16. Exercise

The performance of muscular exercise not only throws a strain on the musculoskeletal system itself but it also tests the reserves of virtually every system in the body. Exercising muscles have to be supplied with much greater quantities of fuel and oxygen than muscles at rest, larger quantities of waste products have to be removed and a corresponding amount of heat has to be dissipated. Thus the respiratory, cardiovascular, alimentary, renal and endocrine system are all clearly involved, as is also the skin.

16.1 Energetics

The energy for muscular work is derived from the hydrolysis of adenosine triphosphate (ATP). As stores are limited, exercise demands an increased supply from *aerobic* and *anaerobic metabolism.* The contribution from these two sources vary greatly. Anaerobic metabolism only contributes up to 20% of the total energy utilized during brief explosive exercise. This contribution increases to about 33% for events such as a 400 m race lasting under a minute and reaches 90% in events lasting 10 min or more. Anaerobic metabolism can supply ATP very rapidly at the onset of exercise. It is, however, less efficient than aerobic metabolism and there are limits to the amounts which can be supplied in this way. Aerobic metabolism is limited by the delivery of oxygen to the tissues but if that does not fail it can continue virtually indefinitely.

In muscle metabolism the fatty acids provide the major substrate for energy production in red skeletal muscle and cardiac muscle. Glycogen is less important for energy metabolism in red muscle but plays a more important role in white muscle.

The *efficiency* of the conversion of metabolic energy into external work is of the order of 20%. This means, for example, that someone on a bicycle ergometer doing external work at the rate of 200 J/s (200 W) is also generating in the same time 800 J as thermal energy which must be dissipated. During steady exercise body temperature will rise and stabilize between 40 °C and 41 °C at which temperature the maximum energy output is considerably above that attainable at resting temperatures of 36-37 °C.

The external work of exercise may be very difficult to estimate. In running on the level most of this work is due to the acceleration and deceleration of the limbs, viscous work on the joints and muscles and, especially in the untrained, the continual raising of the centre of gravity. At the other extreme, a climber making a slow vertical ascent is performing work, most of which is easily calculable, in lifting his body weight metre by metre.

The exercise physiologist in estimating efficiency is usually content to measure the O_2 consumption of a subject in conditions where the actual external work load (expressed in watts) can be estimated reasonably accurately. It is assumed that either aerobic metabolism is the sole source of energy or that the anaerobic contribution is balanced by reoxidation of the products of that metabolic pathway. The efficiency is calculated after converting the O_2 cost of exercise measured in mL/s into units of watts. On a mixed diet 1 mL (STPD) of oxygen yields 20.2 J of energy. Hence 1 mL (STPD) of O_2 consumed per second yields 20.2 W, i.e.:

% efficiency = (rate of external work x 100)/(O_2 cost of exercise x 20.2).

If exercise is started abruptly, maintained at a constant level and then finished abruptly, the O_2 uptake lags behind the energy output. When a uniform rate of O_2 uptake has been reached, it may be assumed that the O_2 requirement and O_2 uptake are in balance. The deficit in O_2 uptake over the first few minutes of exercise compared with requirement is known as the *oxygen debt.* After exercise has stopped the O_2 uptake remains elevated for a considerable period even though the immediate O_2 requirement of the individual may have returned to basal levels. This repayment is theoretically identical with the oxygen debt, expressed in litres of O_2, but the precision of measurement of basal and post-exercise O_2 uptakes must be very exact for this to be demonstrated. Inasmuch as some lactate may be excreted rather than reoxidized there may be a discrepancy between the debt and repayment. On the other hand, the increased body temperatures at the completion of severe exercise may demand a greater rate of O_2 uptake than the debt would theoretically indicate.

If the steady-state maximum O_2 uptake of the individual cannot meet the O_2 requirement then the oxygen debt will progressively increase in proportion to the severity of the external work load. There is an upper limit to the O_2 debt (8-18 L) that can be incurred and, that reached, exhaustion will quickly terminate the exercise. In practice, maximal O_2 consumption can only be measured under conditions causing exhaustion and considerable motivation of the subject is necessary.

16.2 Physicochemical Changes in Muscle

If the metabolism of muscle cells increases (potentially twenty-fold) then the state of the cells and their environs will alter. The heat output will be proportional to the increased metabolic rate so that temperature will rise locally, the P_{O2} will fall as its utilization increases, the P_{CO2} will rise as its production increases, and acidity will increase due to the rising P_{CO2} and to accumulation of lactic and other acids.

These changes, together with some ionic changes such as an increase of K+ and perhaps of other metabolic products, will bring about considerable vasodilatation of the arterioles in the muscle. This fall in arteriolar resistance increases the flow through the capillary beds and results in three important changes:

(i) The increased blood flow makes more O_2 available to the muscles. The amount of O_2 extracted from each mL of blood cannot rise more than two- or three-fold so that the vast proportion of the twenty-fold increase in O_2 consumption is made possible by the increased blood flow.

(ii) The number of capillaries open and carrying blood increases enormously, therefore the velocity of flow in the capillaries increases only slightly. The estimated eighty-fold increase in the number of open capillaries in severe exercise will result in the mean distance between cell and capillaries falling by a factor of nine. This increases proportionately the gradient of P_{O2} on which the O_2 flux from blood cells depends. Furthermore the off-loading of O_2 is aided by the high P_{CO2}, high temperature and increased acidity, all of which move the oxyhaemoglobin dissociation curve to the right.

(iii) In exercise employing most of the skeletal musculature of the body a very large proportion of the cardiac output may be pouring through the muscle beds, up to 20-25 L/min. This necessitates very large changes in cardiovascular activity to preserve the mean systolic arterial pressure within acceptable limits.

16.3 Circulatory Responses in Exercise

These include increases in (i) heart rate, stroke volume and cardiac output, (ii) changes in blood pressure and (iii) redistribution of blood flow.

(i) *The heart rate* may rise, predominantly by shortening of diastole, to around 200 beats per minute. At rates higher than this the diastolic filling time is too short to allow any further increase in cardiac output. It follows, therefore, that an individual with a resting heart rate of 90/min can only double his heart rate whereas another with a rate of 60/min can triple this. At the onset of light exercise nearly all the increased *cardiac output* is due to an increase in heart rate with little change in *stroke volume*, though this balance may alter as exercise proceeds. As the intensity of exercise increases up to about half the maximum of which an individual is capable, stroke volume increases substantially. Beyond this increases in cardiac output up to a maximum are almost entirely due to further increases in heart rate.

(ii) The changes in *blood pressure* during exercise depend on age, the type and severity of the exercise and on training. However exercise increases cardiac output and hence systolic pressure but there are two opposing influences on diastolic pressure. The diminution in peripheral resistance speeds the rate of fall in arterial pressure as the blood leaves the major arteries for the muscle beds, whereas the increased heart rate shortens the time during which this occurs in diastole. In the young subject this will lead to a small fall in diastolic pressure, a larger rise in systolic pressure and hence an increase in pulse pressure and also in mean arterial pressure. With age the rise in cardiac output tends to cause a progressively greater rise in mean pressure, particularly in the unfit.

In athletes in their late twenties undergoing moderate to severe exercise (about 3 L/min) the diastolic pressures may remain unchanged at about 70 mmHg, the mean pressure rising from 100 o 120 mmHg. During extensive exercise the systolic pressures may increase to as high as 200 mmHg. In contrast in veteran athletes blood pressures in excess of 300 mmHg have been recorded during moderate exercise on bicycle ergometers.

(iii) The *redistribution of blood flow* during exercise. The brain flow in unaltered, coronary blood flow increases with the cardiac output, the renal and splanchnic flow are reduced with exercise and the skin blood flow is influenced by the need to dissipate heat.

The cardiovascular changes occurring in exercise are under reflex control. The initial vasodilatation in the muscles (not in itself a reflex response) will result in a fall in total peripheral resistance (TPR) and hence a fall in mean arterial blood pressure (MAP). This will be detected by the arterial baroreceptors and lead to a reflex increase in heart rate, to vasoconstriction in other body tissues (except in skeletal muscles, heart and brain) and to venoconstriction. Limb proprioceptor reflexes from he exercising limbs also cause increases in cardiac output, TPR and MAP. Furthermore the increase in ventilation that accompanies

exercise simulates the lung stretch receptors to increase reflexly heart rate and decrease TPR. The latter response will partially counteract the other vasoconstricting reflexes.

Many of these cardiovascular changes occur in anticipation of the exercise, initiated by the higher centres in the CNS and mediated by intense sympathetic activity and adrenaline secretion.

Changes in Blood Flow Distribution During Exercise

Tissue	Rest ml/min (%)	Light-Medium mL/min-mL/min	Heavy mL/min (%)
Cerebral	750 (13.0)	750-750	750 (3.0)
Coronary	250 (4.5)	350-650	1000 (4.0)
Renal	1100 (19.0)	900-600	250 (1.0)
Splanchnic	1400 (24.0)	1100-600	300 (1.2)
Skin	500 (8.5)	1500-1800	600 (2.4)
Others	600 (10.5)	400-300	100 (0.4)
Sk. muscle	1200 (20.5)	4500-10800	22000 (88.0)
Cardiac output	5800 (100.0)	9500-15500	25000 (100.)

16.4 Respiratory Responses in Exercise

Pulmonary ventilation increases with light to moderate exercise in proportion to the work being performed and the increased demand for the supply of O_2 and the excretion of CO_2. This increase involves increases both in frequency and tidal volume although frequency only increases to any extent in more strenuous exercise. At the onset of exercise there is an immediate increase in pulmonary ventilation and a similar, usually larger, fall at the end of exercise.

The abrupt changes must result from nervous influences, because like the circulatory responses, their onset may precede the onset of the exercise and their rapid reversal occurs ahead of changes in temperature or blood chemistry. The following nervous influences have been suggested: (i) a neural drive from higher centres to the respiratory centre; (ii) a learned response which anticipates the drive to increase pulmonary ventilation; and (iii) an input from receptors in the limbs.

At higher work rates pulmonary ventilation increases disproportionately, presumably because of the increased body temperature and increased acidity from anaerobic metabolism and the respiratory exchange ratio increases. This increase in the respiratory exchange ratio should be interpreted largely as an increased ventilatory response which washes out of the body considerable amounts of CO_2, rather than as the result of production of CO_2 by anaerobic glycolysis. Although the term 'anaerobic threshold' is used to describe the level at which this hyperventilation becomes apparent, there is in fact a continuous increase in lactate and also an increase in body temperature (as skin blood flow is reduced relatively a higher work rates) which contribute to this phenomenon. Indeed a subject at rest heated to temperatures commonplace in exercise will hyperventilate to very low CO_2 levels. This often results in tetany.

16.5 Fitness

The term fitness implies a state in which all organic dysfunction has been eliminated including that arising from disuse.

Measurement of VO_2max is an objective test which, with care, can be reasonably informative. It seems that the fairly general assumption, that an athlete is born with a personal potential to achieve a higher power output than the majority of people if sufficiently trained, is a valid one and that training is a minor determinant of the VO_2max, at least in comparing the fit with the super-fit.

Although any step of the oxygen transport system can in particular disease states limit VO_2, it is probable that cardiac output is the most likely limiting factor in the performance of individuals without overt malfunction or disease. This evidence of diminished ability to increase cardiac output might suggest a lack of fitness. Measurement of blood pressure and the recording of heart rate after exercise (during which the heart rate exceeded 160/min) are fairly simple initial tests. The heart rate in reasonably fit individuals should return halfway towards its resting rate in 1-1.5 min. For athletes in training the corresponding time would be nearer 45 s. A blood pressure above the average for age and sex would be a cause of concern, particularly if the pulse pressure were less than 40 mmHg or the diastolic pressure in exercise were elevated.

Training. The effects of training are not easy to measure with precision and the matter has generated as much argument as has the concept of fitness. It is, however, agreed that at a cellular level in the muscle there is an increase in myoglobin, mitochondria, enzyme activity and capillary density, changes which most obviously promote aerobic metabolism. Muscles themselves, including the myocardium, hypertrophy and cardiac volume increases. The observed effects of these changes are a reduced heart rate at rest and at submaximal work rates metabolic usage of fat rather than carbohydrate with a slower build-up of lactate concentrations.