous to have a larger number of vessels available than to have fewer vessels regularly dilated to a maximal diameter.

### 5.6.2 Bodybuilding Versus Running

The structural versus functional adaptive response in the arteriolar bed is not necessarily the same in elite runners as found in the body builders. Although several adaptive responses within and between muscle fibers are of a similar nature, others are different as evidenced by the finding of a similar VO₂max between body builders and healthy sedentary subjects. Interestingly, a study which examined muscle blood flow in the rat during different speeds of running concluded that the increase in muscle blood flow during exercise was directly related to the extent of the fast oxidative glycolytic fiber population recruited (Laughlin and Armstrong, 1982). Furthermore, the strong positive linear relationship between years of training and Gmax indicates that improvements in Gmax are a strong adaptive response specific to bodybuilding.
There are major differences in the training stimuli involved in running and body building. Based on the findings of Walloe and Wesche (1988), that the rate of blood flow is proportional to the force of contraction, it is possible that during body building, which typically utilizes a set of ten to fifteen repetitions to muscle failure, maximal flows are elicited, whereas they are not during running. Furthermore, during a typical training set, the body builders do not allow the muscle to relax between the eccentric and concentric phases thereby creating an ischemic intramuscular environment. Such stimulation is likely to elicit a significant reactive hyperemia post exercise. This is intuitive in that body builders rely on "pumping up" through the use of such ischemic sets immediately prior to posing in training or in competition. On the other hand, during running the force exerted in each contraction is likely below the level achieved during body building, and therefore, in response to running training, there may be a functional optimization within the structural limitations.

Finally, evidence for structural changes in the arterioles of bodybuilders is given in the strong linear relationship \((r=0.94, p<0.002)\) found between \(G_{\text{max}}\) and \(G_{\text{rest}}\) (Fig. 4.4.6). Within the other groups, no significant relationship was exhibited (Fig. 4.4.5). In order to examine the possibility that this relationship may just be a reflection of the mean arterial pressure relationship, the relationship of \(BF_{\text{max}}\) and \(BF_{\text{rest}}\) was also examined. An almost identically strong relationship was found \((r=0.93, p<0.003)\) (Fig. 4.4.7). These data are very interesting when considering the fact that the neurally driven vasoconstriction is likely overridden during reactive hyperemia. Recalling Ohm's law, these findings suggest that a centrally mediated explanation of the observed \(G_{\text{rest}}\) in bodybuilders is unlikely.

These relationships suggest a common factor that determines resting and maximal vascular conductance and blood flow in body builders. Moreover, given that \(G_{\text{max}}\)
reflects the size of the arteriolar bed, in the power athletes, the elite runners, and the healthy sedentary subjects, Gmax is probably not related to the resting vascular conductance; however, it is in body builders, suggesting that the higher Grest also reflects a structural change in the size of the arteriolar bed.

In conclusion, the findings of a very high maximal vascular conductance in conjunction with a lower mean arterial pressure in body builders, indicates a hemodynamic adaptation qualitatively similar to that observed with chronic endurance training. However, based on the whole body nature of bodybuilding, the magnitude of this adaptation is relatively greater, e.g., a decreased resting mean arterial pressure, due to a decreased TPR, occurs to a larger extent in response to chronic body building rather than to chronic endurance training.
5.7 Maximal Aerobic Power and Maximal Vascular Conductance

Figure 4.5.2 depicts the relationship between VO$_2$ max and Gmax in all groups. It can be seen that the relationship within the body builders group is quite different than that seen within the other groups. In fact, a significant negative relationship ($r=-0.88$, $p<0.03$) was found between VO$_2$ max and Gmax when one subject with an exceptionally high VO$_2$ max was excluded; this is probably the direct result of an increased muscle mass with increased years of training and performance level. The muscle mass increase of the body builders was disproportionately in upper body muscles, and therefore, likely resulted in the lower VO$_2$ max (expressed relative to body mass). An alternative interpretation is that the body builders have a lower extraction of oxygen relative to Gmax, than any other group.

The dissociation of VO$_2$ max and Gmax in the body builders compared to the healthy sedentary’s and elite runners as illustrated in figure 4.5.2 clearly shows that the body builders are different from the other groups, including the power athletes. They tend to have a lower VO$_2$ max than would be predicted from their Gmax. Based on this finding, it may be concluded that Gmax can be increased without a simultaneous increase in VO$_2$ max.

Figure 5.6.1 illustrates that across a wide spectrum of endurance fitness, when body builders, elite runners, and heart failure subjects are excluded, a linear relationship exists between VO$_2$ max and Gmax ($r=0.86$, $p<0.0001$). The data for the healthy sedentary subjects from the present study fit very well with the prediction equation derived from the other groups. It is interesting to note that the data from the power athletes also fit very well to the prediction equation, despite the extensive training program these subjects have participated in over several years. The three groups which deviated from the trendline were the elite runners, the body
builders, and the heart failure patients in the study by Reading (1990). Reading attributed the deviation from the line in the heart failure subjects to a cardiac limitation to VO\(_{2}\)max. This is an unlikely explanation for the body builders. It is more likely that the body builders have a poorer extraction of oxygen for a given G\(_{\text{max}}\), and therefore, are unable to achieve the predicted VO\(_{2}\)max. On the other hand, the elite runners in the present study were indeed elite relative to the endurance trained subjects used in both Reading (1990) or Snell (1987) studies. Therefore, it is quite possible that these subjects fall above the prediction equation because they are able to extract more oxygen for a given G\(_{\text{max}}\).
Figure 5.6.1
Relationship between maximal vascular conductance and maximal aerobic power. JR = Reading, 1990; PS = Snell et al. 1987; JR-CHF = heart failure, Reading, 1990; HS = healthy sedentary, for the current investigation. The line indicates the best fit for the data of Snell et al. (1987) and Reading (1990).
Conclusions

The first hypothesis of this study was accepted:

1. Body builders demonstrated a higher maximal vascular conductance and lower blood pressure than power athletes, elite runners, and healthy sedentary subjects in response to maximal calf ergometry.

Additionally, body builders demonstrated a higher peripheral vascular conductance at rest than elite runners, power athletes, and healthy sedentary subjects.

The second hypothesis of this study was also accepted:

2. Elite runners demonstrated a higher maximal oxygen consumption, and body builders demonstrated a lower maximal oxygen consumption than would predicted by maximal vascular conductance by the relationship established by healthy sedentary and endurance trained individuals.

In conclusion, the findings of a very high maximal vascular conductance in conjunction with a lower mean arterial pressure in body builders, indicates a hemodynamic adaptation qualitatively similar to that observed with chronic endurance training. The type of resistance training employed by body builders may elicit vascular adaptations which are quite different from the traditional resistance
training methods employed by body builders. Future resistance training studies examining the training adaptations in muscle, and cardiovascular responses to exercise, will have to re-evaluate the resistance training method employed.

The data from the elite runners has shown an optimization of oxygen extraction for a given maximal vascular conductance. The body builder data has shown a poorer extraction of oxygen for a given maximal vascular conductance. This is evidence of a central limitation to maximal aerobic power in this group. In contrast to the body builders, the power athlete data suggests that the type of training they employ has not dissociated the relationship between maximal oxygen consumption and maximal vascular conductance.

Recalling the the association of total peripheral resistance and maximal oxygen consumption shown by Clausen (1976), combined with the data from this study and the work of Reading (1992) and Snell (1987), it can be postulated that the size of the arteriolar structures within active muscle tissues influences total peripheral resistance even though maximal blood flow rates within those active muscles are probably not achieved during maximal or near maximal oxygen consumption (Anderson and Saltin, 1985). This suggests an alternative interpretation of the significance of maximal vascular conductance. Perhaps during maximal or near maximal whole body exercise, a higher maximal conductance allows for a greater blood flow prior to an elevation of systemic blood pressure.
**Recommendations for Future Research**

Due to the fact that this study was cross-sectional by design, direct proof of a structural enlargement of the arteriolar bed should be the main objective of future studies.

The cardiorespiratory and cardiovascular adaptations resulting from body building, power training and high intensity endurance running were examined in subjects who had participated in specific extensive training programs over a period of years. This study clearly demonstrates a need for a longitudinal exercise training study involving various groups utilizing the two distinctive types of resistance training. Previous studies which attempted to determine whether resistance training alone could increase VO$_2$max may have failed because they did not use the bodybuilding method of resistance training.

Venous occlusion plethsmography should be utilized in conjunction with the skeletal muscle biopsy technique to examine the structural changes in the size of the arteriolar bed.

Since the elite runners exhibited both a high blood flow and a larger Gmax compared to the healthy sedentary subjects, it may well be that Gmax plays a role in the improvements seen in VO$_2$max. Therefore, further studies on Gmax and its role as a possible predictor of the microcirculatory response to endurance training should be conducted.

Further resistance training studies should be conducted to examine the various strength training methods, since the adaptive responses can be dramatically different.
REFERENCES


Reading, J.L., Masters Thesis: *Skeletal muscle vascular conductance, aerobic power and left ventricular ejection fraction in heart failure, healthy sedentary and endurance trained subjects*. University of Toronto, 1990.


APPENDIX 1

Approval by Review Committee on the Use of Human Subjects

University of Toronto

OFFICE OF RESEARCH SERVICES

Approval by Review Committee on the Use of Human Subjects

Principal Investigator: Dr. J. Goodman, Physical and Health Education

Title: Peripheral Blood Flow and Maximum Oxygen Consumption in Elite body Builders, Elite Distance Runners, and Healthy Sedentary Subjects

Review Committee: Dr. S. Thomas, Rehabilitation Medicine
Dr. R. Goode, Physical and Health Education
Professor R. Hewitt, Religious Studies

Documents Submitted to Review Committee: Study outline, consent form

Subjects: Elite runners, elite bodybuilders, and age matched healthy sedentary subjects

Procedures: As described in the attached consent form

Method for Obtaining Consent: Consent form as attached. Subjects are to be given a copy of the form to keep.

Remarks:

Date of Approval: May 3, 1993

*During the course of the research, any significant deviations from the approved protocol and/or any unanticipated developments within the research should be brought to the attention of the Office of Research Services.

SP/tf

cc: Review Committee
Professor B. Kidd

Susan Pilon
Executive Officer
Human Subjects Review Committee

Simcoe Hall 27 King's College Circle Toronto Ontario M5S 1A1 Telephone 416/ 978-2163 Fax 416/ 971-2010
APPENDIX 2

Informed Consent

School of Physical and Health Education

University of Toronto

Lower Leg Muscle Maximal Conductance and Maximal Oxygen Uptake in Elite Runners, Elite Body Builders, and Healthy Sedentary Subjects

I __________________________ agree to participate in a research project sponsored by the University of Toronto. The project is designed to obtain information about the effects of different modes of training on the relationship between the maximal blood flow in my leg (called maximal conductance and blood flow) and my maximal oxygen uptake, or cardiovascular fitness.

I understand that two tests will be administered during the course of the study.

1. I will perform an exercise test on a treadmill. The exercise begins at a level that I can easily accomplish and will be advanced in stages, depending on my fitness level, up to my maximum exercise capacity, that is, to the point of exhaustion. The researchers may stop the test at any time because of signs of fatigue or I may stop when I wish because of fatigue or discomfort. I will breathe though a mouth piece which allows for the collection of my expired gases, for measurement of my oxygen consumption during exercise.

The exercise component of the test carries a low risk. There is a possibility that other changes may occur during the test, including abnormal blood pressure, fainting, disorders of the heart rhythm and, in rare instances, a heart attack (1/10,000 chance of a cardiac complication). Every effort will be made to minimize these through the preliminary examination and by observations during the testing.

2. Measurement of my lower leg blood flow, at rest, after light exercise, and after maximal exercise. During this test, the flow of blood to my leg will be temporarily stopped, using blood pressure cuffs filled with air at high pressure. An elastic band will be placed around my calf during this test, which measures my leg blood low. I will also have a small band on my finger, which will measure my blood pressure. I will exercise my calf muscle by moving my foot up and down against a pedal. During maximal exercise I will experience mild discomfort due to lack of blood flow to my lower leg but the discomfort will soon pass when exercise has stopped. This procedure will not result in any limitation in the immediate or long-term other than some temporary mild muscle fatigue. There are no known risks to this procedure.

I understand that any information obtained from this study may be published; however, my name will never be revealed. I have read the above and all questions have been answered to my satisfaction. I understand that I may ask additional questions and that I may withdraw from the study at any time without prejudice to my treatment. I have also been provided with a copy of this consent form.

Signature: __________________________ Date: ________________

Witness: __________________________ Date: ________________
APPENDIX 3

Performance and Training Data Questionnaire

<table>
<thead>
<tr>
<th>Name:</th>
<th>Date:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age:</td>
<td>Birth Date:</td>
</tr>
</tbody>
</table>

**Physical Data**

<table>
<thead>
<tr>
<th>Weight:</th>
<th>Date and results of most recent Body Composition Test:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height:</td>
<td>Date and results of most recent Exercise Tests:</td>
</tr>
</tbody>
</table>

**Best Achievements During the Past Twelve Months**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
</table>

**Please Describe Your Training:**

<table>
<thead>
<tr>
<th>How many years have you been training for your sport?</th>
</tr>
</thead>
</table>

Answer the following with regards to resistance training over the past twelve months:

<table>
<thead>
<tr>
<th>How many days per week?</th>
<th>How many hours per day?</th>
<th>How much volume?</th>
<th>Does your program include exercises for the lower leg?</th>
</tr>
</thead>
</table>

Answer the following with regards to running over the past twelve months:

<table>
<thead>
<tr>
<th>How many days per week?</th>
<th>How many hours per day?</th>
<th>How much volume?</th>
</tr>
</thead>
</table>

Does your program include any additional activities (e.g. cycling or swimming)?

If yes, please describe:

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
</table>

Is there anything else you would like us to know regarding your training?

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
</table>
Table 4  Heart Rate and Blood Pressure  
(30 sec Average)  
Values are means ± SEM

<table>
<thead>
<tr>
<th></th>
<th>HS</th>
<th>ER</th>
<th>PA</th>
<th>BB</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>8</td>
<td>10</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>At Rest</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart Rate (b min⁻¹)</td>
<td>67 ± 3 b</td>
<td>50 ± 3 ac</td>
<td>72 ± 3</td>
<td>66 ± 3</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>95.1 ± 4.6</td>
<td>90.7 ± 2.1</td>
<td>88.6 ± 2.6</td>
<td>82.7 ± 2.7</td>
</tr>
<tr>
<td>Following Submaximal Calf Ergometry</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart Rate (b min⁻¹)</td>
<td>74 ± 1.9</td>
<td>59 ± 2.7 ac</td>
<td>72 ± 2.9</td>
<td>75 ± 4.9 b</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>101.4 ± 3.7</td>
<td>99.6 ± 3.0</td>
<td>99.3 ± 3.8</td>
<td>88.2 ± 3.6</td>
</tr>
<tr>
<td>Following Maximal Calf Ergometry</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart Rate (b min⁻¹)</td>
<td>81.3 ± 2.0</td>
<td>67.4 ± 4.0</td>
<td>74.8 ± 4.2</td>
<td>77.6 ± 4.0</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>119.7 ± 2.9</td>
<td>120.4 ± 4.8</td>
<td>118.9 ± 2.8</td>
<td>103.3 ± 4.5 abc</td>
</tr>
</tbody>
</table>

a=p<0.05, (vs. HS), b=p<0.05 (vs. ER), c=p<0.05 (vs. PA)
<table>
<thead>
<tr>
<th></th>
<th>HS</th>
<th>ER</th>
<th>PA</th>
<th>BB</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>n</strong></td>
<td>8</td>
<td>8</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>Heart Rate (b min(^{-1}))</td>
<td>68±3</td>
<td>52±3</td>
<td>68±4</td>
<td>65±3</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>97.5±4.5</td>
<td>93.5±2.7</td>
<td>95.1±3.6</td>
<td>83.7±2.6</td>
</tr>
<tr>
<td>Blood Flow(^1)</td>
<td>3.60±0.24</td>
<td>3.16±0.17</td>
<td>3.53±0.28</td>
<td>4.73±0.41(^{abc})</td>
</tr>
<tr>
<td>Conductance(^2)</td>
<td>0.036±0.003</td>
<td>0.030±0.002</td>
<td>0.036±0.003</td>
<td>0.059±0.003(^{abc})</td>
</tr>
</tbody>
</table>

\(^{a}=p<0.05\) (vs. HS), \(^{b}=p<0.05\) (vs. ER), \(^{c}=p<0.05\) (vs. PA)

1 (ml (min 100ml\(^{-1}\)))
2 (ml (min 100ml mmHg\(^{-1}\)))
Table 6  Submaximal Blood Flow Measures  
Values are means ± SEM

<table>
<thead>
<tr>
<th></th>
<th>HS</th>
<th>ER</th>
<th>PA</th>
<th>BB</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>8</td>
<td>8</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>Heart Rate (beats min⁻¹)</td>
<td>89±9&lt;sup&gt;b*&lt;/sup&gt;</td>
<td>67±5&lt;sup&gt;c&lt;/sup&gt;</td>
<td>70±3</td>
<td>80±5&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>101.8±3.9</td>
<td>99.5±4.3</td>
<td>96.7±3.5</td>
<td>89.7±4.3</td>
</tr>
<tr>
<td>Blood Flow&lt;sup&gt;1&lt;/sup&gt;</td>
<td>23.6±3.5</td>
<td>26.58±4.10</td>
<td>24.88±2.74</td>
<td>26.40±3.71</td>
</tr>
<tr>
<td>Conductance&lt;sup&gt;2&lt;/sup&gt;</td>
<td>0.207±0.030</td>
<td>0.270±0.038</td>
<td>0.230±0.021</td>
<td>0.253±0.032</td>
</tr>
</tbody>
</table>

<sup>a</sup>=p<0.05 (vs. HS), <sup>b</sup>=p<0.05 (vs. ER), <sup>c</sup>=p<0.05 (vs. PA)

<sup>1</sup>(ml (min 100ml)<sup>−1</sup>)  
<sup>2</sup>(ml (min 100ml mmHg)<sup>−1</sup>)
Table 7  Maximal Blood Flow Measures  
Values are means ± SEM

<table>
<thead>
<tr>
<th></th>
<th>HS</th>
<th>ER</th>
<th>PA</th>
<th>BB</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>8</td>
<td>10</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>Heart Rate (b min⁻¹)</td>
<td>76±3</td>
<td>58±3</td>
<td>73±5</td>
<td>89±11</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>117.1±3.5</td>
<td>121.9±6.1</td>
<td>120.4±2.4</td>
<td>110.3±3.7</td>
</tr>
<tr>
<td>Blood Flow¹</td>
<td>54.52±3.00</td>
<td>87.09±5.28 Isle</td>
<td>68.48±3.90</td>
<td>89.14±6.49 Isle</td>
</tr>
<tr>
<td>Conductance²</td>
<td>0.456±0.022 Isle</td>
<td>0.721±0.038 Isle</td>
<td>0.570±0.034</td>
<td>0.807±0.054 Isle</td>
</tr>
</tbody>
</table>

a=p<0.05 (vs. HS), b=p<0.05 (vs. ER), c=p<0.05 (vs. PA)

¹ (ml (min 100ml)⁻¹)
² (ml (min 100ml mmHg)⁻¹)
<table>
<thead>
<tr>
<th></th>
<th>HS</th>
<th>ER</th>
<th>PA</th>
<th>BB</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>8</td>
<td>11</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>Calf Girth (cm)</td>
<td>37.3±0.91</td>
<td>34.2±0.34</td>
<td>37.8±0.64</td>
<td>40.4±0.87</td>
</tr>
<tr>
<td>Calf Skinfold (mm)</td>
<td>8.7±0.88</td>
<td>5.0±0.33</td>
<td>5.6±0.40</td>
<td>6.5±0.73</td>
</tr>
</tbody>
</table>

\[ a=p<0.05 \text{ (vs. HS)} , b=p<0.05 \text{ (vs. ER)} , c=p<0.05 \text{ (vs. PA)} \]
APPENDIX 9

The Significance of Bodybuilding

Adaptive Responses

Body building is often portrayed as a fringe sport, where the enthusiasts are seen as physical anomalies, and the sport is perceived as being riddled with the abuse of anabolic steroids and extreme surgical alterations. Most importantly however, it is not an olympic sport, and lacks the international organization necessary for legitimization. As such, it has not been as extensively studied as have other sports, such as, track and field. The intent of this study was not to alter the popular view of the sport, but rather to elucidate the cardiovascular training adaptations resulting from the training methods employed by bodybuilding enthusiasts.

The following adaptive responses may be obtained from body building training:

1. an increased resistance to fatigue (Tesch, 1987).
2. a decreased arterial lactate concentration in response to effort (Tesch, 1987).
3. an increased resting vascular conductance (this study).
4. a positive alteration in resting blood pressure and pressor response to effort (this study).
5. an increased maximal vascular conductance (this study).
6. an increased capacity for aerobic work in previously untrained individuals (Shantz, 1982; Tesch and Larsson, 1982; Tesch et al., 1989; Bell and Jacobs, 1990; Tesch, 1992).
7. an increased strength and power in those not previously strength trained.
The changes outlined above would benefit many groups of active and previously inactive individuals.

**Runners**

Many resistance training programs in which runners participate have not fully utilized body building as a resistance training method. One reason may have been simply that this type of training is difficult to justify to the endurance athlete because of the time required for both single training bouts, and to achieve the volume of work necessary to promote the appropriate muscular changes. Such a large volume of training time and effort might well be at the expense of the endurance activity itself.

However, body building training would benefit runners in many ways. First of all, they would gain at least the same amount of strength and power that are gained during a traditional resistance training program. More importantly however, this study has shown that these gains can be made without negative effects on the cardiovascular system. A more ambitious resistance training program might yield even greater gains in strength and power along with additional cardiovascular adaptations.

These gains in strength and power could be developed while simultaneously improving muscular endurance. Fast and slow fiber types would adapt in ways which would positively alter the lactate response to various workloads. In this way, beneficial changes to the lactate threshold would occur, which would benefit middle and long distance runners.
Power Athletes

Power athletes could employ the bodybuilding method without any detriment to power performance (Hakkinen et al., 1984). The cardiovascular benefits, such as a lowered mean arterial pressure, resulting from the bodybuilding method, versus the power training method, are an added benefit for health reasons. Furthermore, in many power events muscular endurance is also very important. This is obvious for events such as the 200 m and 400 m sprint, but also has value for jumpers, since it would allow a greater volume of training in a given session (personal correspondence with University of Toronto Track Club jumps coach; Carl Georgevski).

The multi event athletes are a special group which would perhaps reap the greatest benefit of all from the bodybuilding method. The decathlon consists of eight track and field events which are considered power events. The other two are the 400 m and the 1,500 m. Consequently, decathletes tend to follow the power athlete training model described in 2.4.3, and 1,500 m performances tend to be below what would be expected (as evidenced by the fact that the decathlon performance in this event is a lower fraction of normal event specific athlete performances). The bodybuilding method may provide a means for these athletes to improve on the weakest part of their performance, and still maximize their power development.
Other Populations

The lower VO$_2$max scores reported for untrained individuals in response to cycle ergometry compared to treadmill running has been attributed to a muscular limitation of the quadriceps muscle (MacNab and Conger, 1966; Sheppard, 1977). It seems likely that muscle function limitations are more significant at lower fitness levels, such as in sedentary elderly populations. It is likely that in these individuals, the relative muscle strength (%1RM) required even for basic activity is so great that blood flow is occluded, and glycolytic fibers are recruited (Edgerton 1978); therefore, a large amount of venous lactate is produced, resulting in premature fatiguing of the whole muscle. The bodybuilding method of resistance training might help to relieve this limitation by increasing absolute strength, vascular conductance, and the oxidative capability of all fiber types, all at the same time.