Handbook of
Sports Medicine
and Science

Volleyball

Second Edition

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Publication

FIVB
FÉDÉRATION INTERNATIONALE DE VOLLEYBALL

EDITED BY

Jonathan C. Reeser and Roald Bahr

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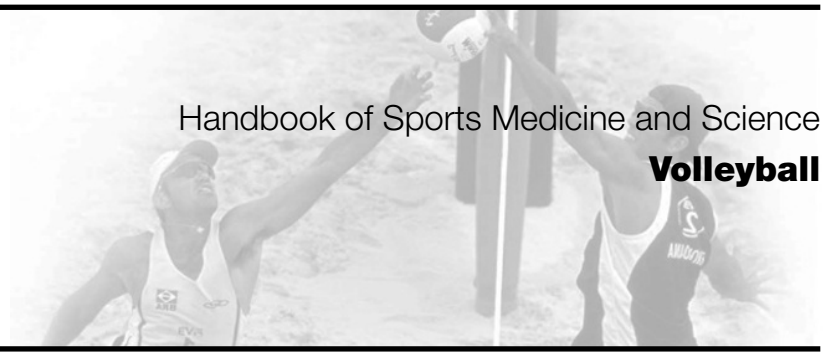

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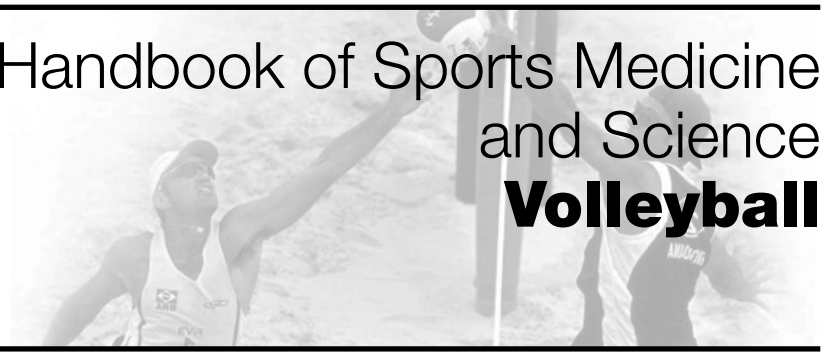

SECOND EDITION

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Foreword

Volleyball is truly a universal sport; it can be played by people of all ages and nationalities in different locations anywhere in the world. The sport has experienced rapid development in recent years and is enjoying a golden era of success. We are seeing more nations compete at the FIVB's flagship events and more people playing the sport at amateur level.

As the international federation responsible for volleyball, the FIVB has a duty to protect the health and well-being of volleyball athletes, and to educate and inform its 222 national federations on medical best practice. The FIVB's Medical Commission is fundamental to this and carries out medical research in a number of areas, including prevention and treatment of common volleyball injuries, to ensure the volleyball environment is a safe one.

At a time when more and more people are participating in volleyball, it is important to raise awareness of the sport's demands as well as promoting its health benefits. We therefore value this handbook as an important tool for volleyball medical professionals around the world and as a useful resource for players, coaches, and officials alike.

Dr. Ary S. Graça
FIVB President

Foreword

Introduced to the world of sport in 1895, volleyball has become one of the most popular sports on the international sporting scene. Volleyball made its Olympic debut at the 1964 Games in Tokyo, and beach volleyball followed in 1996 at the Olympic Games in Atlanta. For both events, men and women compete. With great demands on biomechanics, coordination and physiology, the volleyball athlete must train for both the performance of skills and for physiological conditioning.

A first edition on volleyball for the IOC Medical Commission's Handbooks of Sports Medicine and Science series appeared in 2003. This publication has enjoyed widespread use as a source of authoritative information on all aspects of volleyball competition. Both the first edition and the present edition were developed under the editorial leadership of Drs Jonathan Reeser (USA) and Roald Bahr (Norway). The co-editors benefitted from the expertise of a team of contributing authors representing outstanding clinicians and scientists. Authoritative information has been presented for various topics of volleyball sports science, topics of sports medicine, volleyball for special populations, and the special topics of ergogenic aids, doping, and sports psychology.

We are very grateful to the editorial team and all the contributing authors for the quality of their work in making this second edition of the *IOC Handbook on Volleyball* a highly valuable publication in both sports medicine and sports science literature.

Thomas Bach
IOC President

Preface and acknowledgments

Herewith, we proudly present the second edition of the *IOC Handbook of Sports Medicine and Science: Volleyball*. More than 10 years have elapsed since the first edition appeared, and over that time the sport has rapidly evolved. In addition, we have witnessed a veritable explosion of clinical and basic scientific literature pertaining to volleyball. This is reflected in the content of this handbook: the table of contents has been revised and most chapters have been essentially rewritten – typically with the inclusion of new contributors in order to bring a fresh perspective to the material.

While the content of the second edition has been updated, the process by which it was brought into being remained essentially unchanged. We remain ever appreciative of Dr Howard G. (Skip) Knuttgen, coordinator of scientific publications for the IOC Medical Commission, for his patient oversight of this project. We extend our gratitude to the staff at Wiley-Blackwell for their myriad contributions to bringing this second edition to fruition. We also acknowledge the support of the Fédération Internationale de Volleyball (FIVB) and its President, Dr Ary Da Silva Graça Filho, and that of the IOC Medical and Scientific Department (Richard Budgett, Director). Finally, we extend our sincere appreciation to our many contributors, without which this handbook would not have happened.

We sincerely hope that we have succeeded in providing you, the reader, with a work that captures most (if not all) of the advances that have become integral to our present understanding of volleyball sport science and to the practice of sports medicine as applied to our fabulous sport. As before, we invite you to share your comments and suggestions, so that future iterations of this Handbook may continually improve.

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PART 1
VOLLEYBALL SPORT SCIENCE

Chapter 1

Energy demands of volleyball

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Introduction

Volleyball, like all team sports, requires repetitive bouts of high-intensity exercise. For the volleyball player to achieve competitive success, he/she must possess the ability to rapidly generate power while executing precise sport-specific skills such as spiking and blocking. In addition, the ability to maintain sufficient power output for the full duration of matches is obviously of critical importance to sporting success. The extent and speed of recovery from exercise are influenced by the intensity and duration of the preceding bout of exercise, the nutritional status of the individual, and the time available for metabolic recovery. Volleyball athletes must perform numerous maximum effort jumps and quick, short sprints, interspersed by variable periods of exercise of lower intensity or brief periods of rest. The energy used during periods of high-intensity play is derived largely from anaerobic metabolism. Over the course of the match, however, the contribution of aerobic metabolism increases to cover the total energy cost. The cycles of activity and rest are imposed by the pattern of play which vary greatly from player to player and from one match to another, as the tactics and ability of the opposition also influence the demands on each player.

Compared with continuous exercise activities such as running and cycling, relatively little attention has been directed to the energy expenditure during games that involve complex movement patterns. This may be because of the lack of adequate experimental models to study these activities in the laboratory. However, some standardized models of intermittent exercise have been developed recently that simulate the activity patterns observed in team sport. This chapter describes how these protocols, as well as measurements made during competition itself, have shed some light on the metabolic processes that occur during match-play exercise and their importance for achieving peak performance.

Activity patterns and work rate in volleyball and other sports

Based upon unpublished data collected by the Fédération Internationale de Volleyball (FIVB) during the 2015 World League and Grand Prix competitions, it appears that the work periods for elite male indoor players range between 6–8 seconds in length, while for elite female indoor players the work periods typically measure 7–9 seconds. Furthermore, the ball is "in play" for approximately 15% of the duration of the match, resulting in a work:rest ratio of approximately 1:6. These data reflect the fact that volleyball athletes must generate explosive power, then recover quickly so as to be ready for the next point.

Volleyball, Second Edition. Edited by Jonathan C. Reeser and Roald Bahr.

It is predictable that players suffer from progressive fatigue as the competitive match wears on, as manifested by a drop in the work rate during the second half of the match (fewer number or reduced height of maximum jumps performed). A recent study of professional soccer players using Pro-Zone technology to analyze time spent in different activities during 28 English Premier League matches found that during the last 15 minutes of a match, athletes cover approximately 20% less distance than they had covered during the opening of the match. There was also a noticeable decline in high-intensity running immediately after the most intense 5-minute period of the game, with the greatest deficits (~40–50%) in attacking players and central defenders. It is common to see more goals scored in the later stages of games as players become fatigued and more mistakes are made. Injuries are also more likely to occur late in the game when fatigue becomes more prevalent.

The development of fatigue during match-play seems to be related at least in part to depletion of muscle glycogen stores. It has been shown that football players who start a match with a low thigh muscle glycogen content cover 25% less distance than those who have normal prematch thigh muscle glycogen stores (see Table 1.1). Furthermore, players with a low initial muscle glycogen content covered 50% of the total distance walking and only 15% sprinting, compared with 27% walking and 24% sprinting for the players with normal to high muscle glycogen stores. Blood lactate concentration is consistently lower at the end of a match compared with values measured at half-time, and this ties in with the observation that the greatest rate of decline in muscle glycogen occurs in the first half of the match. Players who start matches with low glycogen stores in their leg muscles are likely to be close to complete glycogen depletion by half-time and these findings have important implications for training and the nutritional preparation of players. Until relatively recently, however, these issues have largely been ignored.

Table 1.1 Maximum rates of ATP resynthesis that can be achieved by the metabolic pathways available to muscle cells.

	μmol/min/g muscle
PCr hydrolysis	440
Lactate formation	180
CHO oxidation	40
Fat oxidation	20

ATP, adenosine triphosphate; CHO, carbodhydrate; PCr, phosphocreatine.

Metabolic responses to intermittent high-intensity exercise

All cellular activities, including nerve transmission, biosynthesis, and muscle contractility, are fueled by the chemical energy released when the high-energy phosphate bond(s) in the adenosine triphosphate (ATP) molecule are broken (Figure 1.1). ATP is broken down under the influence of a specific enzyme (an ATPase) to adenosine diphosphate (ADP) and inorganic phosphate (P_i) to yield energy for muscle activity or to power other reactions. This high-energy phosphate bond is an immediate source of energy, the so-called energy currency of the cell. All other energy-producing reactions must channel their output through this mechanism.

There are three principal means by which cells maintain their supply of readily available ATP. The first and most rapid of the routes begins with the conversion of phosphocreatine (PCr) to creatine and phosphate. However, the phosphate group is not liberated as inorganic phosphate, but is rather transferred directly to an ADP molecule to re-form ATP. This reaction is catalyzed by the enzyme creatine kinase, which is present in skeletal muscle at very

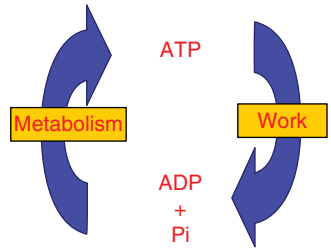

Figure 1.1 Energy is released to allow cells to do work when the ATP molecule is hydrolyzed to ADP and Pi. The ATP level in the cells must be maintained to allow work to continue, so other metabolic pathways must provide the energy for ATP resynthesis.

high activities, allowing the reaction to occur rapidly. In the second pathway, glucose-6-phosphate (derived from the breakdown of muscle glycogen or from glucose taken up from the bloodstream) is metabolized to lactate and produces ATP by substrate-level phosphorylation reactions. Neither of these reactions requires oxygen, and the pathways are therefore commonly considered to be "anaerobic."

In the third pathway, the products of carbohydrate, lipid, protein, and alcohol metabolism can enter the tricarboxylic acid (TCA) cycle (also known as the citric acid or Krebs cycle, after Sir Hans Krebs, who first described it) in the mitochondria and can be oxidized to carbon dioxide and water. This process is known as oxidative phosphorylation, and in the presence of oxygen yields substantial energy used in the synthesis of ATP.

Adenosine triphosphate, then, is the immediate source of cellular energy and the purpose of the three mechanisms described is to regenerate ATP at sufficient rates to prevent a significant decline in the intramuscular ATP concentration. If the ATP concentration falls, the concentrations of ADP and adenosine monophosphate (AMP) will rise. The concentration ratio of ATP to ADP and AMP is a marker for the energy status of the cell. If the ratio is high, the cell is in effect "fully charged." This energy charge is monitored in every cell; a fall in the ATP concentration or a rise in the concentration of ADP or AMP will activate the metabolic pathways necessary to increase ATP production. This is achieved by activation or inhibition of key regulatory enzymes by changes in the concentration of the adenine nucleotides.

It may also be helpful to think of the various pathways that can be used to resynthesize ATP in terms of the maximum rates of resynthesis that can be achieved. Some typical values are shown in Table 1.1. It is important to note that these rates are influenced by many factors, including muscle fiber type, fitness level, and the nutritional status of the athlete.

Since substantial storage of ATP in tissues is not possible (the amount of chemical energy stored in each molecule of ATP is rather small and it would be inefficient to store more because of the mass that would have to be carried), the challenge during exercise is for the cell to resynthesize ATP as fast as it is broken down, thereby maintaining an adequate intracellular supply of energy. A 70 kg runner moving at a speed of 15 km/h will require about 3.5 L of oxygen per minute, or about 1.17 kW. To meet this energy demand, the runner must break down about half a kilogram of ATP every minute to maintain pace. Given that the total ATP content of the body is about 50 g, this means that each molecule of ATP in the body turns over on average about once every 6 seconds. In active muscle cells, the rate of turnover will be much higher.

The data in Table 1.1 show the maximum rate of ATP resynthesis that can be generated by the various metabolic pathways in muscle and most other tissues. The power which each of these systems can generate is dependent upon the capacity of the system (Table 1.2). The capacity of the oxidative metabolic pathways is essentially unlimited because of the very large amounts of substrate stored and the fact that these substrates can be replenished during exercise by ingestion.

Every time a player initiates a burst of activity, the rates of PCr utilization and glycolysis increase. Muscle samples collected from soccer players during and after matches show progressive reduction in the [PCr] and severalfold increases in blood and muscle lactate concentrations as the intensity and duration of exercise increase. Thus, the anaerobic energy systems are heavily taxed during periods of intense exercise during match-play.

Glycolysis converts one 6-carbon glucose molecule to two 3-carbon molecules, while allowing some of the energy liberated to be conserved as ATP. The key reactions of anaerobic glycolysis are shown in Figure 1.2. It is important to note that there is an initial investment in the form of energy, but there is a net yield of ATP when glycogen

Table 1.2 Power that can be generated by the various metabolic pathways and the total capacity of those systems.

	Power (W/kg)	Capacity (J/kg)
ATP/PCr hydrolysis	800	400
Lactate formation	325	1000
Oxidative metabolism	200	Unlimited

ATP, adenosine triphosphate; PCr, phosphocreatine.

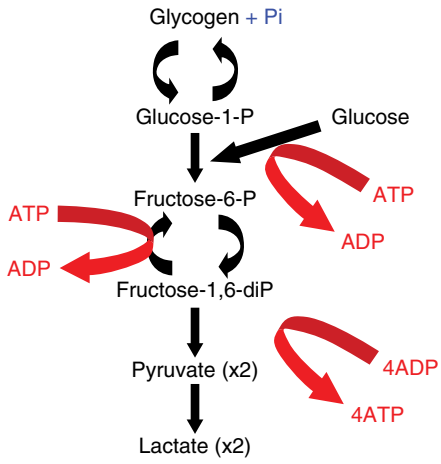

Figure 1.2 Key reactions of glycolysis, showing steps where energy must be added to the system and where ATP is generated.

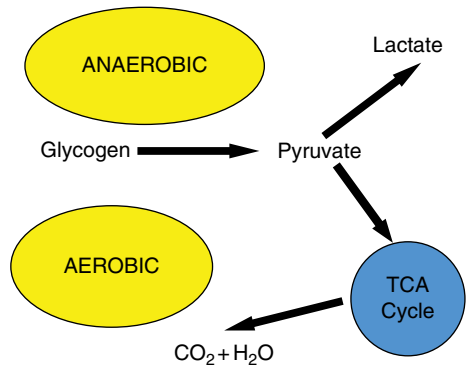

Figure 1.3 The metabolic fate of carbohydrate (glycogen) stored in muscle depends on exercise intensity and the metabolic capacity of the muscle cell.

(or glucose) is broken down to lactate. Degradation of glycogen to lactate allows formation of ATP at relatively high rates; the enzymes that catalyze the reactions of glycolysis are present at high activities and the substrate (glycogen) is normally present in relatively large amounts, though the concentration falls rapidly during high-intensity activity. For each glucose residue converted to lactate, three ATP molecules are formed if glycogen is the starting point, and two are formed if glucose is the substrate.

When muscle glycogen is rapidly broken down, pyruvate is produced at a rate faster than it can be oxidized via the TCA cycle. This leads to depletion of nicotinamide adenine dinucleotide (NAD), which acts as a co-factor in the glycolytic pathway by accepting a hydrogen atom, being converted in the process to NADH. NAD is present in the cell in very small amounts, and for glycolysis to continue, it must be regenerated. At rest and during low-intensity exercise, this is accomplished by the oxidation of the pyruvate produced by glycolysis to carbon dioxide and water. During high-intensity exercise, however, glycolysis proceeds faster than the capacity of the aerobic pathway to dispose of the pyruvate formed (Figure 1.3).

Conversion of pyruvate to lactate occurs faster than the metabolism of pyruvate to carbon dioxide and water, thereby allowing energy production by glycolysis to continue at high rates. Thus, although lactate formation is often seen as a negative process, it is actually a positive process in that it allows high-intensity work to be performed. Anaerobic glycolysis is clearly an important source of ATP resynthesis during high-intensity activity, and becomes especially important during repeated bouts of high-intensity exercise when the creatine phosphate content of the muscle may be depleted. Anaerobic glycolysis produces only three molecules of ATP for each molecule of glucose 6-phosphate derived from muscle glycogen, compared with 38 molecules of ATP when the glucose molecule is completely oxidized to carbon dioxide and water. However, the limited capacity of ATP regeneration made possible by shunting pyruvate to the anaerobic glycolytic pathway may be considered compensated for by the speed of the reaction and the high power that can as a result be generated.

The reactions of glycolysis result in a release of hydrogen ions that cause the muscle pH to fall as lactate accumulates. This has a variety of effects on the muscle as shown in Figure 1.4. Some of the hydrogen ions are immediately buffered within the muscle cell and some diffuse out of the cell into the extracellular space, limiting the intracellular in intracellular pH.

Despite the negative effects of a falling pH, the energy made available by anaerobic glycolysis allows a higher intensity of exercise than would otherwise be possible.

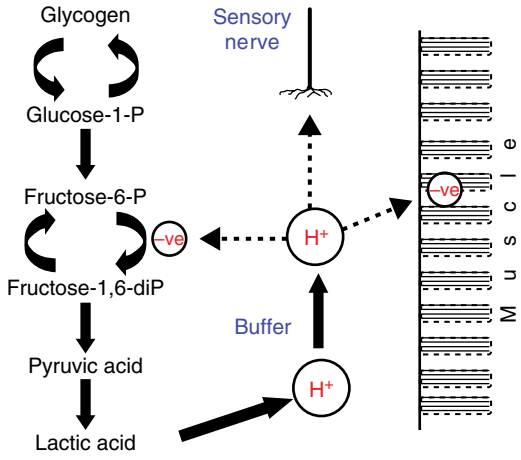

Figure 1.4 The glycolytic pathway (note that several steps have been omitted to show only key reactions and consequences).

Aerobic metabolism ultimately provides all the energy used by the body, and this is achieved by the oxidation of stored or ingested fuels in the form of carbohydrate, protein, fat, and alcohol (Figure 1.5).

The pyruvate that results from glycolysis (which occurs in the cell's cytoplasm) normally moves into the mitochondria where it is aerobically metabolized via the citric acid cycle. This cyclical series of biochemical reactions leads to the oxidation of pyruvate to carbon dioxide and water, and in the process generates ATP via oxidative phosphorylation (aerobic metabolism). The citric acid cycle is the final common pathway for the metabolism of carbohydrate, fats, and protein. Aerobic metabolism generates about 90% of all the ATP produced by the body, while anaerobic metabolism produces the remaining 10%.

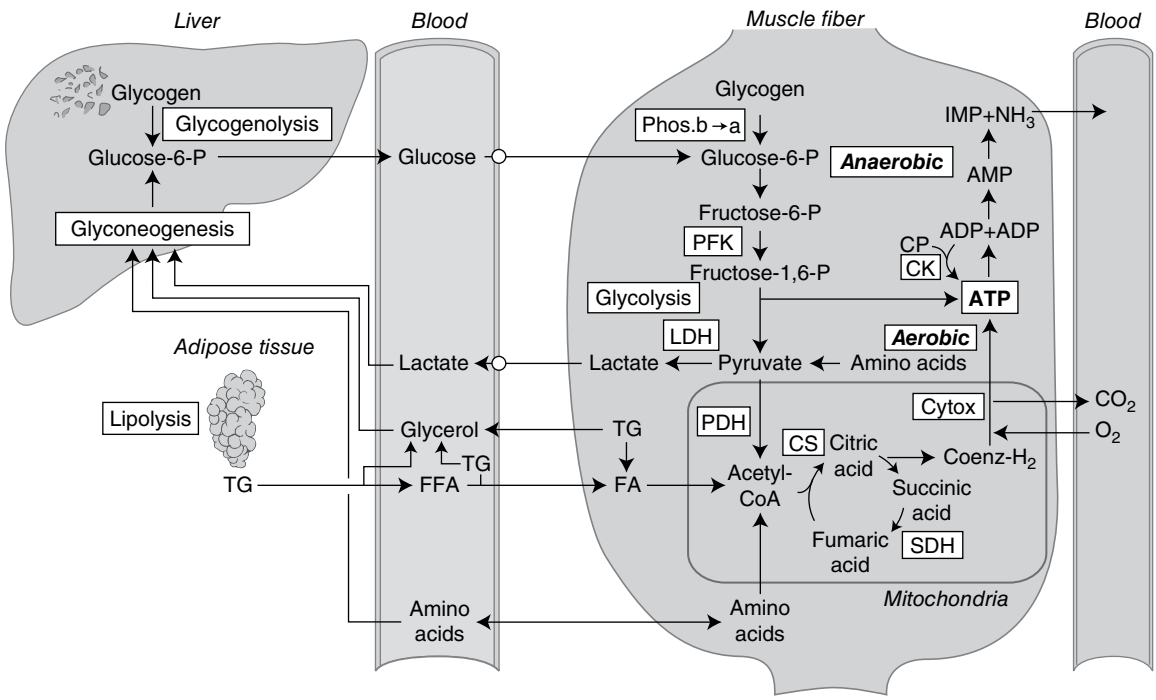

Figure 1.5 Integration of substrate metabolism across different tissues. ADP, adenosine diphosphate; AMP, adenosine monophosphate; ATP, adenosine triphosphate; CK, creatine kinase; CP, creatine phosphate; CS, citrate synthase; Cytox, cytochrome oxidase; FA, fatty acids; FFA, free fatty acids; IMP, inosine monophosphate; LDH, lactate dehydrogenase; NH3, ammonia; PDH, pyruvate dehydrogenase; PFK, phosphofructokinase; Phos., phosphorylase; SDH, succinate dehydrogenase; TG, triglycerides. Source: Bangsbo (1994). Reproduced with permission of John Wiley & Sons.

By definition, oxygen is required in the final series of aerobic metabolic reactions, combining with hydrogen ions to produce water. Oxygen is extracted from inspired air by the lungs, where it binds to hemoglobin and is transported to the tissues by red cells within the bloodstream. One measure of an athlete's fitness is his/her ability to utilize oxygen to efficiently produce ATP. This capacity is commonly known as the maximal oxygen uptake, or VO_2max. Although respiration is the principal source of oxygen utilized during aerobic metabolism, it is important to note that skeletal muscle does store a small amount of oxygen bound to myoglobin, a heme-containing molecule that can provide an immediately available source of oxygen in the absence of adequate respiratory oxygen delivery.

Fatigue and recovery in multiple sprint sports

Fatigue is an inevitable consequence of sufficiently intense or prolonged exercise. Many factors contribute to fatigue and in all but a few situations, it is probably futile to look for a single cause of fatigue. Possible causes of fatigue during high-intensity exercise include:

- phosphocreatine depletion
- decrease in pH
- glycogen depletion
- electrolyte imbalance
- central nervous system effects.

Each of these factors may be responsible for limiting exercise performance in specific situations, and some of these have practical implication for the athlete seeking to maximize performance (Mohr *et al*. 1996).

In the early 1990s it was shown that short periods of diet supplementation with creatine could increase muscle phosphocreatine stores and enhance performance in high-intensity sprints (Birch *et al*. 1994). Creatine enhances performance in activities lasting less than about 5 minutes, particularly when performing repeated sprints with short recovery periods (Casey *et al*. 1996). There is also some evidence that a few days of creatine supplementation can promote increases in muscle strength.

The acidosis that results from a high rate of anaerobic glycolysis can be countered by ingestion of buffering agents. Bicarbonate and citrate act as extracellular buffers, promoting the efflux of hydrogen ions from the cells where they are produced into the extracellular space and thus allowing more lactate (and hence ATP) to be produced before the pH becomes limiting. Ingestion of β-alanine can increase the intramuscular concentration of the dipeptide carnosine, and this in turn can increase intracellular buffering capacity and enhance sprint performance.

Glycogen availability per se is not usually considered to be responsible for fatigue during short-term high-intensity exercise, provided that the preexercise glycogen store is not less than 25 mmol/kgww. However, some scientists have suggested that the critical level of muscle glycogen concentration below which impairment of anaerobic ATP resynthesis occurs is somewhat higher than this, at about 45 mmol/kgww. It is possible that glycogen unavailability limits performance during repeated bouts of high-intensity exercise if performed for a prolonged period. Note, however, that this effect depends to a large degree on the extent of the decline in the rate of glycogenolysis and lactate production that occurs under these conditions. As described earlier, the initial muscle glycogen level of soccer players influences their performance (particularly in the second half of a game). A similar message comes from a study of ice hockey players who raised their preexercise muscle glycogen content by 12% after dietary carbohydrate loading before competition. The group of players who glycogen-loaded covered greater distances during the game, and at faster average speeds than the control group that did not carbohydrate load.

Furthermore, in recent years, several studies have documented beneficial effects of ingesting carbohydrate solutions on soccer and tennis performance. It has been observed that a majority of goals in soccer are scored towards the end of matches. This may occur due to a reduction in the work rate of the defenders or because of mental fatigue, leading to lapses in concentration and deterioration in skill. As blood glucose concentration does not decline during soccer-specific exercise

protocols, it can be concluded that carbohydrate ingestion does not improve endurance performance and execution of skills in soccer by preventing the development of hypoglycemia.

According to time-motion analyses and performance measures during match-play, fatigue or reduced performance seems to occur at three different stages in a soccer match: after short-term intense periods in both halves; in the initial phase of the second half; and towards the end of the match. Temporary fatigue after periods of intense exercise does not appear to be linked directly to muscle glycogen concentration, lactate accumulation, acidity or the breakdown of PCr (Krustrup *et al*. 2006). Instead, it may be related to disturbances in muscle ion homeostasis and an impaired excitation of the sarcolemma.

Soccer players' inability to perform maximally in the initial phase of the second half may be due to lower muscle temperatures compared with the end of the first half. Thus, when players perform low-intensity activities in the interval between the two halves, both muscle temperature and performance are better preserved. Muscle glycogen is typically reduced by 40–90% during a game and is probably the most important substrate for energy production. Even when whole muscle glycogen may appear adequate, fatigue toward the end of a match might be related to depletion of glycogen in some individual muscle fibers that have been active during the game. In one study, it was found that whole muscle glycogen decreased by about 43% during a soccer match, but that almost half of the muscle fibers were completely or nearly devoid of glycogen after the contest.

A group of Swedish professional soccer players was studied after playing a midweek game and preparing for another match on the Saturday (Table 1.3). One part of the group was fed a high-carbohydrate (CHO) diet for the few days between the games and the other part of the group consumed their normal diet, which had a relatively low CHO content. Muscle biopsies were taken from the thigh muscles of the players before the Saturday game, at half-time, and at the end of the match. Video analysis of the match was used to measure the distance covered by each of the players during the contest. The fraction of the total distance covered at sprinting speed and walking speed was also determined, the remaining distance being covered at an intermediate speed. The high CHO group had higher muscle glycogen stores at the start of the game, and at the end of the game still had some muscle glycogen left, whereas the control group had none. The total distance covered by the two groups of players in the first half was not significantly different but in the second half, when muscle glycogen concentration was lower, the players on the lower CHO diet were not able to run as far. The high CHO group covered more distance at sprinting speed (24% of a total distance of 12.0 km) and spent less time walking compared with the other group (see Table 1.3) (Jacobs *et al*. 1982).

At rest, sodium concentration inside cells is low and potassium concentration is high, while the situation is reversed in the extracellular environment. When muscles and nerves are activated, sodium enters the cells and potassium exits. In high-intensity exercise, the transmembrane gradients for potassium and sodium in the active muscles fall, causing the membrane potential to fall. This can impair development and propagation of the action potential, but there is little evidence to show failure. The metabolic acidosis that results from high rates of glycolysis helps counter this effect by maintaining membrane excitability.

Table 1.3 Muscle glycogen concentration and distance covered during the first and second half of a soccer match.

Muscle glycogen concentration (g/kg muscle ww)			Distance covered (km)			% Distance covered	
Before	**Half-time**	**End**	**1st half**	**2nd half**	**Total**	**Walking**	**Sprinting**
15	4	1	6.1	5.9	12.0	27	24
7	1	0	5.6	4.1	9.7	50	15