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Hemodynamic alterations during exercise in the physically trained and the physically untrained human, Omaha, Nebraska

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HEMODYNAMIC ALTERATIONS DURING EXERCISE IN THE PHYSICALLY TRAINED AND THE PHYSICALLY UNTRAINED HUMAN

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INTRODUCTION

It is generally accepted that the efficiency of the circulatory system is increased so that bodily demands incurred during exercise are more effectively met by those who have undergone physical training. The untrained individual also, of course, responds to physical work by an increased circulatory effort. It is the purpose of this paper to review the alterations in the circulatory system produced by physical exercise in the physically untrained and the physically trained individual; and to compare, where information permits, the differences made by physical training.

Review of the literature has been limited to those studies based upon experimentation with athletes trained in sports requiring stamina such as swimming, cycling, or long distance running; and normal healthy subjects involved in everyday activities only. Age is not a constant, however the great majority of the subjects used were in their second and third decades. Most of the studies included only males, but a few of the articles did not designate the sex of the subjects under observation. As can be seen the selection of subjects has not been limited by strict criteria common to all studies reviewed. It can only be said that all subjects were healthy and either physically trained or not physically trained.

Methods of technique for the various measurements pertinent to these studies have, as to be expected, improved with the more recent studies. The various techniques will not be delt with

in detail by this paper, but will be mentioned as necessary when the various aspects of circulation measured are discussed. The method of exercise was usually by use of the bicycle ergometer and the position either supine or upright. These factors will be covered with the various studies.

Some general statements characterizing the athletes circulatory system will be beneficial before progressing to a closer look at the response of circulation to exercise and what alters this response. Cuming states that true cardiac hypertrophy occurs following physical training, as seen by autopsies of accidently killed athletes showing a 350 to 540 gram range in heart size. There is an increase in caliber of the coronary vessles which remains proportional to cardiac weight up to 500 grams. Hypertrophy beyond this size outstrips the increase in vessel size. The largest hearts are seen in endurance runners (30). Bevegård shows that heart volume is increased in the athlete. He found an average capacity of 1,087 milliliters in the prone position which was significantly more volume than found in the normal physically untrained subject of the same body weight (6). The total amount of hemoglobin is greater in the athlete than in the physically untrained by a significant difference also, but there is an increase in total blood volume such that the hemoglobin concentration actually remains fairly constant (5, 6, 30). The athlete possesses a slower heart rate as well as a larger stroke volume than the nonathlete (these perameters will be discussed in more detail later). Thus, the circulation system of the physically

trained is larger and more efficient than that of the physically untrained.

CARDIAC OUTPUT

The increased metabolism occuring in muscle associated with exercise requires a more rapid supply of oxygen to the muscle as well as more rapid removal of the catabolic products from the muscle. This demand is met by an increase in cardiac output. It has been traditionally thought that this increase in cardiac output, during exercise in normal subjects, is the result of increases in both stroke volume and heart rate. In recent years a number of studies with the direct Fick method or the indicator dilution technique have failed to support the view of an increasing stroke volume during work. Not all recent studies agree with this concept, but some of the discrepancies can probably be attributed to the fact that measurements in some cases were with the subject in a supine position and in other studies with the subjects in a sitting position. It is well documented that at rest transition from supine to erect position causes a decrease of the stroke volume (38, 1, 35, 46, 29, 14, 53). Differences in technique and material may also, of course, lead to the discrepancies seen in studies concerning this aspect of cardiac output.

Bevegard et al, compared circulatory response to exercise (bicycle ergometer) of well trained cyclists to normal nonathletic subjects by right heart catheterization employing the direct Fick

method with analysis of oxygen saturation and hemoglobin concentration of the blood spectrophotometrically. In this series of studies the increase in cardiac output on transition from rest to exercise was found to be almost exclusively a result of an increase in heart rate when the subjects were in the supine position. This was found to be true for the athletes as well as the nonathletes. In the sitting position it was noted that cardiac output was less than in the supine position by about two liters per minute at rest. and during moderate and heavy exercise. The difference was explained by a smaller stroke volume in the sitting position, both at rest and at work, which is apparantly due to a redistribution of blood to the lower part of the body from the central veins with subsequent decrease of ventricular filling. The stroke volume at rest was considerably less in the sitting than in the supine position, but increased substantially with mild leg exercise in the sitting position. After this initial change it remained constant during continued work of higher intensity as in the supine position, but at a level approximately 10 milliliters less. This difference in stroke volume between the two body positions is compensated for by a higher arteriovenous oxygen difference in the sitting position so that the oxygen transport per pulse beat is equal, and, consequently, the amount of work performed at a given pulse rate is independent of body position (5, 6). This effect of body position during exercise has also been demonstrated by others (10, 51, 39, 34).

In both the trained and the untrained subject exercise results in increased heart rate, minimal stroke volume increase, an increase in oxygen uptake, and an increase in arteriovenous oxygen difference. Whittenberger et al, showed that the normal individual has an increase in oxygen uptake and an increase in arteriovenous oxygen difference after mild exercise in the recumbent position. Work of the right ventricle against pressure increased three times fromthe resting level and that of the left ventricle increased about two times when exercise produced an oxygen consumption of 500 cc/min/M^2 (1 E). Dexter,et al, using normal subjects and the direct Fick method found that oxygen consumption during exercise increased three to four times from resting values with a widening arteriovenous oxygen difference, but that stroke output increase in only 50% of the individuals he tested. Associated with the oxygen consumption increase was a three fold increase in the right ventricular work against pressure and less than a two fold increase against left ventricular pressure (13). Cuming notes in his studies that the heart rate increases lineraly during exercise with respect to the work load and oxygen consumption between heart rates of 120 and 170 beats per minute, and believes that the increased cardiac output is due mostly to increased heart rate with little alteration in stroke volume (12). Kahler et al, found that in exercise producing a four to five fold increase in oxygen consumption there occurred a 68% increase in heart rate, 17% increase in stroke volume and an increase in left ventricular minute work of 129% in the normal untrained subject.

To show that the autonomic system is important to optimum circulatory response to exercise and that the sympathetic system appears to play the major role, atropine and guanethidine were administered in combination to these subjects. The heart rate increased only by 28% in response to the same amount of exercise, the stroke volume increased only by 1%, and the left ventricular minute work increased just 5%. Block of the parasympathetic system alone caused an increased heart rate and left ventricular minute work at rest and did not interfere with circulatory response to exercise (28). Donald et al, tested normal subjects by cardiac catheterization while they exercised in a supine position using a bicycle ergometer. Oxygen uptake increased promptly from a mean of 139.4 ml/min/M^2 upon initiating exercise, reached a steady level by the second minute and maintained a volume of 295 ml/min/M^2 for the remaining five minutes. Oxygen saturation of mixed venous blood decreased at the beginning of exercise reaching a steady level in one minute while the arterial saturation fell slightly in most subjects but increased again upon cessation of exercise. With light exercise oxygen uptake was two times the resting level, arteriovenous oxygen difference increased from 3.10 volumes percent to 5.69 volumes percent, and the cardiac output increased from 4.32 l/min/M^2 to 5.26 l/min/M^2. The parameters were found to gradually increase with increase in severity of exercise to an oxygen uptake of six to eight times resting values, an arteriovenous oxygen difference four times that found at rest, and an increase by two fold in cardiac output in heavy exercise.

The mean heart rate at rest was observed to be 81 beats per minute. During the first minute of exercise there was a rapid increase and thereafter as exercise continued only raised slightly or remained at a steady level until exercise was stopped. Light exercise resulted in a mean heart rate of 93 beats per minute and a mean heart rate of 152 beats per minute was achieved with heavy exercise (15). Chapman noted in his studies using brachial artery and vein catheterization and Evens Blue dye measurements that the stroke volume increased with increasing levels of exercise, but that the relation is probably not linear. The relative importance of this mechanism he believes to be greatest at transition from rest to mild exercise and at levels of exercise approaching maximum oxygen intake (10). Though these parameters of cardiac output exist in the physically untrained as well as the physically trained, the athletes' circulation has been modified to better adapt to physical stress and therefore responds to a different degree for a given quantity of physical work.

As previously noted, the physically trained possess a slower heart rate and a larger stroke volume at rest than do the physically untrained. The higher oxygen transport capacity in the athlete is explained by this increase in stroke volume and is a prerequisite for a large cardiac putput during exercise and for a large mean peripheral oxygen utilization (6, 12). Bevegård found that exercise in the supine position producing an increase in cardiac output was almost exclusive a result of an increase in heart

rate, and that this was true for athletes as well as nonathletes. He observed only a 9% increase in stroke volume on transition from rest to moderate work in the cyclists from which the measurements were taken which was approximately the same increase observed in the untrained. The cardiac output and oxygen uptake at rest were larger in the athletes than in the nonathletes, and the arteriovenous oxygen difference was found to be only slightly higher in the athletes (about 10 ml/L blood) at rest and during exercise (5, 6). Increase in stroke volume and/or increase in peripheral oxygen utilization can increase oxygen transport capacity, and, not in full agreement with Bevegård's studies, Musshoff et al, found both a larger stroke volume and a higher oxygen utilization to explain the increased oxygen transport seen in athletes when compared to nonathletes. The average arteriovenous oxygen difference between the two groups was observed to be 10 ml/L blood at rest and during light work, if values at corresponding pulse rates are compared. During heavy work at a pulse rate of 170 beats per minute the arteriovenous oxygen difference was 30 ml/L blood. During work the stroke volume increased in both groups but was 15-30 milliliters larger in the group of athletes. However, at rest it was the same for the two groups. With a lower pulse rate the athletes were found to have a cardiac output one liter per minute lower, with an oxygen uptake slightly higher than the ordinary subjects (37). Freedman et al, tested college cross country track members in the trained and untrained condition using the direct Fick method and various levels of exercise on the bicycle

ergometer. It was observed that variations in response to cardiac output and arteriovenous oxygen difference to increased tissue need between different individuals and the same individual on different occasions exceeds the difference existing between trained and untrained athletes (21). Completely sedentary individuals may differ more from untrained athletes than untrained athletes differ from trained athletes, but these findings do imply that there is little fluxuation in circulatory response to stress in athletes who train seasonally. Wang et al, studied the response of cardiac output in sedentary individuals and varsity swimmers to upright graded treadmill exercise using an indicator dilution method. In the sedentary group at rest he found a stroke volume ranging from 21 to 42 cc/M^2, a heart rate ranging from 52 to 75 beats per minute and a cardiac output of 2.6 to 4.2 L/min/M^2. The swimmers at rest showed stroke volumes from 34 to 48 cc/M^2, heart rates ranging from 51 to 65 beats per minute and cardiac outputs of 2.9 to 4.7 L/min/M^2. At submaximum exercise the sedentary group had stroke volumes from 43 to 65 cc/M^2, heart rates from 186 to 192 beats per minute, and cardiac output from 8.3 to 11.8 L/min/M^2. The athletes for the same amount of exercise showed stroke volumes from 52 to 79 cc/M^2, heart rates from 180 to 197 beats per minute, and cardiac output from 9.5 to 14.2 L/min/M^2. Values for stroke volumes during hard exercise did not average over 10% above resting supine values in both groups. In an earlier study using similar techniques Wang observed similar response by trained

and untrained individuals to exercise. He concludes that athletes possess a higher initial stroke volume and lower resting heart rate with a slightly higher cardiac output than do untrained individuals, and that the more marked increase in cardiac output of the athlete during exercise is the result principally of tachycardia (53, 52).

Due to the lack of control in selecting subjects and the various techniques employed to measure cardiac output during exercise it is not too surprising that all the studies are not in full agreement. Being guided by the more recent and more accurate methods of study, I believe it is not unreasonable to conclude that the main difference between athletes and nonathletes is the size of the stroke volume. The physically trained have the advantage with a higher initial or resting stroke volume and a lower resting heart rate, since the pattern of response to exercise in both the trained and the untrained seems to be nearly identicle. That is, the increase in cardiac output is principally produced by an increase in heart rate to about the same level for a given amount of exercise in both groups with most evidence indicating that the athlete has only a slightly increased ability to extract oxygen peripherally from the circulation and neither group shows much over 10% increase in stroke volume during exercise. Thus, the athletes' cardiac output is capable of surpassing the cardiac output of the physically untrained during exercise due to modification of the heart which allows it to deliver more blood volume per given contraction whether the body is at rest or at work.

BLOOD PRESSURE

The introduction of heart catheterization has allowed the developement of the intravascular pressure recording technique. It has been generally found that the arterial and the peripheral venous blood pressures rise while the central venous pressure shows little or no change in the untrained subject during exercise. Riley et al, measured arterial pressure in normal subjects working upright on a bicycle ergometer and found an increase from the resting level of 112/65 (mean pressure of 84) to 153/91 (mean pressure of 123) taken at the highest load (40). Dexter et al, studied the effect of exercise on the normal subject in the supine position and found an increase in the brachial arterial mean pressure in all cases except one (13). Eskildsen et al, using the direct method measured arterial pressure in normal subjects working in the sitting position on the cycle ergometer. He found generally that the pressure increased rapidly during the transition from rest to work, and fell rapidly when the work stopped. An increase in load was followed by an increase of the pressure. As a rule the diastolic pressure followed the systolic pressure, but on a minor scale (18). Fraser and Chapman recorded the arterial pressures of normal subjects working on a treadmill. They found that during exercise the systolic pressure increases, the diastolic pressure falls and the mean pressureremains practically unaltered (20). Donald et al, using normal subjects in the supine position on the bicycle ergometer with direct catheterization found that the average systolic pressure in the bra-

chial artery increased rapidly at the onset of the work, and reached a steady level within one minute except in subjects working at the highest loads where it required two minutes to achieve the steady level. The pressure rise was roughly related to the intensity of the work. The average diastolic pressure increased only slightly at low loads; and the average mean arterial pressure rose during the first five minutes of work, then was maintained during the rest of the working period (15). Cuming noted in his studies that systolic blood pressure shows a gradual increase with increasing work loads for the normal subject. By indirect measurement it was observed that the diastolic pressure decreased, which consequently produced a large increase in pulse pressure (this is contrary to observations by direct measurement as noted) (2). Ellis found an increase in systolic arterial pressure, but insignificant increase in diastolic pressure in the normal subject exercising on the bicycle ergometer (17). Most authors agree that at the transition from rest to work the normal subject experiences an immediate rise in arterial blood pressure and that this rise to a steady state level during work is roughly proportional to the load.

Simultaneous determinations of peripheral and central venous pressures were performed on physically trained subjects by Holmgren (26) and found to vary independently of each other during heavy exercise, but remain in close agreement during the first minute of work. This rise seen in peripheral pressure with prolonged work may be an increase in blood flow through the veins from which

measurements were taken and/or increased venous resistance. These findings help explain the descrepancies between the results obtained with the peripheral and central recordings. McCrea et al, studied venous pressure in normal subjects working on a cycle ergometer measuring indirectly from a vein on the dorsum of the left hand. An increase at the transition from rest to work was seen in every subject. The magnitude varied between 5 and 15 centimeters of water and was proportional to the work load. At the cessation of exercise the pressure fell rapidly to the resting level (20). Schneider and Collins used three indirect methods for the measurement of venous pressure in normal subjects exercising on a bicycle ergometer. The reaction observed fell into two groups; one group established and increased steady level after two to four minutes, and in the other group there was a delayed rise with a constant level not being reached until after ten minutes. This distinction was observed only during light exercise. With heavy exercise the pressure rose in all subjects within one minute. The pressure returned slowly to resting levels after work (taking up to 27 minutes upon cessation of heavy work) (33). Barger et al, measured venous pressure directly from a left forearm vein in normal subjects exercising on a treadmill. The arm was supported at the level of the Xiphoid process and the work load increased to near maximal capicity. It was found that during the period of increasing work the peripheral venous pressure increased in proportion to the load. The increase began at a pulse rate of 100 beats per minute and was

found to average 73 millimeters of water at a mean pulse rate of 181 beats per minute. A linear relationship between pulse rate and peripheral venous pressure was observed to exist (55). It has been shown using direct catetherization that the central venous pressure does not rise at all or is only slightly increased in the normal exercising subject (43, 25, 13, 23, 12). There appears to be reasonable agreement that peripheral venous pressure is increased during exercise and varies directly with the degree of work performed. Also, there apparently is little if any alteration in central venous pressure during exercise.

Studies comparing blood pressure alterations between the physically trained and the physically untrained are limited. Bevegård et al, observed the intracardiac and intravascular differences between cyclists and nonathletes during exercise using direct cardiac catheterization. During heavy exercise with a heart rate close to 160 beats per minute both the intracardiac and the intravascular pressures were considerably higher in the athletes than in the ordinary subjects. The mean brachial artery pressure for athletes was found to be 210/80 millimeters of mercury compared to 160/78 millimeters of mercury for the nonathletes, and the mean right ventricular pressure at this pulse rate for the athletes was 60/4 millimeters of mercury contrasted to a value of 45/0 millimeters of mercury for the nonathletes. However, in relation to the cardiac output the pressure levels were of the same level except for higher ventricular filling pressures in the athletes during exercise. As a consequence of the larger stroke volumes the pulse amplitude in

the brachial artery during exercise was approximately 50 millimeters of mercury greater for the athletes. The diastolic pressure was seen to increase only slightly in both groups from the resting level (6). Thus, at rest intracardiac as well as intravascular pressures are of the same amplitude in both the physically trained and the physically untrained, but during exercise there is a larger increase in these pressures for a given pulse rate in the athlete.

CORONARY CIRCULATION

As previously noted the athletes heart becomes hypertrophied with increase in cardiac vessel caliber up to a point as physical training proceeds (12). This increased capacity at rest as well as during exercise may be the only difference between the cardiac circulation of the athlete and the nonathlete. Lombardo et al, found that normal physically untrained subjects had an average coronary blood flow at rest of 77 cc/100gm/min with an average oxygen consumption of 9.4 cc/100gm/min and an average extraction of 12 volumes percent. This was about 5% of the total cardiac output of these subjects with a relatively high amount of oxygen being extracted. During moderate exercise there was observed to be an increase in coronary blood flow without significant increase in oxygen extraction, indicating that increases in work load are met primarily by increased coronary flow. The rise in coronary flow per 100 grams of left ventricular muscle averaged 45% with an increase cardiac output increase of 63%. A decline in coronary resistance was associ-

iated with this flow increase and an increase in left ventricular work during exercise was observed to be proportionally greater than the oxygen consumption elevation, indicating an increase in cardiac efficiency during exercise (31). Thus, there is evidence that the increased metabolic demands of the heart resulting from the necessary increase in cardiac output to meet the demands of the body during exercise is largly met by increased coronary flow rate. It has been seen that stroke volume increases only slightly with most change being in the heart rate as cardiac output increases, and that this is true for the physically trained as well as the physically untrained. This suggests that coronary circulation is controlled primarily by the increase in heart rate during exercise. Not only is the athlete working with a larger stroke volume to begin with, but the increase to similar levels of heart rate during exercise from a generally slower resting rate permits a potentially larger coronary circulation per given time period. This, however, does not imply that coronary flow in the athlete is greater per muscle mass per time. In fact, one would suspect that if the heart enlarges to greater than 500 grams and outstrips increase in vessel caliber (12), there would actually be less coronary flow per muscle mass. Though no comparitive studies could be found concerning coronary blood flow in athletes and nonathletes; it appears that increased stroke volume and vessel size in the athlete would account for the necessary larger coronary flow for the larger heart of the athlete. There could be found no evidence that exercise causes a larger per-

centage increase in coronary flow in the athlete as compared to the physically untrained.

PULMONARY CIRCULATION

There is an increase in blood flow through the lungs of the normal individual during exercise which partially compensates for the decreased diastolic filling time resulting from increases in heart rate. Sjöstrad observed in his studies that there is neither an increase nor decrease in intrathoracic blood content during work in the normal subject. When cardiac output increases to several times its resting value, there is not a demand for the redistribution of blood from the greater circulation to the heart and lungs - - - rather, the reserve volume of the lesser circulation is called upon to increase the filling rate of the left ventricle. His subjects experienced a threefold increase in lung capillary flow rate during exercise. He concludes that the reserve blood in lungs compensates for occasional variations in supply and output and, due to resultant tension of lung vessel walls, it serves as a source of potential energy which determines the maximum capacity of circulation (45). Dexter et al, tested normal, untrained individuals by direct catheterization and found that there was an increase in pulmonary blood flow (5 to 6 L/min/M^2) without corresponding increases in pressure when oxygen consumption remained below 400 cc/min/M^2 indicating opening of new vascular area and/or further widening of those already present. With identical pulmonary flows it was noted that when oxygen consumption increased to above 400

cc/min/M^2, the pressure did rise when the subject was in the prone position, but not when the upright position was used. This was thought to possibly be due to changes in intrapleural pressure. In the upright position there is an increase in chest volume and the greater stretch of the lungs produced by gravity allows the elastic properties of the lungs to lower the intrapleural pressure. Since intrapleural pressure is the true baseline of vascular pressures in the chest, a given level of pulmonary artery pressure might be unchanged when referred to an arbitrary zero point, but increased when referred to the lower intrapleural base line. This would suggest a slightly under estimated upright pulmonary pressure. Considering this alteration in pulmonary pressure seen with position change during exercise, Dexter observed only a slight rise in mean pulmonary capillary pressure (13). Donald et al, studying normal subjects supine on the bicycle ergometer with cardiac catheterization found that exercise caused an increase in pulmonary artery pressure which varied from subject to subject and appeared unrelated to the severity of the exercise. He also notes that pressures taken with reference to a fixed zero level may not show significant pulmonary pressure change. It was observed that pulmonary artery resistance showed a slight increase during exercise and that the work performed by the right ventricle was seen to increase in a rough direct relation to the degree of exercise (15). Riley et al, noted some minimal increase in work of the right ventricle in the normal subject during exercise, but observed a decrease in mean pulmonary

artery pressure. He explains this decrease in diastolic pressure with unchanged systolic pressure during a cardiac output increase of two to two and one half fold as due to a sharp decrease in pulmonary vascular resistance (40). Johnson et al, observed a rapid increase in pulmonary capillary blood flow with onset of exercise in normal subjects working on a treadmill. The increase reached a plateau after 60 seconds and within five minutes after cessation of exercise the flow rate had returned to resting level (27). Bevegård found that in the supine position the pulmonary arterial wedge pressure increased in athletes from an average of 9.4 millimeters of mercury at rest to 15.9 at moderate work loads and 17.0 millimeters of mercury at more severe work loads. This increase was noted to be more marked than that shown by nonathletes which averaged from a resting value of 9.4 millimeters of mercury to 10.5 during moderate exercise and 11.0 millimeters of mercury at the most severe degree of exercise. This difference between athlete and nonathlete is explained as the result of larger central blood volume possessed by the athlete, which may represent a higher potential energy that helps maintain the large stroke volume with short effective filling time at high heart rates. The pressure in the pulmonary artery at rest was within the same range as in ordinary subjects, but during exercise this pressure was higher in the athletes in proportion to the higher arterial wedge pressures. The pressure gradient over the pulmonary vascular bed in relation to cardiac output was approximately the same in both the group of athletes and nonath-

letic group (6). The more recent studies of pulmonary flow during exercise agree that blood flow through the lungs is increased during work in the normal subject with increases in pulmonary artery pressure (Riley (27) disagrees with this) that vary with body position, but would probably be more constant if an arbitrary zero base line were used. There is little, if any, difference in pulmonary blood pressure during rest between the physically trained and the physically untrained, but during exercise it has been observed that the pulmonary artery pressure increases in the athlete. The pressure gradient over the pulmonary vascular bed is apparantly the same in both groups during exercise indicating that blood is reaching the athletes' left atrium under higher pressure than it is in the nonathlete which helps explain the maintainence of the increased stroke volume at high heart rates.

VISCERAL CIRCULATION

As muscles are called upon to perform more work the increase in blood flow required is met in part by the increase in cardiac output and in part by a decrease in blood flow and volume to the viscera which is not in such need of an increase in circulation. Braunwald and Kelly using indicator dilution technique observed an increase in central blood volume (described as both sides of the heart, the lungs, and a significant fraction of the large arteries) in normal subjects exercising in the supine position on a bicycle ergometer. There was an average increase of 285 milliliters during

exercise which was explained by a shift of blood out of the splanchnic vasculature (9). Wade et al, determined splanchnic blood flow using BSP clearance and extraction and splanchnic blood volume by following the tracer concentration of arterial and hepatic venous blood after injection of I^{131} - labelled human serum albumin into a peripheral vein. Normal, untrained subjects performing light exercise were used. Splanchnic flow was seen to decrease by 250-450 milliliters per minute during exercise and return to resting levels by 18 to 22 minutes after cessation of exercise. At rest the oxygen uptake was observed to be 20 to 34% of total uptake and during exercise was only 7 to 11% of the total oxygen uptake. The blood volume showed a decrease of 300 to 700 milliliters with only a slight hematocrit alteration indicating that total blood volume remained nearly the same during exercise. The decreased splanchnic volume was explained as due to more vigorous respiratory movements of the diaphram and contraction of abdominal musculature causing an increased intra-abdominal pressure with consequent expression of blood from abdominal vessels (49). Bradley used sodium PAH clearance to study renal and liver blood flow respectively in normal subjects exercising in the supine position on a bicycle ergometer. At a cardiac output two times resting level he observed a sharp fall in both hepatic and renal blood flow with a larger decrease in hepatic flow (8). Barclay, et al, measured the renal plasma flow (Diadone method) in normal, untrained subjects who had just run 440 yards at maximum speed. It was noted that plasma flow decreased to 18 to 54% of the

initial testing flow remained below resting flow value 10 to 40 minutes after cessation of exercise (2). Merrill and Cargill measured renal plasma flow in normal subjects during exercise (stepping up and down 12½ inch steps) via sodium PAH. They found that two thirds of their subjects showed an appreciable decrease in renal plasma flow and concluded that mild exercise is not likely to cause a decrease in renal plasma flow, but that severe exercise may cause renal shutdown even in normal subjects (36). There is good agreement, then, that during exercise the visceral circulation is decreased which may be important in augmenting venous return to the heart and permitting more rapid adjustment in cardiac output and increased muscle blood flow. Though these studies have all been on the untrained subject there is little reason to assume that the athletes' visceral circulation responds differently to exercise. Perhaps, due to the already increased total blood volume and increased efficiency in cardiac output, it may require more severe exercise to produce equal visceral flow and volume changes.

CUTANEOUS CIRCULATION

Most investigators have found that during the first few minutes of exercise skin circulation is decreased, but with prolonged work again rises to above resting flow. Fox and Edholm observed cutaneous circulation by means of heat measurement, nerve block, and venous occlusion plethysmography. They found that cutaneous vessels in the hands, lips, and pinna of the ears are con-

trolled by vasoconstrictor nerves; and that blood flow in the skin of the forearm, calf, thigh, upper arm, cheeks, forehead, chin, neck and trunk is regulated mainly by a vasodilator mechanism. They believe that the bradykinin producing enzyme contained in sweat may be the vasodilator, but the final proof or disproof of this hypothesis will be available only when a block to the bradykinin mechanism is found (19). Barger et al, measured the reactive hyperemia of the middle volar forearm surface of the normal, untrained subject working on a treadmill. They noted that the appearance and persistance of cutaneous hyperemia (vessel tone) usually remained within control limits until heavy work was performed. With heavy exercise the skin vessel tone was observed to increase. They concluded that conditions producing an increase in cutaneous vessel tone are cumulative in nature, but are not significiant until exhaustion is approached (4). Bishop et al, measured blood flow alterations in the resting arm during leg exercise and noted an initial increase in axillary artiovenus oxygen difference followed after variable time by a decrease in arteriovenus oxygen difference indicating a reduction of deduced total arm blood flow during early exercise. Hand calorimetry revealed a reduction of heat elimination in the early minutes of exercise which was followed by an increase in heat elimination. The rate of clearance from forearms of injected radioactive sodium was not significiantly altered between rest and exercise. They, therefore, concluded that blood flow to the resting arm during leg exercise is initally decreased but remains so for only a few minutes. and that this decrease occurs principally in the cutaneous circu-

lation (7). Christensen et al, measured cutaneous blood flow in normal exercising (bicycle ergometer) subjects via plethysmography and thermocouple temperature measurements. At the beginning of heavy work an instantaneous and considerable decrease in skin circulation occured which remained decreased for several minutes. Within six minutes the flow had increased to above resting levels. They believe that the vasoconstriction seen initially during exercise is of nervous origin - either cortical impulses or reflex influence from the working muscle (11). Lowenthal et al, observed an increase in cutaneous blood flow during exercise by measuring the hand and fingers with the venous occlusion plethysmograph. No timed measurements were reported and it is assumed that this is an overall measurement or else taken sometime after the initial decreased flow had occured as noted by other investigators (32). Only the findings of Barger et al, (4) are not in agreement with an initial decrease in cutaneous circulation upon initiating exercise which is followed by an increase in blood flow as exercise is prolonged. This might possibly be due to using the forearm for the study of cutaneous circulation rather than the hand as was done in most other investigations (remembering that Fox and Edholm (19) found vasoconstriction control predominent in the hand, but not in the forearm). One might explain the initial decrease in cutaneous flow as a mechanism to aid flow and volume increase to the muscles, then as the body temperature increases and the cooling mechanism of vasodilation is needed, blood flow through the skin increases to above resting levels.

No comparison studies could be found with athletes, but, again, there is little reason to believe a similar cutaneous blood flow response to exercise would not be expected in the physically trained.

MUSCLE CIRCULATION

There is an increase in blood flow through active muscle in both the physically trained and physically untrained. With a given degree of exercise this blood flow increase is larger in the physically trained. Roddie and Shepherd have found that skeletal muscle vessels are supplied with both vasoconstrictor and vasodialator nerve fibers. Many stimuli (mainly through baroreceptor reflexes originating inside the chest cavity) modify the constrictors, but the dilators are activated only by emotional stress. When at rest the muscle vessels are under considerable constrictor tone (41). Dornhorst observed in his studies that blood flow through the exercising calf and forearm increases and nearly always increases further immediately upon cessation of exercise. It appears that contracting muscle hinders blood flow and the extent of hindrence varies with the degree of contraction and the muscle being tested (flow can actually be arrested by maximum calf contraction). The decrease of post-exercise hyperemia is very nearly exponential for periods of exercise up to about five minutes --- after longer period of time an additional longer period become apparant. Post-exercise dilation is strikingly independent of the actual flow achieved or permitted implying a lack of highly diffusable substance producing the dila-

tion. It was also found that local oxygen tension is not critical. Bradykinin is perhaps the most plausable dilating material at present, but as previously stated a blocking agent is needed to prove this (16). In agreement with this post-exercise vessel dilation are the findings of Sharpey-Schafer who observed a flow increase of 5/20 fold upon cessation of exercise. It was also noted that with this arteriolar dilation there occured a venous constriction after exercise (44). Wisham et al, determined blood flow in the gastroenemius and biceps of normal untrained subject with radioactive sodium. The clearance rate of the radioactive sodium was significantly increased by moderate exercise, and fatiguing exercise markedly increased the clearance rate which was maintained 8 to 10 minutes following cessation of the exercise (54). Grant measured changes in forearm volume of normal exercising subjects and found that with a single sustained contraction limb volume decreased during the first three to five seconds after which the volume increased to a maximum by 30 seconds. When the grasp was released there was an immediate further small rise in volume followed by a subsidence to the resting volume. He thus concludes that while strong muscle volume does compress vessels, it is not enough to prevent dilation to a certain extent and increase in blood flow during exercise (22). Rohter et al, compared six swimmers in training with six normal nonathletes with the plethysmograph during exercise. A significant larger increase in the muscle flow was observed in the athlete during exercise when compared to the nonathlete, but resting flows were essentially the same in both groups. Thus, physical training may make more capilliaries avail-

able which would enhance performance by assuring increased oxygen supply and more efficient removal of metabolites. On the other hand, the increase muscle flow seen in athletes may be due to increased flowthrough non-nutrient arteriovenus anastomoses. The exact function of the arteriovenus anastomoses is not clear, but they may increase efficiency and faciliate muscle contraction (42). Wakim, et al, concludes from their investigation that repeatedly activated muscles develop an efficient blood supply either by the development of new capillaries or the opening up of hitherto unused ones (50). Thus, there is good agreement that blood flow through active muscle is increased and that trained muscle has the facility of increasing this flow still further from the resting level.

SUMMARY

The various hemodynamic alterations occuring in man during exercise have been reviewed and comparisons made between the physically untrained and the physically trained where information permitted. Due to the lack of a basic set of criteria common to all studies reviewed regarding age, sex, methods and techniques of measurement and degree of physical fitness; only general conclusions can be drawn with reference to the results of the various investigations. There was general agreement that exercise causes an increase in heart rate, a slight increase in stroke volume, an increase in oxygen uptake, and an increase in arteriovenus oxygen difference in both the trained athlete and the nonathlete. The athlete possesses a larger cardiac

output due mainly to a larger stroke volume, but as in the nonathlete the increase in output during exercise is principally due to an increase in heart rate. It has also been observed that the athletes' ability to extract oxygen peripherally is slightly greater than the nonathletes' ability. Arterial blood pressure and peripheral venous pressure increases with exercise and varies with the severity of work in both groups, while the central venous pressure shows little if any change. This increase in blood pressure (intravascular and intracardiac) is larger in the athlete for a given work load. Both groups show an increase in coronary blood flow during exercise which is primarily controlled by the increase in heart rate. No evidence could be found indicating that there is a larger percentage increase in coronary blood flow in the athlete, though with a larger heart and larger caliber of the coronary vessels greater blood volume does flow through the muscle of the athletes' heart. Blood flow through the lungs is increased and the pulmonary artery pressure increased during exercise in both the physically trained and the physically untrained. The increase in pulmonary artery pressure is higher in the athlete for a given amount of work, but the pressure gradient over the pulmonary vascular bed is the same in the two groups. This increased blood pressure to the left antrim in the athlete aids in maintaining the increased stroke volume at high heart rates. Blood flow through the viscera is decreased in the normal nonathlete during exercise which may augment venous return to the heart. No studies on visceral flow in the physically trained were found. There is an in-

itial decrease in cutaneous blood flow during exercise followed by an increase in flow to above resting levels as exercise is continued in the nonathlete. Again, no specific investigations using athletes were found. Blood flow through active muscle increases both in the physically trained and the physically untrained. This increase in flow is seen to be greater in the athlete. It is thus seen that physical training stimulates the development of a more efficient circulatory system allowing the trained individual to perform greater amounts of work for longer periods of time than the untrained individual.

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