The effect of weight training on the heart

Andrew C. Fry, C.S.C.S.
Center for Youth Fitness & Sports Research
University of Nebraska
Lincoln, Nebraska

There are still many people who do not relate weight training to adaptations of the heart. Most people are primarily concerned with muscular and neural adaptations. However, the literature shows an increasing awareness of training induced adaptations of the heart. Many studies have examined the effects of timed circuit weight training on the cardiovascular system. But what are the effects of weight training on the performance and physical characteristics of the heart itself? Let us first look at the variables of heart performance.

Factors Influencing Heart Performance

The performance of the heart is measured in several ways. Heart rate of course is the first one to come to mind. As illustrated by Figure 1, both the sympathetic and the parasympathetic nervous system act to control the heart rate. Also, blood-carried catecholamines will influence HR. Stroke volume (SV) is another measure. This is the amount of blood expelled by the heart each beat. The SV is affected in a number of different ways, also illustrated by Figure 1. Besides catecholamine reaction and increased sympathetic input, there are a number of adaptations of the heart itself that can affect this. The bottom line is that there is an increase in the end-diastolic ventricular volume. Thus, with a greater volume of blood in the left ventricle, more blood can be pumped out.

As was noted earlier, heart rate is an important measurement of heart performance. When the heart rate is multiplied by SV, the total amount of blood pumped by the heart in one minute is determined. This is called cardiac output. Figure 1 shows the factors affecting cardiac output.

Another important measurement concerning the entire circulatory system is that of blood pressure. This is determined by multiplying cardiac output by peripheral vascular resistance, as shown in Figure 1. For all practical purposes, this is measured with a sphygmomano-


Figure 1 - Factors Affecting Heart Performance
meter. Both systolic and diastolic pressures are determined. Systolic pressure is the first figure given when reporting blood pressure. It is the amount of pressure that pushes blood through an artery during contraction of the heart, or systole. It is measured as the amount of pressure exerted on a column of mercury. The diastolic pressure is the pressure present in the arteries during diastole, or when the heart is in a relaxed state.

**Effects of Weight Training on Coronary Heart Disease (CHD)**

Paffenbarger, et al., in 1970 (24) and Paffenbarger and Hale in 1975 (25) observed the work habits and cardiac disease rates of San Francisco longshoremen. In both studies it was observed that the workers with the more physically stressful jobs had a lower incidence of CHD. It was also noticed by the researchers that much of the work done by the physically active longshoremen was either isometric and/or isometric in nature. It was calculated that the calorie expenditure of the more active workers was about 925 calories greater per day. The researchers tried to account for possible other sources of CHD, such as cigarette smoking. This study has led some scholars to believe that isometric and isotonic types of training such as weight training may have more beneficial effects on the heart than was once suspected.

**Effects of Weight Training on Heart Rate**

Several studies have been done showing that weight training of various types reduces a decrease in the resting heart rates of individuals. Morganroth, et al., in 1975 (21) observed a number of athletes, including a group of shot putters and wrestlers. The types of exertion in these sports is not unlike weight lifting (i.e., isometric and isotonic). It was noticed that their resting HR was lower than for an average individual.

It was noted by Scala in a 1984 thesis (32) that subjects taking part in a non-circuit Olympic style workout also exhibited lower than normal resting HR. The same results were reported by Vorosseyev in 1978 (32) while studying Russian Olympic weight lifters.

Several studies have noticed differences in HR following a short-term training protocol. Kanakis and Hickson in 1980 (13) trained nine males five days per week for 10 weeks. All exercises were lower body. Resting HR decreased from a pre-test of 65 to a post-test of 58. Stone, et al., in 1983 (30) trained 34 males for six weeks. Training included squats, bench press, and incline press three times per week. Resting HR decreased from a mean of 84.5 to 73.7. In 1979, Laird, et al., (16) found similar results in hypertensive adolescents. It was also noted by Blessing in 1983 and Gonyea (32) that HR during submaximal workloads was decreased after a short-term weight training format. Kusnititz and Keeny in 1958 (15) found that not only did resting HR decrease, but the subject's recovery rate decreased significantly also.

Both Ekblom, et al., in 1973 (5) and Frick, et al., in 1967 (10) studied the effect of short-term aerobic training on the sympathetic/parasympathetic input to the heart. Using drug treatment, the sympathetic and/or parasympathetic neural passages were blocked. Pre- and post-test results showed that the initial adaptation of the heart to exercise was due to changes in the neural input. It is speculated that this sort of adaptation could also occur via anaerobic work such as weight training.

**Effect of Weight Training on Stroke Volume and the Ventricle**

**Stroke volume.** In the Kanakis and Hickson study, the stroke volume (SV) was increased slightly, but no significant changes in the dimensions of the left ventricle were observed, either at end diastole or end systole. This may be accounted for in part by an increase in the shortening of the left ventricle during contraction. Recently, Fleck, et al. (8) used echocardiography to study the hearts of 17 highly strength trained subjects as well as a control group. Increased SV was observed in the trained group, both in absolute terms as well as in relation to body surface area.

**Left ventricle adaptations.** A number of researchers have observed physical changes in the structure of the left ventricle following weight training. The techniques most commonly used were X-rays and echocardiographs. Both of these techniques are noninvasive, with echocardiography being the preferred method.

Using X-rays, Abramyan and Dzhuganyon (32) in 1969 found that weight lifters had slightly larger left ventricular volume than normal. The increase however, was not as large as in endurance trained athletes. It was determined that this increase was related to the total volume of work done and the number of years trained.

An increase in the left ventricular mass was observed by Morganroth, et al. The shot putters and wrestlers in his study had a mean LV mass of 330g versus 211g for the control group. Brown, et al., (32) also found supporting data regarding increased LV mass.

Left ventricular wall thickness can also be increased according to Peronnet, et al. (26). It was found, using echocardiography, that athletes in general have thicker LV walls than sedentary people. It was also noted that the type of exercise was related to the level of wall thickness. Those athletes involving isometric exercise or high resistance isotonic exercise exhibited the highest levels of LV wall thickness. Morganroth, et al., also showed that wrestlers and shot putters had significantly thicker LV walls than did aerobically trained athletes. It appears that there may be a correlation between anaerobic training and LV wall thickness. The Fleck, et al. (8) study also reported an increase in posterior wall thickness and mass of the left ventricle, both absolute and as related to body surface area.

**Septal Thickness.** Menapace, et al. (20) using echocardiography, observed an increase in interventricular septum thickness in competitive weight lifters. He also found an increase in the ratio between the thickness of the septum and that of the free wall of the LV. These findings are supported by Fleck, et al. (8) who found an increase in diastolic septal
thickness in their weight training subjects. Once again, these were in terms of absolute values and as related to body surface area.

**Chamber Size.** Even though the performance of the LV was altered and the physical characteristics were changed in many ways, many studies found that there was no significant change in the size of the chamber (13, 20, 21, 26). Change in chamber size occurred only in aerobically trained individuals, not anaerobically trained subjects such as competitive weight lifters.

**Longitudinal Studies.** Ricci, et al. (28) trained 12 subjects for 20 weeks. Training consisted of five upper body isotonic exercises performed three times per week. The subjects showed a significant increase in strength, but no changes were observed in the wall thickness or the chamber size. There was a 4 percent increase in LV mass following the study.

Snoeckx, et al. (29) used weightlifting running and very low intensity weight training with low volumes over an 18 week period. Even at these low levels, an increase in LV wall thickness was noticed.

Jaweed, et al. (12) studied the effects of a weight training protocol on rats. The rats had weights strapped to their bodies and had to climb a ladder. Exercise bouts occurred twice a day, five days per week, for six weeks. The researchers observed a significant increase in the heart weight, and in the heart weight/body weight ratio. There was evidence of myocardial hypertrophy.

Muntz, et al. (22) did an interesting study on isometrically trained cats. Eleven cats were trained for two to nine months by having to hold a weighted bar with their paw in order to get their food. Radiopaque markers were implanted on the LV walls, both internally and externally, and anteriorly and posteriorly. At the conclusion of their training period, the cats were sacrificed and the weights of each chamber of the heart was recorded. All chambers significantly increased in weight, even when related to body weight. After one month of training, LV wall thickness increased 13.5 percent.

After six months of training, the wall thickness increased 32.5 percent. After sacrifice, it has also noted that the cardiac muscle fibers had increased in size significantly when compared to the control group.

Kanakis and Hickson also noted increases in LV wall thickness and LV mass in their previously mentioned study.

**LV Performance Versus LV Wall Thickness.** In a unique study, Lundin, et al., (17) studied hypertensive rats. With drug treatment, the sympathetic input and catecholamine reaction was negated. Increases in blood pressure were manipulated via infused blood and a constricting “noose” around the aorta. In order to maintain a normal stroke volume, it was observed that the cardiac muscle hypertrophied. Thus, LV performance was related to LV wall thickness.

**Effect of Weight Training on Blood Pressure.**

MacDougall, et al. (18) monitored arterial blood pressure during isotonic weight lifting via a catheter in the brachial artery. A mean BP of 355/281 mmHg was recorded for subjects while doing a double leg press, while the highest reported pressure was 450/310 mmHg. In another study MacDougall, et al. (19) also reported a case of extremely high pressures being the possible cause of brainstem damage in a weight lifter.

In a recent related article, Fleck and Dean (9) also monitored intra-arterial blood pressure during weight training exercises. They compared experienced body builders with novice weight trained individuals and with a sedentary control group. They found that the body builders had significantly lower mean blood pressures during exercise than did either of the other groups. The highest recorded mean blood pressure of 185/144 was found among the novice lifters during one arm dumbbell presses, which was considerably lower than the values recorded by MacDougall, et al.

Several studies have demonstrated an increase in BP during isometric training (1, 4). Both Fardy (6) and Nutter, et al. (23), report that the increase in blood pressure depends on the contraction intensity, the length of time the contraction is held, and the amount of muscle mass contracted. Petrofsky (27) even speculated that the amount of fast twitch fibers utilized was related to the increase in BP.

Kiveloff and Huber (14) studied the effects of an isometric training regimen on hypertensive subjects. Six second isometric contractions, repeated three times daily for five to eight weeks were associated with the lowering of resting BP. Brossseau found similar results in a 1981 study (2).

Fixler and Laird (7) used isotonic weight training with adolescent hypertensive boys which resulted in a decreased resting BP. In 1983, Stone found a decrease in resting systolic pressure, but no change in diastolic pressures after eight weeks of olympic style weight training (31). Goldberg, et al. (11), however found decreases in both the systolic and diastolic pressures after 16 weeks of weight training.

Collander and Tesch (3) observed the effects of weight training on resting and exercise blood pressure among normotensive subjects. They found no significant difference in resting and exercise (cycle ergometer) systolic blood pressure between experienced body builders and a control group of medical students.

In a study done in 1979, Viitasalo (33) showed a relationship between increased lean body mass and increased BP. On the other hand, both Stone et al. and Goldberg et al. demonstrated a decrease in BP despite an increase in lean body mass. There is the possibility that outside factors may have caused the increased BP in Viitasalo's study.

It should be noted that possible causes of chronic elevated BP among weight lifters are over training, the use of androgens, and an increase in body mass (32, 33).

**Effect of Weight Training on Myocardial Oxygen Consumption.**

Myocardial Oxygen Consumption (MVO,) is generally estimated by using the double product formula of HR x Systolic BP. In the Goldberg, et al., study (11), the double product was calculated for five males and six
females. The subjects were trained for 16 weeks, doing five upper and three lower body weight lifting exercises. Because of the significant decrease in BP, the double product values for all the subjects decreased, signifying increased myocardial oxygen efficiency. The subjects trained via olympic lifting in the Stone, et al., study cited above (31) also showed a decrease in the double product in the post-test analysis. In Colliander and Tesch’s study (3) the experienced body builders showed a significantly lower double product during exercise than did the control group. Since the systolic blood pressures did not differ, the lower double product was the result of a lower heart rate during the exercise. It stands to reason that with decreased BP and/or HR via weight training, as illustrated by numerous studies already cited, the myocardial VO₂ would improve somewhat.

Summary of Exercise Effects

Weight training can induce decreases in HR, both resting and at submaximal workloads. The changes can be significant, but not nearly as great as those experienced by aerobically trained individuals. Stroke volume can also increase slightly, though once again, not nearly as much as in the aerobically trained person. The increase in SV is apparently not caused by an increase in chamber size, but rather through an increased end diastolic volume of the heart.

While the chamber size may not change significantly, the LV walls and the septum may increase in thickness, a sign of muscle hypertrophy. The mass of the LV may also increase. There is evidence that increased mass may occur in all the chambers. There also seems to be a relationship between LV wall thickness and LV performance as measured by SV.

The heart also becomes more efficient at oxygen utilization. Both by decreasing the heart rate and decreasing the blood pressure, the heart has to do less work and is thus oxygen sparing. This can be estimated by the double product. As evidence by the preceding review, weight training does cause the heart to adapt in a number of ways. These should be taken into account when training programs are developed for various populations.

References


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Friday, June 20
□ #1 - Strength and Conditioning Facility Design — Tom Richardson
□ #2 - The Effects of Variability and Recovery Methods on Exercise Capacity — Bud Charniga
□ #3 - Goal Setting in Strength and Conditioning — Ed Cerny
□ #4 - Method of Exercise Variability to Enhance Recovery and Avoid Over-Training — Istvan Javorek
□ #5 - The Effects of Weight Training on the Cardiovascular System — Dr. Mike Stone
□ #6 - Observable Technique Breakdown in Plyometrics and Strength Training Due to Fatigue — Robb Rogers, C.S.C.S.
□ #7 - Bridging the Gap: Principles of Power Development and its Relationship to Athletic Performance — Frank Costello
□ #8 - Exercise and Nutrition — Dr. Joe Dzidras
□ #9 - Test and Measurements Data Collections and Presentation — Doug Semenic, C.S.C.S.
□ #10 - Research Considerations on Power — Brad Hatfield
□ #11 - Scientific Conditioning and Nutritional Considerations for Boxing — Mackie Shilstone
□ #12 - Special Olympics Training School — Frank Costello
□ #13 - Anabolic Steroids: The Hidden Addiction — Larry Pacifico
□ #14 - NSCA Annual Business Meeting

Free Communications
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Saturday, June 21
□ #15 - Biomechanics of Specificity — Dr. Everett Harmann
□ #16 - Program Considerations During Maturation — Gene Baker
□ #17 - Review of Muscular Force and Function (Structure, Excitation and Contraction) — Dr. Gary Dudley, C.S.C.S.
□ #18 - Basic Introduction to Strength Program Construction — Bruno Pauelto, C.S.C.S.
□ #19 - Administrative Considerations in High School Strength and Conditioning Programs: Time, Facility, Program — Jay Omer
□ #20 - Considerations in Eccentric Training — Dini McCurry
□ #21 - Specificity of Exercise to Improve Lateral Speed and Agility — Vern Gambetta
□ #22 - Gaining Support for the High School Strength and Conditioning Program — Randy Johnson
□ #23 - The Challenge of Championship Conditioning — Keynote Speaker — Rater Johnson
□ #24 - Conditioning Methods: Improving Speed and Acceleration — Vern Gambetta
□ #25 - Exercise Methods: Chest, Triceps, Shoulders — Pat Jacobs
□ #26 - Development of a Program and its Variables — Dr. William Kraemer
□ #27 - Conditioning Methods: Plyometrics — Dr. Don Chu
□ #29 - Periodization — Dr. Harold O'Bryant
□ #30 - Conditioning Methods: Warm-Up and Flexibility — Dan Wathen, C.S.C.S.
□ #31 - Exercise Methods: Legs and Neck — Keith Irwin
□ #32 - Administrative Variables of Program Design — Ed Bielik

Sunday, June 22
□ #33 - Designing Your Program for Swimming — Tim McClellan
□ #34 - Designing Your Program for Basketball — Lawton Hydrick
□ #35 - Designing Your Program for Wrestling — Mark Hood
□ #36 - Designing Your Program for Shot and Discus — Oskar Jakobsen
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