CHAPTER 1

Exercise Limitations

It would be futile to accomplish with a greater number of things what can be accomplished with fewer.
-William of Ockham

1.1 INTRODUCTION

The study of exercise is important to the bioengineer. To understand exercise responses is to understand physiological responses to natural stresses to which the body has become attuned. This understanding can be used to facilitate communication with physiologists, veterinarians, occupational hygienists, or medical personnel on multidisciplinary research, development, or management teams. Familiarity with exercise physiology may be a requirement for the proper design decisions when developing a new bioengineering product. Bioengineers, especially those who have accumulated some experience and reputation in their field, are often requested to evaluate research or management proposals or design reports from their subordinates. A basic understanding of exercise physiology can be an invaluable aid toward making the proper evaluation. Furthermore, there is something to be said for the individual who seeks knowledge of the surrounding world for the sake of global understanding and self-actualization. This is the type of individual who would relish the opportunity to study the material with which this book is filled, and this is the type of individual who will see new ways to describe and formulate physiological information.

Like many exercise physiology texts, this book must deal with a broad scope of material. After all, exercise responses are both all-consuming and highly integrated: most physiological systems, artificially divided and separately studied, become one total supportive mechanism for the performance of the physical stress of exercise.

Unlike many exercise physiology texts, the emphasis here is on quantitative description as much as possible. This means that the book is not intended to be a physiology primer; others will have to be used for introductory purposes. This book is intended to demonstrate the vast amount of physiological material that can be quantitatively predicted. For this reason, some physiological facts are not included here, but the hope is that the equations, models, and tables of numerical values will make up for any omission.

Models play an important part in the engineering world. As Grodins (1981) states:

[Models] ... clarify our thinking about a problem by explicitly identifying and clearly stating every assumption and limitation and ... set the stage for a rigorous analysis usually expressed in mathematical language.... They provide a compact, clear, rigorously integrated summary of current conventional wisdom about how some natural system works.... Textbooks in the biological sciences are often swollen with detailed verbal descriptions which do not depart very far from raw

1This statement, known as Ockham's Razor, or the Principle of Parsimony, is the basis for selecting the simplest possible model to describe a process.
EXERCISE INTENSITY AND DURATION

Generally, intense exercise can be performed for short times only. The intensity-duration curve for any particular individual plots generally as a hyperbola asymptotically approaching each axis (Figure 1.2.1). Although Figure 1.2.1 was used to describe exercise limitations imposed by respiratory protective masks, the general shape is still valid for exercise of various intensities; it shows that for very high rates of work, very short performance times can be expected.

In an interesting summary article, Riegel (1981) compared world-class athletic performance records for running, race walking, cross-country skiing, roller and speed skating, cycling, freestyle swimming, and manpowered flight. He plotted time against distance on logarithmic scales and found a linear relationship between the times of 210 and 13,800 sec (Figure 1.2.2). Below 180-240 sec, athletic competition includes sprints and other activity involving transient body processes. Above 13,800 sec, competition is rarely, if ever, carried to the limit of endurance. Thus over the linear range of 210-13,800 sec (3.5-230 min), performance time is predicted by the following equation:

\[ t = ax^b \]  

(1.2.1)

where  
- \( t \) = endurance time, sec
- \( a \) = constant, sec/km\(^b\)
- \( x \) = distance, km
- \( b \) = fatigue factor, dimensionless

The constant \( a \) is dependent on the units of measurement and has no particular significance. The exponent \( b \) determines the rate at which average speed decreases with distance. Values

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\(^2\)Cobelli et al. (1984) state that "the principal difficulty attached to the mathematical analysis of physiological and medical systems stems from the mismatch between the complexity of the processes in question and the limited data available from such systems."
EXERCISE INTENSITY AND DURATION

Figure 1.2.1  Schematic representation of performance time while exercising wearing a protective mask. (Adapted and redrawn with permission from Johnson and Cummings, 1975.)

TABLE 1.2.1  Specific Constants and Data for the Endurance Equation

<table>
<thead>
<tr>
<th>Activity</th>
<th>a^sec/km^b (min/km^b)</th>
<th>b^a</th>
<th>Distance Range, km</th>
<th>Time Range, sec</th>
<th>(min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Running, men</td>
<td>137.9 (2.299)</td>
<td>1.07732</td>
<td>1.5–42.2</td>
<td>210–7,740 (3.5–129)</td>
<td></td>
</tr>
<tr>
<td>Running, men over 40</td>
<td>154.1 (2.569)</td>
<td>1.05352</td>
<td>1.5–42.2</td>
<td>234–7,860 (3.9–131)</td>
<td></td>
</tr>
<tr>
<td>Running, men over 50</td>
<td>170.5 (2.841)</td>
<td>1.05374</td>
<td>1.5–42.2</td>
<td>252–8,700 (4.2–145)</td>
<td></td>
</tr>
<tr>
<td>Running, men over 60</td>
<td>192.2 (3.204)</td>
<td>1.05603</td>
<td>1.5–42.2</td>
<td>294–10,100 (4.9–168)</td>
<td></td>
</tr>
<tr>
<td>Running, men over 70</td>
<td>219.2 (3.654)</td>
<td>1.06370</td>
<td>1.5–42.2</td>
<td>324–11,300 (5.4–189)</td>
<td></td>
</tr>
<tr>
<td>Running, women</td>
<td>155.9 (2.598)</td>
<td>1.08283</td>
<td>1.5–42.2</td>
<td>234–8,820 (3.9–147)</td>
<td></td>
</tr>
<tr>
<td>Swimming, men</td>
<td>596.2 (9.936)</td>
<td>1.02977</td>
<td>0.4–1.5</td>
<td>234–900 (3.9–15)</td>
<td></td>
</tr>
<tr>
<td>Swimming, women</td>
<td>634.7 (10.578)</td>
<td>1.03256</td>
<td>0.4–1.5</td>
<td>246–960 (4.1–16)</td>
<td></td>
</tr>
<tr>
<td>Nordic skiing, men</td>
<td>170.2 (2.836)</td>
<td>1.01421</td>
<td>15–50</td>
<td>2,640–6,940 (44–149)</td>
<td></td>
</tr>
<tr>
<td>Race walking, men</td>
<td>213.9 (3.565)</td>
<td>1.05379</td>
<td>1.6–50</td>
<td>354–13,300 (5.9–222)</td>
<td></td>
</tr>
<tr>
<td>Roller skating, men</td>
<td>95.3 (1.589)</td>
<td>1.13709</td>
<td>3–10</td>
<td>336–1,320 (5.6–22)</td>
<td></td>
</tr>
<tr>
<td>Cycling, men</td>
<td>60.9 (1.015)</td>
<td>1.04834</td>
<td>4–100</td>
<td>264–7,680 (4.4–128)</td>
<td></td>
</tr>
<tr>
<td>Speed skating men</td>
<td>76.0 (1.266)</td>
<td>1.06017</td>
<td>3–10</td>
<td>246–900 (4.1–15)</td>
<td></td>
</tr>
<tr>
<td>Man-powered flight</td>
<td>194.3 (3.238)</td>
<td>1.10189</td>
<td>1.8–36.2</td>
<td>384–10,100 (6.4–169)</td>
<td></td>
</tr>
</tbody>
</table>

Source: Adapted and used with permission from Riegel, 1981.

^Based on records up to November 1, 1979.

for these constants, obtained by a least-squares analysis, are found in Table 1.2.1. World-class runners, men and women, have an identical fatigue factor of 1.08; men and women swimmers share a fatigue factor of 1.03.

^This term refers to a standard procedure in statistical regression where the constants are determined such that they minimize the sum of the squares of deviations of the individual data points from the line fitted through them.
Manipulating the endurance equation gives, for average speed,

\[ s = \frac{a^{1-b}}{a} \]  

(1.2.2)

where \( s \) = speed, km/sec

These speeds, seen in Figure 1.2.3, are instructive for characterizing individual sports. In cycling, aerodynamic drag is the dominant form of resistance, and cyclists often line up one behind the other, with the lead cyclist breaking the wind for the rest. Speed skaters also operate at high speeds, with their inherent drag, and must also negotiate many turns. Runners are affected by the large forces they must develop or absorb as they overcome the inertia from rapid limb movement. Their bodily centers of gravity rise and fall with each step. Race walkers are not jolted with each step, as runners are, but their body motions must be more contorted and require great stretching effort and use of more of their total musculature. Swimmers compete in a medium that is relatively viscous, which limits their speeds considerably.

Men and women swim and run at the same distances in world-class events. In swimming, women attain speeds of about 94% of those of a man. In running, women achieve about 88% of the speed of men.
Figure 1.2.3 Speed decreases as distance increases for all world-class activities. Shown here is the average speed from the endurance equation. (Redrawn with permission from Riegel, 1981.)

Figure 1.2.4 Comparison of running records for men and women of different ages. Runners provide the greatest amount of data for performance comparison. (Redrawn with permission from Riegel, 1981.)
When comparing running records, age can be seen to decrease average attainable speed (Figure 1.2.4). A septuagenarian can run 70% as fast as a world-class man. The difference with age appears to be greater for the shorter running distances than it does for longer distances. At longer distances, the speed of the fastest 40-year-old is nearly the same as that of a world-class man. It is unclear how much of this is due to relative short-term endurance loss or due to different training or competitive factors with older men. Certainly, older men who hold other jobs cannot spend full time training, nor are they subject to the highest acclaim when winning a race.

Returning to Figure 1.2.1, there are several dashed-line hyperbolas that appear in the plot. This figure suggests that several factors can limit exercise performance. Those shown to be important while exercising wearing a mask are cardiovascular, respiratory, thermal, and long-term effects. Although each of these can contribute to the exercise performance limitation, it is the factor determining shortest time at any particular steady work rate which is the limiting factor in exercise performance. The overall work rate performance time characteristic is the locus of points formed from the individual stress limitations. Approximate time and work rate data have been obtained from published reports, and supporting experimental data appear in Table 1.2.2.

The conceptual framework appearing in Figure 1.2.1 is relative only. Normal individuals not wearing masks probably will not experience a respiratory limitation to exercise. Imposition of heavy clothing may move the thermal stress limitation curve to the left from its position in Figure 1.2.1 such that it dominates the whole figure.

The implications of this hypothetical intensity-duration concept are many. First the model implies that the various types of stress can be studied independently from one another at appropriate levels of work. Second, any interactions between stress, if they occur, would be found at work rates and performance times where two component stress limitation curves

### Table 1.2.2 Subject Data at Their Voluntary End Points for Different Rates of Work

<table>
<thead>
<tr>
<th>Work Rate, N·m/sec</th>
<th>Performance Time, sec</th>
<th>Final Heart Rate, beats/sec (beats/min)</th>
<th>Final Exhalation Time, sec</th>
<th>Final Rectal Temperature, °C</th>
</tr>
</thead>
<tbody>
<tr>
<td>150</td>
<td>4260 (71.0)</td>
<td>2.93 (176)</td>
<td>0.96</td>
<td>38.83^a</td>
</tr>
<tr>
<td>175</td>
<td>3430 (57.2)</td>
<td>2.83 (170)</td>
<td>0.91</td>
<td>38.50^a</td>
</tr>
<tr>
<td>200</td>
<td>2400 (40.0)</td>
<td>2.93 (176)</td>
<td>0.79</td>
<td>38.66^a</td>
</tr>
<tr>
<td>225</td>
<td>2110 (35.2)</td>
<td>3.13 (188)</td>
<td>0.70</td>
<td>39.00^a</td>
</tr>
<tr>
<td>275</td>
<td>438 (7.3)</td>
<td>2.98 (179)</td>
<td>0.55^a</td>
<td>38.03</td>
</tr>
<tr>
<td>300</td>
<td>240 (4.0)</td>
<td>3.00 (180)</td>
<td>0.50^a</td>
<td>37.50</td>
</tr>
<tr>
<td>325</td>
<td>204 (3.4)</td>
<td>2.93 (176)</td>
<td>0.49^a</td>
<td>37.50</td>
</tr>
<tr>
<td>350</td>
<td>150 (2.5)</td>
<td>3.05 (183)</td>
<td>0.55^a</td>
<td>37.39</td>
</tr>
<tr>
<td>375</td>
<td>144 (2.4)</td>
<td>3.00 (180)</td>
<td>0.50^a</td>
<td>37.39</td>
</tr>
<tr>
<td>400</td>
<td>120 (2.0)</td>
<td>2.93 (176)</td>
<td>0.50^a</td>
<td>37.61</td>
</tr>
</tbody>
</table>

*Subject A*

| 200                | 3660 (61.0)           | 2.68 (161)                             | 0.842                      | 38.83                       |
| 250                | 1560 (26.0)           | 2.53 (152)                             | 0.913                      | 37.89                       |
| 350                | 420 (7.0)             | 2.72 (163)                             | 0.560^a                    | 37.36                       |
| 400                | 240 (4.0)             | 2.65 (159)                             | 0.544^a                    | 36.83                       |

*Source:* Adapted and used with permission from Johnson, 1976.

^aDenotes probable limiting measurement.
Figure 1.2.5 Differences in rectal temperature of cats with carotid bodies intact when breathing air and carbon dioxide. Reference for the comparison was temperature at the end of the 1200 sec (20 min) period of air breathing. Thereafter, cats were made to breathe either air (open circles) or air and carbon dioxide (closed circles). Other studies with carotid bodies surgically modified showed less carbon dioxide effect on rectal temperature. (Adapted and redrawn with permission from Jennings and Szlyk, 1986.)

intersect on the overall work limitation curve. Thus a cardiovascular-respiratory interaction and a respiratory-thermal interaction could be found, but no cardiovascular-thermal interaction would be expected as long as the respiratory limitation was interposed between them.

There is limited evidence to suggest a respiratory-thermal interaction. Johnson and Berlin (1973) present very tenuous and indirect evidence of this interaction. Jennings and Szlyk (1986) gave a stronger physiological basis to the interaction by demonstrating that the carotid bodies, important in respiratory control (see Section 4.3.1), can also affect temperature regulation (Figure 1.2.5). In their animals they showed that hypoxic stimulation of the carotid bodies suppresses shivering.

Body temperature has been found to have a direct effect on heart rate (Rubin, 1987), and, therefore, a thermal-cardiac interaction might also be expected in some humans. The implications of this intensity-duration concept cannot be drawn too far.

1.3 MUSCLE METABOLISM

Although the previous section suggests many possible limitations to exercise performance, the most widely considered limitation involves the basic energy mechanisms of the muscles themselves. For exercise durations of 0-900 sec (0-15 min), these mechanisms most surely dominate exercise capacity.

1.3.1 Muscle Fiber Structure

Individual muscle fibers have been found to be composed of fibrils (about 1 µm in diameter), which are themselves composed of the protein filaments actin and myosin (White et al.,
EXERCISE INTENSITY AND DURATION

Figure 1.3.1 Relationships between actin and myosin filaments in three muscle conditions. (Redrawn with permission from White et al., 1959.)

These filaments are crosslinked, either directly or indirectly, by chemical bonds (Figure 1.3.1). When muscle contraction occurs, these bonds must be broken and other bonds, which slide the actin filaments along the myosin filaments, must be established. Such a process requires a source of energy which is immediate and can deliver energy over a considerable amount of time.

The force per unit area (also called tension) that a muscle develops varies with the length of the muscle fiber. Tension developed can be measured either during an isometric (or constant-length) contraction or on an unstimulated, passive muscle fiber. Length of the muscle fiber is usually related to resting length. The length-tension relationship between muscles, which affects their efficiencies, is discussed in detail in Section 5.2.5.

Two types of muscle fibers, slow twitch and fast twitch, have been identified. Fast-twitch fibers are primarily those concerned with fine, rapid, precise movement. Slow-twitch fibers are involved in strong, gross, sustained movements (Ganong, 1963). Fast-twitch fibers appear to be more adapted for anaerobic contraction, whereas slow-twitch fibers utilize oxygen better (Kamon, 1981). Therefore, a higher proportion of slow-twitch fibers in a given muscle mass should provide a better aerobic endurance of the muscle.

1.3.2 Muscle Energy Sources

Organic phosphate compounds are the fundamental energy sources for muscle cells. Of particular importance is adenosine triphosphate (ATP). ATP can be hydrolyzed by actomyosin, which affects its physical state. When ATP is hydrolyzed, it forms phosphate

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*The muscle can lock (establish stable crosslinking) at any point between 65 and 120% of the resting length (White et al., 1959). A study of mechanisms involved in the process is given by Davis (1986).

Adenosine is an organic nucleic acid adenine linked to ribose (White et al., 1959). When one ring hydroxide is replaced with three phosphate groups (phosphorus and oxygen), the result is adenosme triphosphate. There are two high-energy pyrophosphate bonds in ATP.
EXERCISE INTENSITY AND DURATION

plus free energy plus adenosine diphosphate (ADP). ADP contains one energy-rich bond and can also be used as a muscle energy source. Adenosine monophosphate (AMP), the final product of ATP and ADP hydrolysis, contains no usable energy for muscular contraction. There are severe restrictions on AMP as a phosphate acceptor; it cannot accept phosphate either from anaerobic glycolysis or from oxidative reactions, which may be one reason why ATP is almost immediately formed from ADP whenever possible.

ATP is used as the energy-rich carrier not only for muscular contraction but also for resting metabolic processes, such as protein formation and osmotic maintenance. For these, there is ample store of ATP within the muscle. ATP formation occurs continually by the oxidation of carbohydrate or acetoacetate (White et al., 1959).

Maximally contracting mammalian muscle uses approximately $1.7 \times 10^{-5}$ moles of ATP per gram per second (White et al., 1959). However, ATP stores in skeletal muscle tissue amount to $5 \times 10^{-6}$ moles per gram of tissue, which can meet muscle demands for no more than 1/2 sec of intense activity.

Initial replenishment of ATP occurs through the transfer of creatine phosphate (also called phosphagen) into creatine, a reaction which is catalyzed by creatine kinase (White et al., 1959). In the resting state, muscle contains four to six times as much creatine phosphate as it does ATP. Phosphocreatine, however, cannot directly affect actomyosin.

Even considering phosphocreatine, the total supply of high-energy phosphate cannot sustain activity for more than a few seconds. Glycogen is a polysaccharide present in muscle tissue in large amounts. When required, glycogen is decomposed into glucose and pyruvic acid. This pyruvic acid, in turn, becomes lactic acid. ATP is formed in this process. All these reactions proceed without oxygen. During intense muscle activity, the oxygen content of blood flowing through muscle tissue can be rapidly depleted (anaerobic conditions).

When sufficient oxygen is available (aerobic conditions), either in muscle tissue or elsewhere, these processes are reversed. ATP is reformed from ADP and AMP, creatine phosphate is reformed from creatine and phosphate, and glycogen is reformed from glucose or lactic acid. Energy for these processes is derived from the complete oxidation of carbohydrates, fatty acids, or amino acids to form carbon dioxide and water (Molé, 1983).

Following the manner of Astrand and Rodahl (1970), the foregoing reactions can be summarized by chemical equations:

**Anaerobic:**

$$ATP \Leftrightarrow ADP + P + \text{free energy} \quad (1.3.1)$$

$$\text{creatine phosphate} + ADP \Leftrightarrow \text{creatine} + ATP \quad (1.3.2)$$

$$\text{glycogen or glucose} + P + ADP \rightarrow \text{lactate} + ATP \quad (1.3.3)$$

**Aerobic:**

$$\text{glycogen or fatty acids} + P + ADP + O_2 \rightarrow CO_2 + H_2O + ATP \quad (1.3.4)$$

**All conditions:**

$$2ADP \Leftrightarrow ATP + AMP \quad (1.3.5)$$

Anaerobic and aerobic processes can occur simultaneously in different parts of the body. Lactic acid freely diffuses from muscle cells into interstitial fluid and thence to the blood.

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6Glycogen has been likened to animal starch. If the amount of energy equivalent to glycogen were present in the form of the simple sugar glucose, the osmotic balance of muscle tissue would be gravely upset.
where it is carried to the liver. Most of the lactic acid is resynthesized to glycogen in the liver, at the expense of liver ATP. Liver glycogen is released as blood glucose for utilization by muscle.

If muscular work is at a pace slow enough for sufficient oxygen delivery for aerobic measures to prevail, then the glucose is directly utilized in muscle to generate ATP. If oxygen is not available, then anaerobic processes yield sufficient ATP for muscular action. Because there is a limit to the amount of anaerobic metabolites that can be tolerated by muscle tissue, there is also a limit to the duration of anaerobic metabolism. Oxygen is required to chemically remove these metabolites from the tissue. The greater the concentration of metabolites, the greater is the amount of oxygen required to reform resting levels of glycogen and phosphocreatine. This, in turn, leads to the concept of oxygen debt.

1.3.3 Oxygen Debt

At the cessation of exercise there remains an elevated need for oxygen (Figure 1.3.2). The amount of oxygen utilized after exercise, above normal resting levels, is termed the oxygen debt. As we have discussed, much of this oxygen debt is accumulated by muscle biochemistry. However, there are other contributing factors to oxygen debt: (1) elevated body temperature immediately following exercise increases bodily metabolism in general, which requires more than resting levels of oxygen to service; (2) increased blood epinephrine levels increase general bodily metabolism; (3) increased respiratory and cardiac muscle activity requires oxygen; (4) refilling of body oxygen stores requires excess oxygen; and (5) there is some thermal inefficiency in replenishing muscle chemical stores. Considering only lactic acid oxygen debt, the total amount of oxygen required to return the body to its normal resting state is about double. Viewed the other way, the efficiency of anaerobic processes is about 50% of aerobic processes (Astrand and Rodahl, 1970).

Figure 1.3.2 Oxygen uptake at the beginning of exercise increases gradually until reaching a level high enough to meet demands of the tissues. At the end of exercise, oxygen uptake gradually returns to the resting level as the oxygen debt is filled. (Adapted and redrawn with permission from Astrand and Rodahl, 1970.)

7Plasma lactate may play a part in the release of adrenocorticotropic hormone (ACTH) and other hormones associated with mobilization reactions of bodily systems to exercise (Farrell et al., 1983).

8There is a very intricate regulation of blood glucose, the complete description of which is outside the purview of this book. Basically, glucose input depends mostly on ingested carbohydrate, which in turn is dependent on hypothalamic and thyroid function. Insulin acts to remove glucose from the blood and produce liver glycogen. Epinephrine and glucagon decrease liver glycogen and increase blood glucose (White et al., 1959).

9Also reformed is oxymyoglobin. Muscle tissue contains a protein similar to blood hemoglobin, which also binds to, and stores, oxygen. The major difference between myoglobin and hemoglobin is that the former stores one oxygen atom, whereas the latter stores four oxygen atoms for every hemoglobin molecule in the oxidated state.
This muscular cycle is reflected in the amount of heat generated by the muscles. There is a small amount of resting heat produced by the muscles reflecting basic muscle metabolism; there is an initial heat produced during muscle contraction and relaxation; and there is a heat of recovery during the restoration of the muscle to its preactivated state. Heat of recovery is nearly equal to initial muscle energy expenditure (Mende and Cuervo, 1976).

That muscular activity results in heat as well as useful mechanical work means that muscles are less than 100% efficient. In fact, the large muscles are about 20-30% efficient, about the same as a gasoline engine (Morehouse and Miller, 1967). Efficiency is diminished by excessive loads, excessive rate of work, and fatigue (see Chapter 5).

During heavy work there is a discrepancy between muscular energy demand and aerobic energy available. In Figure 1.3.3 can be seen the relative energy contributions of aerobic fuel utilization and the two anaerobic contributions of anaerobic glycolysis and phosphocreatine utilization. Similar information is available from Table 1.3.1 and Figure 1.3.4. As the level of work decreases, such that performance time increases, the relative contribution of aerobic energy provision increases.

The more a person must rely on anaerobic processes to perform any given task, the greater will be that person's oxygen debt. From Table 1.3.1, an athlete competing in a 60-120 sec (1-2 min) event requires about 167 kN·m (40 kcal) to be repaid as a lactic acid oxygen debt. For each cubic meter (1000 L) of oxygen used, about 20,900 kN·m (5000 kcal) will be delivered, resulting in a lactic acid oxygen debt of 0.008 m³ (8 L). Reformation of ATP and creatine phosphate requires about 0.001-0.0015 extra cubic meters of oxygen (total thus far of 0.0095 m³). Assuming the basic efficiency of oxygen repayment is about 50%, an increase in oxygen uptake of about 0.0019-0.0020 m³ follows the exercise.10

With performance times up to 120 sec, anaerobic power dominates aerobic power. At about 120 sec, each is of equal importance. With longer performance time, aerobic power prevails (Figure 1.3.4). Therefore, at performance times below about 120 sec exercise is

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**Figure 1.3.3** Energy transfer kinetics. (Redrawn with permission from Molé, 1983.)

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10Blood lactate levels decline more rapidly while exercising during the recovery period than while resting (Stamford et al, 1981).
**TABLE 1.3.1** Contributions of Anaerobic (Lactate) Energy Sources to Total Work Requirement

<table>
<thead>
<tr>
<th>Performance Time, Sec</th>
<th>Total Energy</th>
<th>Anaerobic Sources</th>
<th>Aerobic Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>kN·m (kcal)</td>
<td>kN·m/sec</td>
<td>kN·m %</td>
</tr>
<tr>
<td>10</td>
<td>121 (29)</td>
<td>12.1</td>
<td>105 85</td>
</tr>
<tr>
<td>60</td>
<td>251 (60)</td>
<td>4.18</td>
<td>167 65-70</td>
</tr>
<tr>
<td>120</td>
<td>376 (90)</td>
<td>3.14</td>
<td>188 50</td>
</tr>
<tr>
<td>240</td>
<td>607 (145)</td>
<td>2.53</td>
<td>188 30</td>
</tr>
<tr>
<td>600</td>
<td>1,190 (285)</td>
<td>1.99</td>
<td>146 10-15</td>
</tr>
<tr>
<td>1,800</td>
<td>3,050 (730)</td>
<td>1.70</td>
<td>125 5</td>
</tr>
<tr>
<td>3,600</td>
<td>5,440 (1,300)</td>
<td>1.51</td>
<td>84.7 2</td>
</tr>
<tr>
<td>7,200</td>
<td>10,000 (2,400)</td>
<td>1.39</td>
<td>62.7 1</td>
</tr>
</tbody>
</table>

*Source:* Adapted and used with permission from Astrand and Rodahl, 1970.

*aBased on the following assumptions: (1) 20.9 kN·m energy is equivalent to oxygen uptake of 0.001 m³; (2) an individual's maximal aerobic capacity is 188 kN·m; (3) 100% of maximal oxygen uptake can be maintained during 600 sec, 95% during 1800 sec, 85% during 3600 sec, and 80% during 7200 sec.

**Figure 1.3.4** Relative contributions of total energy requirement from aerobic and anaerobic processes. At 120 sec, both processes are of equal importance. (Adapted and redrawn with permission from Astrand and Rodahl, 1970.)

mostly limited by cellular mechanisms; above 120 sec, up to 3600 sec, performance decrement is more likely to be from systemic causes which interfere with oxygen transport.

### 1.3.4 Maximal Oxygen Uptake

If an individual exercises while utilizing large muscle groups (so that small muscle fatigue is not a performance factor), performing dynamic, not static work (static work inhibits blood
flow), and for a performance time exceeding about 180 sec (so that oxygen can reach steady state before the cessation of exercise), there will be found a rate of oxygen delivery to the muscles which cannot be exceeded. This value is termed the maximal oxygen uptake, or maximal aerobic power for the individual. Maximal oxygen uptake appears at a relatively high work rate (250 N-m/sec in Figure 1.3.5), but not necessarily at the highest attainable work rate, the highest work rate that can be performed for at least 180 sec. Although the rate of work can be increased, the rate at which oxygen is delivered to and used by the body cannot be increased. There is a significant and fast-rising increase in blood lactic acid, indicating that anaerobic metabolism has already begun (Figure 1.3.6).

Below the maximal oxygen uptake, the rate of oxygen use is directly proportional to the rate of work (Figure 1.3.6). The actual rate of oxygen use will depend on the muscle groups used and their relative efficiencies. When maximal oxygen uptake is reached, it too depends on the muscles used and the way in which they are used (Astrand and Rodahl, 1970). As long as exercise is performed in an upright position, and with the legs or arms and legs together, there is no appreciable difference in oxygen uptake (Table 1.3.2) for different kinds of exercise (running, cycling, cross-country skiing, etc.). While supine, however, legs-only exercise gives a maximal oxygen uptake of about 85% of upright maximal oxygen uptake, and swimming (arms plus legs) yields 90%.

The exact mechanism limiting oxygen uptake has been the subject of controversy. Faulkner et al. (1971) suggest that the limiting mechanism is the rate at which blood can be pumped by the heart. With a higher capacity, more oxygen could be delivered to the muscles.

Figure 1.3.5 Oxygen uptake increases with time and work load up to the maximum oxygen consumption. Thereafter, oxygen uptake remains constant and additional required energy is produced by a combination of aerobic and anaerobic processes. Symbols refer to different work levels. (Adapted and redrawn with permission from Astrand and Rodahl, 1970.)
Figure 1.3.6 Steady-state oxygen consumption related to work rate. Oxygen uptake increases linearly with work rate until maximum oxygen uptake is reached. Blood lactic acid begins to rise before maximum oxygen uptake is reached. Symbols refer to work levels in the previous figure. (Adapted and redrawn with permission from Astrand and Rodahl, 1970.)

### TABLE 1.3.2 Maximal Oxygen Uptake for Tasks Using Arm and Leg Muscles

<table>
<thead>
<tr>
<th>Age, yr</th>
<th>Women, ( \text{m}^3\text{/sec}) (L/min)</th>
<th>Men, ( \text{m}^3\text{/sec}) (L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-29</td>
<td>(3.57 \times 10^{-5}) ((2.14 \pm 0.25))</td>
<td>(5.27 \times 10^{-5}) ((3.16 \pm 0.30))</td>
</tr>
<tr>
<td>30-39</td>
<td>(3.33 \times 10^{-5}) ((2.00 \pm 0.23))</td>
<td>(4.80 \times 10^{-5}) ((2.88 \pm 0.28))</td>
</tr>
<tr>
<td>40-49</td>
<td>(3.08 \times 10^{-5}) ((1.85 \pm 0.25))</td>
<td>(4.33 \times 10^{-5}) ((2.60 \pm 0.25))</td>
</tr>
<tr>
<td>50-59</td>
<td>(2.75 \times 10^{-5}) ((1.65 \pm 0.15))</td>
<td>(3.87 \times 10^{-5}) ((2.32 \pm 0.27))</td>
</tr>
</tbody>
</table>

Source: Adapted and used with permission from Kamon, 1981.

*Numbers in parentheses are averages plus or minus 1 standard deviation.

Maximal oxygen uptakes of about \(4.2 \times 10^{-5} \text{m}^3\text{O}_2/\text{sec}\) (2.5 L/min) are typical for young (20-30 years of age) male nonathletes (Astrand and Rodahl, 1970). This figure becomes \(6.1 \times 10^{-7} \text{m}^3\text{O}_2/\text{kg-s}ec\) for a typical 68 kg man. Well-trained male athletes possess maximal oxygen uptakes twice as high as this, and untrained women have maximum oxygen uptakes 70% as large. There is a rapid increase in maximum oxygen uptake before the age of 20, with no significant sex difference before the age of 12, and a gradual, nearly linear decline with age after 20, reaching about 70% of the age 20 value at age 65.\(^{11}\) There is a large individual

\(^{11}\)Higginbotham et al. (1986) demonstrated that this age-related decline is probably the result of reduced exercise heart rate in older subjects rather than a reduction in stroke volume or peripheral oxygen utilization.
variation, which limits application of these values to particular people. Modern training methods, especially for women, have dramatically altered their relative maximum oxygen uptakes. Active older individuals are likely to possess higher maximum oxygen uptakes than sedentary younger individuals. Since capacity for work depends directly on maximum oxygen uptake, there is a great influence of training on work capacity.

Training increases maximum oxygen uptake and also increases maximum oxygen debt. Muscle metabolism becomes more efficient, and muscle stores of ATP, creatine phosphate, and glycogen increase (Astrand and Rodahl, 1970). Muscle basal metabolism (see Chapter 5) decreases, indicating increased metabolic efficiency (Morehouse and Miller, 1967). Muscle mass increases, the capillary density increases, and myoglobin content increases (Astrand and Rodahl, 1970; Morehouse and Miller, 1967). Heart volume increases dramatically, to the point where it would be considered unhealthy for an untrained individual (see Chapter 3). At the same time, heart rate decreases and blood volume increases. Beyond that, movement efficiency increases due to a learning effect.

Kamon (1981) presented an equation from which can be obtained a relationship between endurance time and the relative work rate, given as a fraction of an individual's maximum oxygen uptake:

\[
t_{wd} = 7200 \left( \frac{V_{O_2,\text{max}}}{V_{O_2}} \right) - 7020
\]

(1.3.6)

where \( t_{wd} \) = endurance time for dynamic work, sec

\( V_{O_2} \) = oxygen uptake, \( \text{m}^3/\text{sec} \)

\( V_{O_2,\text{max}} \) = maximum oxygen uptake, \( \text{m}^3/\text{sec} \)

Equation 1.3.6 can be used for rhythmic or dynamic work tasks. Static effort occludes flow of blood to the muscles and reduces endurance time (Kamon, 1981):

\[
t_{ws} = 11.40 \left( \frac{MT_{\text{max}}}{MT} \right)^{2.42}
\]

(1.3.7)

where \( t_{ws} \) = static effort endurance time, sec

\( MT \) = muscle torque, N·m

\( MT_{\text{max}} \) = maximum muscle torque, N·m

1.3.5 Anaerobic Threshold

The onset of progressive lactic acid accumulation with graded exercise is called the anaerobic threshold (Wasserman et al., 1973). The anaerobic threshold is a benchmark in exercise physiology. Below it, one set of physiological assumptions appears to hold; above it, physiological adjustments are much less simple. The anaerobic threshold occurs at workloads between 50 and 80% of maximal oxygen uptake. Anaerobic threshold for athletes is higher than it is for inactive individuals.

Measured anaerobic threshold has been defined in different ways by different workers. It can be indicated by a threshold level of lactate in the blood (Farrell et al., 1979), increased output of carbon dioxide from the lungs (Sutton and Jones, 1979, Chapter 4), increased rate of respiratory ventilation (linear with work rate below the anaerobic threshold) above the predicted linear value (Davis et al., 1976; Wasserman et al., 1973, Figure 4), an increase in respiratory exchange ratio (rate of carbon dioxide produced divided by rate of oxygen used) above its resting level (Issekutz et al., 1967; Naimark et al., 1964) and various end-tidal gas partial pressure measures (Davis et al., 1976; Martin and Weil, 1979; Wasserman et al., 1973). However, none of these definitions is quite satisfactory; they all suffer from shortcomings of one kind or another. Blood lactate accumulation as a definition suffers from the presence of concurrent lactate removal; therefore, by this definition anaerobic threshold does not accurately reflect the onset of anaerobic metabolism. Rate of exhaled carbon dioxide as a definition suffers from its indirectness and the influence of respiratory anatomical, mechanical, and control factors on carbon dioxide excretion (see Chapter 4). Although