Roundtable:

Cardiovascular effects of weight training

1. Does weight training increase the size of the heart? If so, how does this differ from enlargement from aerobic work? from coronary heart disease?

Daniels: Research indicates that long term weight training causes increases in the size of the heart. While there are some conflicting reports, evidence is accumulating in favor of the position that ventricular wall thickness is increased. Many of the studies which report this, however, are cross-sectional studies which compare weight trained athletes with endurance trained athletes and sedentary controls. The problem with these studies is that they do not account for genetic differences between these subjects which may predispose them to select a particular type of training. There is some evidence that after years of detraining, the size of the heart returns to normal. This would indicate that the increase in size is a training adaptation.

The enlargement seen in weight training differs from that seen with aerobic training. Weight training apparently results in an increase in ventricular wall thickness whereas aerobic training causes an increase in the size of the ventricular cavities. This difference is attributed to the difference in training stimulus. With weight training there is an increased afterload on the heart, i.e., there is increased resistance to the pumping of blood. On the other hand, during aerobic training the increase in venous return results in an increase in the preload on the heart. There are other factors that are also involved but this highlights the difference.

In heart disease, the hypertrophy is an adaptation that occurs in an effort to maintain normal stroke volume. In disease, cardiac muscle length is increased which results in an increased tension during contraction. Obviously, the cellular and biochemical changes that occur are different than those that occur in adaptation to the training stress. The training stimulus results in a stronger heart that is capable of increased force of contraction and there is an increase in the cardiac reserve. The changes that take place in disease are an attempt by an ailing heart to maintain pumping capability by stretching cardiac muscle to increase the force of contraction and is often accompanied by a loss of the cardiac reserve.

Goldberg: Although weight training is not synonymous with isometric exercise, heavier resistive exertion does have a high isometric component. During this type of weight training, mean aortic pressure increases. Studies involved with athletes who participate in activities which have a considerable degree of isometric contraction have demonstrated left ventricular hypertrophy (enlarged left ventricular muscle mass). Kanakis and Hickson (8) showed that a ten week program of strength development increases left ventricular mass as those noted in strength-trained athletes (13). The increase in left ventricular wall mass without proportionate alteration of left ventricular volume may be secondary to the intermittent increases in aortic pressure during weight lifting. This characteristic architectural change is much like that change which occurs in a pressure overload state. In contrast, the alteration in the cardiac structure resulting from aerobic work is most like a volume overload state with increased left ventricular volume, as well as modest increases in left ventricular muscle mass (14). The long term health implications of these adaptive effects are as yet unknown.

Myocardial oxygen consumption or demand is related to architectural, intrinsic myocardial function and other factors such as heart rate and systolic blood pressure. A larger radius or left ventricular chamber diameter will increase the myocardial wall tension and thus increase the heart’s oxygen requirements. The thicker or more hypertrophied wall of the left ventricle reduces myocardial oxygen demand by lowering left ventricular wall tension. Thus, weight training, by not increasing ventricular chamber size while concommittently increasing left ventricular wall thickness, by itself
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potentially results in reduced myocardial oxygen demand. This change in the cardiac structure might be an advantage to patients with limited myocardial oxygen supply (i.e. fixed coronary artery disease) by reducing wall tension. However, this may also represent a drawback to the patient with ischemic heart disease as more muscle is available for ischemia, if no change in the coronary blood supply from collateral circulation was present thus increasing the symptoms of coronary heart disease.

MacDougall: Although there is still considerable controversy surrounding this topic, it is generally considered that weight training causes an increase in ventricular wall thickness but little or no change in ventricular chamber size. Endurance training, on the other hand, is assumed to cause less increase in wall thickness but a greater enlargement in chamber size. These conclusions are based mostly on cross sectional studies and have yet to be substantiated by true longitudinal (before and after training) studies.

Several investigators have found echocardiographic evidence of increased ventricular wall thickness in weight lifters and bodybuilders but such differences are less dramatic when they are normalized to body weight or to lean body weight (1). In our laboratory, we have found that approximately 60 percent of the elite bodybuilders, on which we have performed echocardiography, demonstrate significant ventricular hypertrophy but normal ejection fractions.

It is known that myocardial hypertrophy will result from an increased pressure load on the heart caused by certain pathological conditions such as aortic stenosis. Ventricular hypertrophy in strength athletes is thus considered to be a normal response to the increased peripheral resistance experienced during repeated forceful contractions.

Longhurst: Weight training has been shown to cause enlargement of the heart called concentric hypertrophy. Concentric hypertrophy is an increased thickness of the septum and free wall of the left ventricle with no change or a slight decrease in the internal diameter of the left ventricle. It is analogous although much less than the hypertrophy that results as a result of hypertension. Aerobic conditioning causes cardiac enlargement called eccentric hypertrophy. In this form of hypertrophy, the
heart walls do not thicken, but there is an increased diameter of the left ventricle. Since the same wall thickness extends over a larger surface area, the absolute mass of the left ventricle is increased.

The magnitude of the cardiac enlargement is much greater with aerobic conditioning than with weight training. Studies have demonstrated that the degree of cardiac enlargement with weight training is roughly proportional to the increase in skeletal muscle mass that occurs with this form of exercise. On the other hand, hypertrophy occurring as a result of aerobic conditioning is greater than the change in muscle mass. Therefore if one normalizes the heart weight by the body weight, lean body mass or body surface area, there is no enlargement of the heart as a result of weight training following aerobic training.

Coronary heart disease generally causes only a very mild degree of cardiac hypertrophy. The hypertrophy occurs in individuals that have sustained a myocardial infarction, but only in regions of the heart that have not infarcted. These normal regions hypertrophy to compensate for the regions that are infarcted. Thus, following a myocardial infarction there may be hypertrophy of the normal regions whereas the myocardium in areas with infarction is thinned.

Williams: Alterations in heart size dimensions resulting from training are dependent upon intensity of training and number of years of training. Investigations have suggested that weight lifters have larger ventricular dimensions than controls, although not nearly so developed as those of aerobically-trained athletes. Strength-trained athletes exhibit increased left ventricular mass, increased left ventricular wall thickness, and increased septal thickness as well as an increased septum to free wall ratio. Chamber size, however, has not been reported to be as affected. These latter findings are clearly different from those observations of large chamber sizes in aerobically-trained athletes.

The characteristics of the “athletic heart” are not considered abnormal and in fact, are often associated with dramatic augmentations of cardiac function. However, the increase in heart size observed with coronary heart disease, hypertension, and/or heart failure, is on the other hand, associated with an abnormal decrease in ventricular efficiency. The stimuli for these changes occurring with coronary heart disease are unclear but may be related to ventricular dilation and/or to areas of dysfunctional myocardium which serve to increase the work requirement of other areas of the heart and as affected by the disease process.

2. How does heavy weight training affect acute (during actual lifting) and chronic (normal, resting) blood pressure?

Daniels: The acute response to heavy weight training is an increase in systolic and diastolic blood pressure. A controversy centers around the extent of this increase. One group of investigators measured extremely high systolic and diastolic blood pressures (355/281) during lifting. However, these measures have not been duplicated and, in fact others have measured blood pressure levels considerably lower during similar lifts. In the latter study, experienced bodybuilders had a significantly lower rise in blood pressure than novice weight trainers or sedentary controls. Other studies indicate that blood pressure is somewhat proportional to the amount of muscle mass used for a particular lift.

Although there have been reports of increased resting blood pressure among weight lifters, most studies show that weight training has no chronic effect on resting blood pressure. In fact, other factors (e.g. anabolic steroids) have been suggested as possible causes of the increased blood pressure. In studies that have been performed on the effects of weight training on both normotensive and borderline hypertensive individuals, there was no effect on resting blood pressure. Therefore, weight training was considered to be a safe and effective form of training for these individuals. However, these studies also suggest that weight training is not an effective method for reducing borderline hypertension.

In my opinion, unless someone has severe hypertension, there should be no problem with a standard weight training program. I do not think that individuals who are weight training need to be unduly concerned about developing hypertension.

Longhurst: Any form of isometric exercise, including weight lifting, causes an increase in the systolic, diastolic and mean arterial blood pressures. The increase is gradual and during heavy lifts continues to increase throughout the time that the weight is lifted. If only a very light weight is lifted, i.e. one less than 15 percent of what could be lifted maximally, blood pressure rises 10-15 mmHg then remains constant. There is no evidence that chronic
weight training affects resting blood pressure. That is, weight lifting does not cause individuals to become hypertensive. There is an association of blood pressure with body size in that larger individuals tend to have higher blood pressures than thin individuals. However, weight lifters and particularly bodybuilders who have developed very large muscles do not appear to chronically elevate their blood pressure outside of the normal range.

**Williams:** The acute effects of weight training exercises result in blood pressure increases. Both systolic and diastolic pressures can be affected, depending on 1) the amount of an isometric component, 2) the intensity of contraction, and 3) the length of time of the contraction. However, in the normal cardiovascular system, the intermittent pressure increases during training, even the extreme values observed during high intensity weight training, appear not to be harmful.

Chronic weight training's effect on blood pressure is a point of contention primarily resulting from 1) the observations of hypertension occurring in some weight trained athletes, and 2) the reports of decreases in both systolic and diastolic blood pressure with chronic weight training. These latter findings of improved blood pressure, even with increased lean body mass, are encouraging. However, further study is still required to determine whether the hypertension observations in some weight-trained athletes are the result of the training itself or in fact, reflect potential concomitant variables such as preexisting hypertension, the use of anabolic steroids, or substantially increased body mass.

**MacDougall:** When forceful dynamic contractions are made, arterial blood pressure increases rapidly during each concentric contraction and declines rapidly towards resting level with each eccentric contraction. When maximal or near maximal contractions are made, or when submaximal contractions are continued to failure, blood pressure reaches extremely high values.

We have directly recorded blood pressure from a catheter in the brachial artery in experienced bodybuilders performing maximal lifts of various exercises, and found that mean group values for the leg press exercise (90 percent 1 RM to failure) reached as high as 320/250 mmHg, with pressures in some subjects being considerably higher (3). The degree of pressure rise is partially, but not directly, related to the amount of weight which is lifted and the muscle mass utilized. Pressure also increases with each repetition and reaches greater peak values when repeats of submaximal weights are continued to failure (10-15 RM) than when a maximal weight is lifted (1-2 RM).

We have repeated these studies with untrained subjects and even cardiac patients and have found the same pattern, but peak values were approximately 10 percent to 20 percent lower. Similar results have been reported by Drs. Freedson and Katch at the University of Massachusetts. During these tests catheters were inserted into the femoral artery and subjects performed bench press exercises. When blood pressure is recorded more distally, such as from the radial artery, much smaller increases are seen, possibly due to a "crimping" effect at the elbow.

Several minutes after a training session, blood pressure returns to pre-exercise level. I am aware of no evidence which indicates that regular weight training results in a chronic elevation of resting blood pressure. While there are undoubtedly many hypertensive weight trainers and bodybuilders, reports of a higher than normal incidence of hypertension among such individuals may be due to inadequacies of the conventional shagmomanometer method for measuring blood pressure in these subjects. The use of a standard sized cuff with large arms and a possible increased resistance to compression with larger lean tissue and fluid compartments (especially after subjects have "had their pump") may result in false overestimates of arterial blood pressure.

**Goldberg:** As in other forms of exercise, systolic blood pressure is increased during active weight lifting. While performing eight repetitions of moderately heavy weight, both men and women subjects equaled but did not exceed their peak systolic blood pressure (via doppler recording) as determined during treadmill exercise to volitional exhaustion during one of our pilot studies (3). Another investigation suggests that extremely high blood pressure levels are attained during very heavy lifts (12). However, little data actually exists which concerns the acute systemic blood pressure response to weight lifting.

During muscular exertion, a combination of increased cardiac output and vasoconstriction in the nonactive tissue helps to increase blood flow to the exercising muscles. An increase in blood pressure can be seen as a response to elevate perfusion pressure for needed blood flow to supply the active muscle with oxygen. Higher systolic blood pressure responses appear to be directly related to the percent of maximal voluntary contraction during isometric exertion, or to the degree of ischemia of muscle tissue during exercise (which is probably increased during heavier lifts) (15).

Diastolic blood pressure is also increased during exercise with a greater isometric component. Unlike systolic blood pressure, which is directly related to myocardial oxygen consumption or demand, diastolic blood pressure is related to coronary perfusion pressure (myocardial oxygen supply). Thus, increases in systolic pressure may be somewhat offset by diastolic pressure increases during resistive exercise.
3. What mechanisms affect acute heart rate increases during heavy weight training? What are the effects of weight training on resting heart rate?

MacDougall: Over a 10-12 RM set, heart rate increases rapidly to values which are 80 percent to 85 percent of that reached during maximal dynamic exercises such as running or cycling. A similar response occurs during maximal static or isometric contractions and, in fact, heavy weight lifting may be considered a series of static contractions interspersed by a partial relaxation of that particular muscle group.

Daniels: Heart rate is controlled by neural and hormonal mechanisms. Both of these factors probably play a role in the acute increase in heart rate seen during heavy weight training. The initial rapid response is undoubtedly due to neural stimulation. As muscle exercises, the metabolic demands are increased and there is a need for increased blood flow (i.e., cardiac output). Cardiac output is the quotient of mean arterial blood pressure divided by peripheral vascular resistance. We have already seen that blood pressure is increased during actual lifting. Total peripheral resistance will also be increased due to the isometric component of the lift which causes mechanical compression of the vasculature. There is also an increase in the vasoconstrictor tone in nonexercising muscle. The increased metabolic demand in the face of rising resistance is a factor that combines to increase cardiac output. Cardiac output is the product of the heart rate and stroke volume. Although stroke volume increases with exercise, the most rapid and expeditious way to increase cardiac output is by an increase in heart rate. The two mechanisms mentioned above are the means to increase heart rate. Increased sympathetic nervous activity results in an increase in heart rate. In addition, research has shown that a heavy weight training workout increases circulating levels of norepinephrine and epinephrine which also stimulates the heart.

The resting heart rate is either slightly decreased or not changed significantly by weight training. The stroke volume is reported to be slightly increased as a result of weight training. This enables the resting cardiac output to be maintained at a lower heart rate.

Longhurst: During heavy weight training heart rate increases frequently by 15-30 beats per minute above the resting rate. The mechanisms underlying this increase originate from a reflex generated in exercising...
skeletal muscle and from an increase in neural impulses from the brain, called central command.

The reflex from skeletal muscle presumably is stimulated by production of certain chemical factors and/or the change in muscle tension which stimulates sensory afferent nerves. As a result of the increase in afferent nerve traffic there is a withdrawal of parasympathetic neural tone and an increase of sympathetic neural tone to the heart. These two effects combine to increase heart rate.

Increased central command occurs as a parallel set of impulses along with the increase in motor nerve activity to activate the cardiovascular centers in the brainstem. Central command causes the same changes in autonomic tone, i.e. sympathetic and parasympathetic nervous systems, as does the reflex from skeletal muscle. The combination of the increased central command and the muscle reflex cause the increase in heart rate. The increase in central command causes the initial increase in heart rate during weight lifting. After 10 to 15 seconds, the reflex comes into play and helps maintain the increase rate throughout the period of weight holding.

Chronic weight training does not alter resting heart rate. Conversely, aerobic exercise lowers resting heart rate.

4. Can weight training affect VO₂ max levels, and to what degree? If so, what weight training variables (load, intensity, rest, etc.) can be manipulated to improve VO₂ max?

Goldberg: VO₂ max levels may be altered after weight training by both circuit lifting (utilizing higher repetitions and lower weight) and by more traditional strength acquisition techniques (higher weight with fewer repetitions). As the repetitions are increased and the weight reduced, a possible induction of muscle mitochondrial volume and density could be achieved, increasing the oxidative capacity of the exercised muscle. Studies have demonstrated modest increases in VO₂ in terms of total milliliters of oxygen consumed during exercise and ml of oxygen/kg of body weight (2). We have demonstrated increased maximal oxygen consumption in total milliliters and milliliters per kilogram after moderately heavy non-circuit style weight training which was not significantly different from the randomized subjects performing only aerobic exercise after a period of 16 weeks (11). However, these results were due to a change in body composition and muscle mass. Weight trainers and runners both lost weight. Whereas the runners only preserved lean body mass, weight trainers increased their muscle mass. When oxygen consumption was analyzed by observing VO₂/lean body mass, the weight training group was no different than when they were sedentary. In other words, increase in total oxygen consumption was due to increase in total muscle mass among the weight trainers, and probably not secondary to induction of mitochondrial oxidative enzymes.

Until other circuit training studies are analyzed by observing the effect of oxygen consumption upon lean body mass or by muscle biopsy it is difficult to determine whether weight training induces more efficient (oxygen consumption/unit muscle tissue) VO₂ rather than producing increased maximal oxygen intake by virtue of increased total muscle mass. Additionally, studies concerning oxygen consumption are limited as they usually observe VO₂ during treadmill or cycle ergometry and do not observe the effects of larger amounts of muscle mass which would include exercise of the upper body as well. Thus, the effects of upper body strength conditioning are limited as they have not been subject to the same degree of analysis.

Finally, although oxygen consumption is a useful tool to stratify aerobic fitness levels, clinically aid in understanding exercise limitations and aid in diagnosis and therapy for muscle myopathies, its levels have not been directly related to cardiovascular risk factor improvement. Attainment of a higher VO₂ level, by itself, as an adjunct for health is questionable.

Williams: Studies involving weight training have suggested that moderate increases in maximal oxygen uptake may be possible but are probably more likely to occur when the weight training program consists of high repetitions of primarily large muscle mass exercises on a frequent exercise session basis. And although elite weight lifters may have higher aerobic capacities than their nonathlete counterparts, most weight training programs should be complimented with a more typical aerobic training regimen if increasing one's maximal oxygen uptake is an important expected outcome.
Longhurst: In general, weight training increases maximal oxygen consumption when one does not account for the change in body mass. Therefore, the absolute oxygen consumption in liters per minute is increased. However, when one accounts for the increased body weight that generally occurs as a result of weight training, there is no change in $\text{VO}_2\text{max}$. Thus, when one expresses $\text{VO}_2$ in the units of mls/min/kg there is no change in the $\text{VO}_2\text{max}$. As such, most exercise physiologists do not believe weight training is a good method to improve $\text{VO}_2\text{max}$.

MacDougall: It is doubtful whether traditional weight training, of the high-resistence low-rep variety (6-10 RM) will increase $\text{VO}_2\text{max}$ and, in fact, it probably results in an actual decrease in $\text{VO}_2\text{max}$ relative to body weight. We have found that mitochondrial density decreases in muscle which has undergone hypertrophy through traditional weight training (2). This is apparently due to a dilution effect caused by increased contractile protein.

On the other hand, low-resistance high-rep training (30-40 RM) or circuit type training where the subject proceeds rapidly from one exercise to the next may provide enough stress on the oxidative energy delivery systems to result in an increase in $\text{VO}_2\text{max}$. Such training, however, would not be as effective as conventional endurance training for increasing $\text{VO}_2\text{max}$.

Hurley: Most studies indicate that weight training produces either no significant change or a statistically significant increase of less than 10 percent in maximal oxygen consumption ($\text{VO}_2\text{max}$). When compared to the 15 percent to 44 percent increase in $\text{VO}_2\text{max}$ that is normally observed with high intensity aerobic exercise training, it can be concluded that most weight training programs fail to result in a physiologically significant increase in $\text{VO}_2\text{max}$.

Daniels: Weight training can affect $\text{VO}_2\text{max}$ levels in only relatively untrained individuals. Weight training has not been shown to increase $\text{VO}_2\text{max}$ in trained individuals in a well controlled study. Weight training is not as effective as an aerobic training program in improving $\text{VO}_2\text{max}$. However, weight training may be a viable alternative when more traditional aerobic training programs are impractical or impossible. Weight
training may also be an appropriate choice for maintenance of aerobic fitness when other aerobic training programs are not available. Weight training could also be useful as a preconditioning program to help untrained individuals prepare for a strenuous aerobic training program. Those weight training programs that have shown improvement in \( \dot{V}O_{\text{max}} \) have generally used loads in the range of 40 percent to 60 percent of maximum with 8 to 12 repetitions and sessions lasting 20 to 30 minutes. The frequency was 3 days per week and the studies covered at least a 10 week period. The work/rest ratio was 2 to 1.

5. How does weight training affect stroke volume (SV) during lifting and at rest? How does this in turn affect cardiac output (Q)?

Longhurst: Stroke volume is not altered during lifting. However, since heart rate increases significantly during an episode of weight lifting, cardiac output increases. Like the acute effects of weight lifting on stroke volume, there is no significant change in stroke volume as a result of chronic weight training. That is, stroke volume at rest is unchanged. As mentioned previously, the type of hypertrophy that occurs with weight training is called concentric hypertrophy. This type of hypertrophy does not involve a change in the diameter of the left ventricle. Thus, one would not expect stroke volume to be altered by weight training.

Williams: The effects of weight training on stroke volume during weight lifting and between lifts (rest) are dependent upon the type of weight training being performed. Weight training regimes utilizing low weight with high repetitions of primarily large muscle mass exercises are more likely to result in effects more similar to aerobic exercise training, such as increased stroke volume with exercise and a tapering back to normal during the rest periods. However, if the acute bout of weight training involves heavy resistance and especially, if breath-holding and straining against a closed glottis (Valsalva maneuver) are present, the resultant hemodynamics are much different. Dependent upon the degree and duration of straining, venous return can be dramatically reduced beginning with the initiation of the strain. The declining venous return results in an decrease in stroke volume. In strains of mild intensity and duration, this fall in stroke volume is accompanied by an offsetting rise in heart rate in an attempt to maintain overall cardiac output. However, this increase in heart rate may not be adequate to offset falling stroke volume in strains of greater intensity and duration. At cessation of straining (rest), there is a sudden surge of venous return leading to a sudden increase in stroke volume at a time when heart rate is already accelerated. An increase in cardiac output results, and continues until a reflex bradycardia ensues and stroke volume and cardiac output normalize.

MacDougall: During lifting, there is a SV increase which helps to elevate blood pressure (3). The contraction and relaxation of the muscles apparently acts as a powerful muscle pump which enhances ventricular filling. This is in contrast to static exercise where SV declines due to impaired venous return to the heart.

Since resting heart rates for bodybuilders are quite normal, it is evident that resting SV is also unaffected by weight training.

MacDougall: We have found that even experienced lifters briefly close their glottis (Valsalva maneuver) or exhale forcefully through pursed lips or clenched teeth when performing maximal lifts or when approaching failure during multiple reps. Intrathoracic pressure suddenly increases causing an elevation of systemic blood pressure. Mechanically the Valsalva maneuver performs a fixator function and helps to stabilize certain body segments for muscles to act upon.

Most instructors advise trainers to avoid the Valsalva maneuver while lifting, but I am not certain whether this is sound advice. It has been shown that the increase in intrathoracic pressure with a Valsalva is transmitted to the cerebrospinal fluid as well. Rather than being undesirable, this may be an important safety mechanism which reduces the pressure difference across the cerebral vessels and thus protects them from damage caused by the extreme systemic pressure lifting causes. The increase in intrathoracic pressure would also provide an additional compression pressure for the heart and thus increase SV, despite extreme peripheral resistance. A brief Valsalva maneuver (which is the natural response) might therefore also be the safest response.

Longhurst: Holding your breath during a lift can have profound effects on the cardiovascular system. Scientists called this effect the valsalva maneuver. The affect of a valsalva maneuver is to cause a compression of the veins in the chest because of the high intrathoracic pressures that are generated. Compression of the veins impedes venous return to the heart and therefore reduces cardiac output. The reduction in cardiac output limits the increase in blood pressure and can cause frank decreases in blood pressure, i.e. hypotension. In turn, the hypotension causes a reduction...
in blood flow to the brain and active skeletal muscles. This could have significant consequences if the valsalva maneuver is maintained for a long period of time. The weight lifter could become dizzy or could experience muscle cramps. In addition, the heart rate may increase much higher than it usually does because of the reduction in blood pressure. To avoid these deleterious effects, individuals should not hold their breath any longer than necessary while weight lifting. Holding the breath for a few seconds does not significantly compromise blood pressure. Alternatively, if one can completely avoid holding the breath, there will be a normal increase in blood pressure such as should occur with static exercise. If one maintains a valsalva maneuver for more than 30 seconds, there may be some reduction in weight lifting performance.

Thus, hyperventilation could impair one's performance during weight lifting.

8. What effect do anabolic steroids seem to have on the heart profile when associated with weight training?

Hurley: Myocardial hypertrophy has been observed in strength-trained athletes. It is believed that the pressure overload produced from acute elevations in blood pressure results in an increase in cardiac mass. A recent study by Salkie et al. has addressed the question of whether anabolic steroids can augment the myocardial hypertrophy seen in strength trained athletes. They concluded that anabolic steroids had no effect on left ventricular chamber dimensions, septal or free wall thickness, and mass/volume relationships. Therefore, anabolic steroids do not appear to augment protein synthesis within cardiac muscle of healthy individuals.

The risk profile for heart disease however, is greatly affected by the use of anabolic steroids. In the past two years at least five published studies, all from different laboratories, indicate anabolic steroid use substantially increases the risk for heart disease in a very short period of time by altering the cholesterol containing lipoproteins. These results occur even at low dosage levels (e.g. 5 mg/day). According to data taken from the Framingham studies and our own data, the altered lipoprotein-lipid profile resulting from the use of anabolic steroids can be associated with a four-fold increase in risk for developing heart disease within four weeks of usage.

Daniels: The effects of anabolic steroids on the heart and cardiovascular system are neither well studied nor understood. What little evidence is available indicates that they may have some serious long-term effects on cardiovascular health. Studies indicate anabolic steroids may produce decreases in high density lipoproteins (HDL) while increasing low density lipoprotein (LDL) levels. Other studies indicate blood pressure may be increased. One report shows anabolic steroids produce myocardial hypertrophy and another report indicates the hypertrophy seen after steroid use is no different than that seen with weight training alone. Other reports indicate changes occur at the cellular level, which suggests pathological alterations. The bottom line based on the studies, to date, suggests anabolic steroids have some very detrimental effects on cardiovascular disease risk factors. It is important to note, however, studies have not established a direct link between cardiovascular disease and steroid use. I would suggest that anyone who is interested in this topic read the literature review by Wright and Stone in the NSCA Statement on Anabolic Drug Use.

Goldberg: We have had an opportunity to observe lipid and lipoprotein levels among various competitive weight-trained athletes prior to and after use of anabolic steroids in our laboratory. From a cardiovascular risk factor assessment this drug family appeared to adversely affect the lipoprotein profile among these athletes. Prior to anabolic steroid use, these weight trainers had low total cholesterol and relatively high HDL cholesterol (the protective cholesterol) levels which are associated with low risk for development of coronary heart disease. After just several weeks of anabolic steroid use, the ratio of total cholesterol to HDL cholesterol changed from low risk to very high risk profile for development of coronary heart disease. Other investigators have published similar results and showed this clinical supposition to be true (6). Thus, the potential for development of cardiovascular disease is likely enhanced by the frequent and prolonged use of these steroids. It appears that the effect of anabolic steroids on lipoproteins lowers the protective, high
density lipoprotein cholesterol (17) and without this protective lipoprotein more cholesterol is directed toward the arterial wall for deposition. Also, anabolic steroid use may be associated with elevated blood pressure which represents yet another increased risk factor for development of cardiovascular diseases.

MacDougall: In general, we find resting blood pressure increases when subjects take anabolic steroids but that blood pressure returns to normal within a few weeks of cessation of steroid use. Whether this increase is due to some emotional factor or to a greater fluid retention is not known. Likewise, the long-term effects of anabolic steroids on the circulatory system are not known.

Williams: Substantial evidence indicates the risk for coronary heart disease is directly related to blood levels of low-density lipoprotein cholesterol and inversely related to high-density lipoprotein cholesterol concentrations. Unfortunately, a number of investigations report the habitual use of anabolic steroids by bodybuilders and weight lifters dramatically and predictably reduces high-density lipoprotein cholesterol and increases low-density lipoprotein cholesterol profiles. These results sharply contrast the relatively increased high-density lipoprotein cholesterol and decreased low-density lipoprotein cholesterol levels reported in weight-trained athletes during training who do not receive anabolic steroids. Additionally, there have been suggestions that either by a direct effect or as a result of increased body mass, the use of anabolic steroids may be associated with hypertension. Consequently, the use of anabolic steroids by weight-trained athletes may increase their risk for coronary heart disease.

9. In your opinion, can weight training be an effective means for the prevention of coronary heart disease?

Williams: The singular value of any exercise regime as a means to prevent coronary heart disease is difficult to discern primarily because of the many confounding variables. It does appear that exercise within a prevention program might be evaluated in terms of its effect on the coronary heart disease risk factors. Little information is available as to the effectiveness of weight training in this regard. It does appear, however, in presumably healthy individuals not using anabolic steroids, that high repetition of primarily large muscle mass exercises on a frequent exercise session basis may produce small to moderate improvements in serum lipids and blood pressure regardless of body weight and dietary changes. Consequently, those persons with normal cardiovascular systems wishing to include some weight training in their overall risk reduction program should be encouraged to do so.

Daniels: I think weight training can be an effective part of a program to prevent coronary heart disease. Indeed, one of the early studies which demonstrated a beneficial effect of physical activity on cardiovascular disease was done on longshoremen. The types of activities in which they were involved resemble weight training more than any other type of training program. The recent report on Harvard alumni found that death rates declined steadily as energy expenditure increased from 500 to 3500 kcal per week. Therefore, I think weight training would be an effective means of increasing caloric expenditure and thus be a useful part of a coronary disease prevention program. I think such a prevention program must also be concerned with other factors such as nutrition, tobacco avoidance and stress reduction to be effective. I think in order to derive maximum benefit from a training program one should include some aerobic activity. That aerobic activity may be an intense circuit training program.

However, one concern I have is the lack of a demonstrated effect of weight training on coronary risk factors. None of the three major risk factors (smoking, cholesterol or hypertension) appears to be directly affected by a weight training program. Although I suspect there are few serious weight trainers who are active smokers I know of no data to support my belief. On the other hand, several reports indicate weight training has little or no beneficial effect on blood cholesterol levels or resting blood pressure. Therefore, I would have difficulty in recommending that weight training alone be used as the means to prevent cardiovascular disease. However, if the situation is such that it is the only type of training available I think that it would be preferable to a sedentary lifestyle. It is my opinion that a well balanced fitness program will contain both aerobic and weight training components and that the area of concentration will depend on the individuals interests, capabilities and goals.

Goldberg: Coronary artery disease risk factors have been well established. Weight training has been demonstrated to alter selected factors which could result in a decrease in the incidence of atherosclerosis. We have demonstrated a significant lowering of the cholesterol/HDL-cholesterol ratio among previously sedentary men and women who weight trained for just 16 weeks (4). Other investigators have shown similar beneficial changes (7). Weight training may have a salutary effect on blood pressure as well, with several studies demonstrating resistive exercise being related to lower blood pressure levels (1, 5). In addition, another potential risk factor for development of coronary heart disease is insulin plasma levels which are directly related to the incidence and prevalence of atherosclerotic heart disease (16). Weight training has resulted in lowering of insulin levels after glucose stimulation. Although no long-term research has been
performed, weight training has demonstrated beneficial changes of known and potential coronary risk factors. These beneficial modifications would appear to favorably reduce the possibility of developing coronary heart disease.

Longhurst: No data exists to suggest that weight training prevents coronary heart disease. If anything, the high blood pressures that occur during each period of weight lifting could cause a problem such as a stroke or a ruptured aneurysm in certain individuals predisposed to these problems. I do not believe one should substitute weight training for aerobic conditioning as a means to prevent coronary disease.

Hurley: Until recently it was believed by many exercise physiologists that weight training had no favorable physiological benefits. However, several recent studies suggest that weight training may result in a favorable modification of several risk factors for heart disease. These include hypertension, abnormal lipoprotein-lipid profiles, and hyperinsulinemia. Nevertheless, many questions need to be answered before we can conclude whether weight training is an effective means for modifying risk factors. Some of these unanswered questions include: 1) Are the favorable effects of weight training on risk factors simply an acute post exercise effect or do they represent an actual physiological adaptation from training? 2) How do these effects from weight training compare to those from the more commonly prescribed exercise, i.e., aerobic exercise training? 3) What are the factors involved in weight training that are most important for affecting these risk factors (e.g. number of repetitions, degree of resistance, time of rest intervals, threshold for total workload)?

References for Mark Williams

References for J.D. MacDougall

References for Linn Goldberg