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Accurate indications, adverse reactions, and dosage schedules for drugs are provided in this book, but it is possible they may change. The reader is urged to review the package information data of the manufacturers of the medications mentioned.

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Humans have used many substances to enhance their athletic performance. As early as 200 to 300 A.D., Greek athletes and Nordic Berserkers ingested psychotropic mushrooms and herbs before competition and combat. In the 19th century, caffeine, alcohol, nitroglycerin, ethyl ether, strychnine, and opium were commonly used by athletes. In 1865, swimmers in a canal race in Amsterdam were charged with using ergogenic aids. Cyclists used "speedballs" of heroine and cocaine in 1869. In the late 19th century, a mixture of coca leaf extract and wine, called *vin Mariani* was used by French cyclists and a champion lacrosse team. Amphetamines replaced other stimulants in the competitive arena in the 1940s and 1950s.

In 1962, the International Olympic Committee (IOC) established a committee to control the use and abuse of drugs in athletics. In the 1968 Olympic games, ergogenic drugs were banned and drug testing began. The first person to be disqualified was Hans Gunnar Ljenvall, a Swedish pentathlete who took alcohol to steady his trigger finger. In the late 1960s and early 1970s, there was an increase in the documentation of amphetamine abuse, especially in professional sports. The use of drugs in professional baseball was prohibited in 1971 by then baseball commissioner, Bowie Kuhn. During the 1980s, human growth hormone and human chorionic gonadotropin entered the world of ergogenic aids. During the 1983 Pan-American Games in Caracas, Venezuela, 19 athletes, two from the United States, tested positive for banned substances. A number of other U.S. athletes returned home before competition and drug testing. In 1984, during the XXIII Olympiad in Los Angeles, four U.S. cyclists used blood doping and won four of the team's nine medals.

**DRUGS IN SPORTS**

The use of drugs falls into three general categories: therapeutic, performance enhancing, and recreational. Use of antibiotics for the treatment of an infection is an example of a therapeutic drug. The performance-enhancing drugs, or ergogenic aids, include drugs such as amphetamines and anabolic steroids (Table 15-1). Recreational drugs include alcohol, marijuana, and other mood-altering substances.

The term *ergogenic* comes from the Greek *ergon* (work) and *genan* (to produce). Ergogenic aids increase work output. The American College of Sports Medicine's position statement in 1987 defined ergogenic aids as "physical, mechanical, nutritional, psychological, or pharmacological substances or treatments that either directly improve physiological variables associated with exercise performance or remove subjective restraints that may limit physiological capacity."

The term *doping* originated in South Africa, where dope was the name of an alcoholic beverage used by the native population. According to the International Olympic Committee (IOC), doping is "the administration or use of any substance foreign to the body or any physiological substance taken in abnormal quantity or taken by an abnormal route of entry into the body with the sole intention of increasing in artificial and unfair manner . . . performance in competition. When necessity demands, medical treatment with any substance which, because of its nature, dosage or application, is able to boost the athlete's performance in competition in an artificial and unfair manner, this too is regarded by the IOC as doping."

The IOC has divided the various types of drugs into doping *classes*. It should be noted that the doping...
### Table 15-1. Performance-Enhancing Drugs: Ergogenic Aids

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanisms of Action</th>
<th>Enhancing Effects</th>
<th>Adverse Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amphetamines and sympathomimetics</td>
<td>Stimulate release of catecholamines from nerve cells; displace catecholamines from receptor sites, allowing an increased amount of catecholamines in the synaptic cleft; inhibit reuptake; act as catecholamine agonist; inhibit catecholamine breakdown</td>
<td>Increased alertness, increased self-confidence, elation, euphoria; release of serum free fatty acids; decreased reaction time, appetite; fatigability, mood elevation</td>
<td>Central nervous system (CNS): Tremulousness, anxiety, insomnia, agitation, dizziness, irritability, headaches, psychosis, seizures, possible cerebrovascular accident (CVA) Cardiovascular (CV): Arrhythmias, hypertension, angina pectoris Gastrointestinal (GI): Nausea, vomiting, diarrhea, dry mouth, hyperthermia</td>
</tr>
<tr>
<td>Caffeine</td>
<td>Translocation of intracellular calcium; increase available cAMP; competitive antagonist of adenosine receptors; glycogen sparing by increasing free fatty acid availability</td>
<td>Decreased fatigue; increased concentration and alertness, endurance, muscle contractility, performance</td>
<td>CNS: Anxiety, nervousness, insomnia, delirium, seizures, coma, death CV: Tachycardia, arrhythmias, hypertension Genitourinary (GU): Diuresis</td>
</tr>
<tr>
<td>Cocaine</td>
<td>Increased concentrations of dopaminergic and noradrenergic transmitters at the neural synapse; blocks reuptake antagonism</td>
<td>Euphoria; sense of enhanced mental prowess</td>
<td>CNS: Addiction, CVA, seizures, visual changes, insomnia, confusion, delirium, paranoia, psychosis CV: Ventricular arrhythmia, angina pectoris, myocardial infarction, myocarditis, sudden death Other: Perforation of nasal septum, loss of smell, hyperthermia</td>
</tr>
<tr>
<td>Nicotine (tobacco)</td>
<td>Stimulate of CNS at low doses, depression of CNS (inhibition of catecholamine release) at high doses</td>
<td>CNS stimulation at low doses, decreased appetite, calming effect at high doses</td>
<td>CNS: Depresion at high doses Respiratory (R): Chronic obstructive pulmonary disease, lung cancer CV: Hypertension, coronary artery disease Other: Periordial disease</td>
</tr>
<tr>
<td>Beta blockers</td>
<td>Block beta receptors on end organs β₁: Heart, kidney, adipose tissue β₂: Liver, bronchi, and arteries</td>
<td>Relieving anxiety, decreased tremor and heart rate, improved hand-arm steadiness</td>
<td>CNS: Hallucinations, nightmares, insomnia, depression R: Increased airway resistance CV: Reduced blood pressure, congestive heart failure, retarded heart rate, atrioventricular block GE: Nausea, vomiting, diarrhea, constipation</td>
</tr>
<tr>
<td>Diuretics</td>
<td>Type: Osmotic diuresis; carbonic anhydrase inhibitor; increased concentration of Na⁺ in urine; inhibition of electrolyte reseption; aldosterone antagonism</td>
<td>Weight reduction; dilution of drug concentration in urine</td>
<td>Dehydration, electrolyte imbalance, muscle cramps, orthostatic hypotension; decreased muscle strength, cardiac output, VO₂ max; poor temperature regulation Infection—viral (hepatitis or HIV) or bacterial Immune reactions: Fever, urticaria, hemolytic anemia, Fatal transfusion reaction CV: Increased blood viscosity; decreased blood flow velocity; pulmonary emboli, deep venous thrombosis</td>
</tr>
<tr>
<td>Blood doping and erythropoietin</td>
<td>Increased O₂ delivery to skeletal muscles via increased hemoglobin and hematocrit</td>
<td>Increased aerobic work, enhanced thermal regulation; increased cardiac output secondary to increased blood volume; increased buffering of lactic acid</td>
<td>Nutritional imbalance; vitaminosis; GI upset</td>
</tr>
</tbody>
</table>

### Table 15-2. Nutritional Ergogenic Aids

<table>
<thead>
<tr>
<th>Nutritional Ergogenic Aids</th>
<th>Benefits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amino acids</td>
<td>Nourish muscles</td>
</tr>
<tr>
<td>Vitamin B₆, bee pollen, sodium, bicarbonate, baking powder, vitamin and minerals</td>
<td>Regulate metabolism of sugars, amino acids, and vitamins; maintain connective tissue and cartilage; promote skin, hair, and nail health; enhance immune function</td>
</tr>
</tbody>
</table>
definition is based on the banning of pharmacological classes of agents and not specific agents. The doping method refers to blood doping as well as other pharmacological, chemical, and physical manipulation of the urine. A third class of drugs that is subject to certain restrictions includes alcohol, marijuana, local anesthetics, corticosteroids, and beta blockers. The doping classes are stimulants, narcotics, anabolic agents, diuretics, and peptide hormones and analogs.

**STIMULANTS**

Stimulants are the most commonly used ergogenic aid. The most common type is amphetamines (Table 15-2). Other stimulants include caffeine, nicotine, cocaine, crack cocaine, and over-the-counter sympathomimetic agents such as ephedrine. Stimulants have been most commonly used in events that require endurance, such as cycling, and in sports that require aggressiveness and explosive power. The psychological effects of stimulants include enhanced alertness, increased ability to concentrate, decreased sensation of fatigue, mood elevation, and increased self-confidence and aggression. Stimulants also increase the musculoskeletal system’s ability to improve muscle contractility and increase the release into the circulation of free fatty acids. Negative physiologic effects include anxiety, poor judgment, excessive aggressiveness, schizophrenia-like psychoses, increased heart rate, increased blood pressure, risk for cerebral vascular accident, cardiac arrhythmias, death, and interference with timing of technical skills.

**Amphetamines**

In the 1960s there was an increase in the use of amphetamines among American professional football players. In 1957, the American Medical Association condemned the use of amphetamines in athletics. A controlled substance act was passed by Congress in the 1970s that severely restricted the manufacturing of amphetamines and applied strict guidelines for their use. The therapeutic uses of amphetamines have included the treatment of obesity, narcolepsy, minimal brain dysfunction (hyperkinesis), attention deficit disorder, depression, and severe menstrual cramps.

Amphetamines, sympathomimetic agents, mimic the endogenous catecholamines epinephrine, norepinephrine, and dopamine. Catecholamines stimulate the central and peripheral nervous system via the alpha and beta receptors. The stimulation of the central and peripheral nervous system by amphetamines is by an indirect method. The cardiovascular effects of amphetamines include increased systolic and diastolic blood pressure, increased heart rate, and, with larger doses, reduced positive inotropic effect (via reflex action). Central nervous system effects of amphetamines include stimulation of the medullary respiratory center, the spinal cord, and the reticuloendothelial system. However, the most significant effect of amphetamines appears to be its psychological effects: increased alertness, decreased sense of fatigue, mood elevation, increased self-confidence, elation, and euphoria.

The ergogenic effects of amphetamines were the subject of a controlled study conducted by Smith and Beecher in 1959. They evaluated swimmers, runners, and weight-throwing performers who were all trained athletes. Each subject was given a 14 mg/kg dose of amphetamine 2 to 3 hours before athletic performance. Better performance was noted in 93% of the swimmers, 73% of the runners, and 85% of the weight throwers. Although the percentage of increase in their respective fields ranged from a low of 0.5% for swimmers to a high of 4% for weight throwers, this difference may be significant for a high-class or world-class athlete. Other studies have shown that amphetamines do not produce a positive effect. The psychological benefit from amphetamines appears to be the most significant one. The numerous side effects must be weighed against the known benefits.

The side effects of amphetamine use depend on dose and length of use. The adverse effects on the central nervous system include restlessness, insomnia, instability, agitation, confusion, paranoia, hallucinations, con-

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
<th>Street Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amphetamine</td>
<td>Benzedrine</td>
<td>Uppers, bennies, peaches, greenies</td>
</tr>
<tr>
<td>Dextroamphetamine</td>
<td>Dexedrine</td>
<td>Dexies, oranges, greenies, orange heart caplets</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>Desoxynmethapex</td>
<td>Meth, crystal, whites, speed</td>
</tr>
<tr>
<td>Dextroamphetamine and amphetamine</td>
<td>Biphetamine</td>
<td>Footballs, black beauties</td>
</tr>
</tbody>
</table>

The central nervous system effects of caffeine include anxiety, nervousness, insomnia, delirium, seizures, coma, and in larger doses, death. Withdrawal from long-term caffeine use can result in headaches, drowsiness, lethargy, rhinorrhea, irritability, nervousness, and depression. Cardiovascular side effects of increased caffeine ingestion include palpitations, hypertension, and arrhythmias (supraventricular and ventricular). Another complication is that the mild diuretic effect of caffeine could offset the performance-enhancing effect for endurance athletes. Heat problems can occur with the reduced plasma volume and increased basal metabolic rate. Mild gastrointestinal irritation can also occur with excessive doses of caffeine.

Cocaine

Cocaine comes from the coca bush (Erythroxylon coca), an indigenous plant in the Peruvian Andes. The coca leaf was originally chewed by the Incas. It was used for religious purposes and later became an abused drug. Its benefits were to fight off fatigue and suppress hunger. Sigmund Freud also performed studies using cocaine on himself and felt exhilarated and at ease. Cocaine was also used by Angelo Mariani, a Corsican chemist, who added it to wine ("vin Mariani"), which was widely used by cyclists. In the United States, John Smythe Pemberton used cocaine in the original formula of Coca-Cola; however, it was removed from the formulation in 1903. In 1986, cocaine caused the deaths of professional football player Don Rogers, of the Cleveland Browns, and amateur basketball player Len Bias, from the University of Maryland.

Cocaine and crack cocaine use by athletes has been principally recreational rather than for ergogenic effect. In the general population, one in every six high school seniors has admitted to trying cocaine, and nearly 48% of adults in their late 20s have used cocaine. A 1986 National Football League (NFL) Players Association survey found that half of the respondents considered cocaine the most commonly abused drug in the NFL.

The principal medical use for cocaine today is as an anesthetic. It is used principally in ear, nose, and throat procedures. The methods of use of cocaine include sniffling (snorting), smoking, chewing, and intravenous injection. The drug is usually snorted by illicit users. Cocaine works primarily by blocking reuptake of neurotransmitters. The effects of cocaine are short lived. Since the exhilarating effects last only 5 to 15 minutes, it is not uncommon for users to take multiple doses during the day. Because of its short-lived action, cocaine is a poor ergogenic aid. Unfortunately, cocaine is a very addictive drug. It is no longer considered "the safe drug." Side effects of cocaine include ulceration and perforations of the nasal septum, rhinitis, sinusitis, bronchitis, hyperthermia, agitation, restlessness, insomnia,
Nicotine

Nicotine, the addictive drug found in tobacco, has been used by humans for centuries. Tobacco can be smoked or used in smokeless forms. Smokeless tobacco can be further divided into loose-leaf tobacco (chewing tobacco) and snuff. The use of smoking tobacco in the general population in the United States has been reported at approximately 31.5% of adult men and 25.7% of the adult women. Nicotine use in the athlete population is principally by smokeless tobacco, usually by baseball players. It is hoped that current educational programs and rule changes will reduce the use of chewing tobacco during competition. It has been estimated that 34 to 39% of the U.S. professional baseball players in major and minor leagues use smokeless tobacco.

Nicotine is a potent alkaloid that works on both the central and the peripheral nervous system. It also acts as both a depressant and a stimulant. The effect is dose dependent. At low doses, in the peripheral nervous system, stimulation occurs at the autonomic ganglia. At high doses, ganglionic depression occurs. Also at high doses, nicotine causes inhibition of catecholamine release from the adrenal medulla. In the central nervous system, norepinephrine and dopamine release occurs after nicotine administration. Tobacco appears to have paradoxical calming and stimulating effects because of the aforementioned dose-related effects of nicotine.

The athlete uses nicotine for the stimulatory effect, calming effect, or appetite control. The overwhelming side effects of the nicotine in tobacco far outweigh the benefits. The side effects of tobacco include pulmonary diseases, including various carcinomas; cardiovascular disease, including hypertension and coronary artery disease; gastrointestinal disease; and periodontal diseases, including leukoplakia and the risk of squamous cell carcinoma and others.

Although the benefits as an ergogenic aid to performance are small, the long-term risks of ill health far outweigh any possible benefit from nicotine. Nicotine is classed as a stimulant. It is not specifically listed in the IOC banned substance list. The rules of the NCAA and professional baseball are becoming more restrictive on the use of tobacco.

Sympathomimetics

Sympathomimetic amines are another group of agents that are principally stimulants. The sympathomimetics stimulate the sympathetic nervous system via $\alpha_1$- or $\alpha_2$- and $\beta_1$- or $\beta_2$-receptor stimulation. Examples of the sympathomimetic amines include phenylephrine, ephedrine, and pseudoephedrine. These agents are commonly found in over-the-counter cold, decongestant, and asthma preparations (Table 15–3). The more selective the specific agent is for either alpha or beta stimulation, the more specific is the therapeutic benefit gained. For example, the $\beta_2$-agonists salbutamol and terbutaline are the only approved $\beta_2$-agonists for the treatment of asthma in the inhaled form. Not only are these the only two $\beta_2$-agonists approved by the United States Olympic Committee (USOC) and IOC, but prior written notification of the use of these agents must be received by the USOC and the IOC. Because of the ubiquitous nature of these agents in common cold remedies, appetite suppressants, and nasal decongestants, the use of sympathomimetic amines is common. Although there have been no specific studies that show an obvious ergogenic benefit for the sympathomimetics like ephedrine or phenylephrine, the potential effects are still a concern. The adverse cardiovascular effects include increased blood pressure, (including life-threatening hypertensive episodes), cardiac arrhythmias, palpitations, and myocardial infarction. Central nervous system effects of these agents include nervousness, irritability, insomnia, dizziness, cephalgia, anorexia, agitation, confusion, paranoia, mania, hallucinations, stroke, cerebral vasculitis, and cerebral hemorrhage.

β-ADRENERGIC BLOCKING AGENTS

“Beta blockers” are therapeutic agents for hypertension, angina, and specific cardiac arrhythmias. Other

Table 15–3. USOC/IOC-Banned Cold and Asthma Preparations

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ephedrine</td>
<td>Teldral, Bronkaid, Rynuass, Primatene,</td>
</tr>
<tr>
<td></td>
<td>Bronkaid, Nyquil Nighttime Cold</td>
</tr>
<tr>
<td>Pseudoephedrine</td>
<td>AciTed, Afrin tablets, Afrinol, Coas-Tynol.</td>
</tr>
<tr>
<td></td>
<td>Deconamine, Novadex, Sudafed, Chlor-Trimeton-DC, Drixoral and related compounds</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>Dristan, Neo-Synephrine, Sinex, and related compounds</td>
</tr>
<tr>
<td>Desoxycodonephedrine</td>
<td>Vicks inhaler and related compounds</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>Alka-Seltzer Plus, Allerest, Contact</td>
</tr>
<tr>
<td></td>
<td>Dextramine, Dieac, Sine-Aid, Sine-Off</td>
</tr>
<tr>
<td></td>
<td>Sinusafe, Triaminic, Sterex Cold</td>
</tr>
<tr>
<td></td>
<td>Decongestant, and related compounds</td>
</tr>
<tr>
<td>Isoeugenol HCl</td>
<td>Bronkosei, Bronkometer, Nupotec,</td>
</tr>
<tr>
<td></td>
<td>Dilabron, and related compounds</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>Isuprel, Norisodrine, Metolol-ISO, and related compounds</td>
</tr>
<tr>
<td>Metaproterenol</td>
<td>Alupent, Metaple, and related compounds</td>
</tr>
<tr>
<td>Methoxyphenamine</td>
<td>Ritalin, Ortiofloxol cough syrup, and related compounds</td>
</tr>
<tr>
<td>Methylphenidate HCl</td>
<td>Ritalin and related compounds</td>
</tr>
</tbody>
</table>

uses for β blockers include the treatment of migraines, headaches, essential tremors, overactivity, pheochromocytoma, thyroid toxicosis, and alcohol withdrawal. Beta blockers are agents that block β-adrenergic receptors on end organs. The β₁ receptors are found principally in heart, kidneys, and adipose tissue, and β₂ receptors in the liver, bronchi, and arteries. Beta-blocking agents, therefore, can be specific or nonspecific. Nonspecific agents block both the β₁ and β₂ receptor sites, whereas specific β blockers can block either the β₁ or the β₂ receptor sites. Although there is no pure β₁ or β₂ blocking without overlap, the agents principally affect either the β₁ or β₂ receptor sites. [15]

The most common β-blocking agent used has been propranolol (Inderal), a nonselective beta blocker. [32] Performance-enhancing effects of β blockers are relief of anxiety, decreased tremor, lower heart rate, a general calming effect, and improved hand-arm steadiness. Because of these potential ergogenic effects of using β blockers, the winter game sports of biathlon, bobsled, luge, and ski jumping, and summer game sports of archery, diving, equestrian, fencing, gymnastics, modern pentathlon, sailing, and shooting, have all been targeted by the IOC for β blocker abuse. [19] In events where shooting is involved, the athlete shoots between heartbeats, and β blockers provide the shooter more time to steady his aim between heartbeats. [33] Kruse and coworkers have shown a 13% improvement in pistol shooters using β blockers. [34]

β blockers decrease Vo₂max by 15% or greater and decrease muscle blood flow, muscle oxygen uptake, and blood glucose concentrations. The adverse effects of β blockers include increased airway resistance, nausea, vomiting, mild diarrhea, constipation, hallucinations, nightmares, insomnia, depression, shortened time to fatigue, decreased ability to perform endurance-type activities, hypotension, congestive heart failure, bradycardia, and atrioventricular block. Beta blockers are banned by the National Collegiate Athletic Association (NCAA), IOC, and USOC.

NARCOTICS

Pain can be controlled by narcotics or nonsteroidal anti-inflammatory agents. The latter are legal but should be declared by the tested athlete. The use of narcotic analgesics has been banned by the USOC and the IOC but is not restricted by the NCAA. Use of narcotics may mask symptoms of potentially severe injury or result in addiction, false feelings of invincibility, delusions of athletic prowess, and poor perception of dangerous situations that place the athlete and others at risk. No definitive studies have shown ergogenic effects for narcotics.

DIURETICS

Athletes use diuretics for two effects: rapid weight loss and urine dilution. In sports with weight categories, such as boxing, wrestling, judo, and equestrian, diuretic use is common. Weight loss potential using diuretics has been documented at 4.1% weight reduction over a 24-hour period. Reduction of the concentration of drugs in the urine with diuretics occurs because of more rapid excretion. Diuretics are banned by most governing bodies of athletic events. [9] Negative effects of diuretics are decreased Vo₂max, decreased work load to maximal exercise, and changes in blood lactate concentration. [34]

Other adverse effects include dehydration, hypovolemia, muscle cramps, orthostatic hypotension, electrolyte imbalance, fatigue, and precipitation of gout. [13,34]

BLOOD DOPING AND ERYTHROPOIETIN

Blood Doping

Blood doping is also known as blood boosting, induced erythrocythemia, or blood packing. The definition of blood doping used by the USOC is "the administration of blood or related blood products, including erythropoietin, to an athlete other than for a legitimate medical treatment. This procedure may be preceded by the withdrawal of blood from the athlete, who continues to train in this blood-depleted state." [40] The use of blood doping goes back to the end of World War II, when attempts were made to enable pilots to avoid the adverse effects of high altitude. [35] In the athletic arena it was rumored that blood doping may have been used in 1972, during the Munich games, as well as in the 1976 Montreal games. In the 1984 summer Olympics in Los Angeles, seven U.S. cyclists (four gold medalists) admitted to using blood doping. [11]

The theory behind the use of blood doping is that it increases oxygen delivery to working muscles and increases the capacity for aerobic work, provided that oxygen delivery is the rate-limiting factor and cardiac output and blood distribution are not adversely affected by increased viscosity. Blood doping is also believed to enhance thermal regulation, buffer the inhibitory effect of lactic acid on skeletal muscles cells, and augment cardiac output secondary to increased blood volume and preload. [25] Results documented by Ekblom demonstrated a maximum aerobic power increase of an average of 10%, and this lasted approximately 18 days after retransfusion. [36] Others have shown an increase in Vo₂max by as much as 3.9 to 12.8% and an increase in endurance capacity from 2.5 to 35%. [19]

The actual technique for blood doping starts anywhere from 4 to 8 weeks before competition. Two units of blood are removed from the athlete and the athlete continues to train. The reinfusion takes approximately 1 to 2 hours and the best benefits are obtained within the next 24 hours to a week. [11,25]

Adverse effects of blood doping exist with either autologous or homologous blood transfusion techniques. Homologous transfusions carry the risk of infectious diseases such as hepatitis and AIDS. [30] Approximately 3% of all homologous blood transfusions cause immune reactions, including mild allergic reactions, fever, urticaria, and hemolytic transfusion reactions.
which can be fatal. Homologous and autologous blood transfusions carry the risks of elevated blood viscosity. Increased blood viscosity can lead to decreased cardiac output, decreased blood flow velocity, and decreased peripheral blood oxygen concentration, resulting in reduced aerobic capacity. Blood clots, deep venous thrombosis, and pulmonary emboli are also potential side effects. Most of these transfusion reactions of hyperviscosity are related to hematocrit values greater than 50 to 60%.\(^{37}\)

**Erythropoietin**

Erythropoietin, in blood doping, is used primarily in the athletic population to increase the hematocrit of the athlete’s blood. Erythropoietin is a hormone that is naturally produced in the human kidney. It stimulates bone marrow stem cells to differentiate into red blood cells. It has been shown to increase red blood cell mass as well as hemoglobin and hematocrit.\(^{38}\) Presently, erythropoietin is genetically engineered using recombinant gene technology.\(^{39}\) The effects of long-term recombinant erythropoietin administration on healthy persons has not been determined, but stroke and renal failure are known complications.

Knowing the results of blood-doping studies and the effects of erythropoietin, it would then appear that erythropoietin could be used as an ergogenic aid. Erythropoietin and blood doping are indetectable by present testing methods. Erythropoietin and blood doping are both banned by the NCAA, the USOC, and the IOC.\(^{39,41}\)

**NUTRITIONAL ERGOGENIC AIDS**

History documents use of such ergogenic aids as honey, bee pollen, wheat germ oil, and other natural substances.\(^{40}\) *Nutrients* are proteins, fats, carbohydrates, and vitamins and minerals. These nutrients provide energy, maintain growth and development of the tissues, and regulate metabolic enzymatic processes. The scientific literature is equivocal about the uses of various nutritional supplements and foods as ergogenic aids. Some nutritional ergogenic aids like teas and herbal medications may actually contain stimulants detectable in the urine and subsequently disqualify an athlete. This has, however, not prohibited commercial entities from using various data to support their beliefs.

**Amino Acids**

The amino acids arginine and ornithine have been marketed as agents that increase muscle development, decrease body fat, and increase human growth hormone levels.\(^{41}\) L-Tryptophan has been touted as being able to raise growth hormone levels, enhance performance, and relieve depression and insomnia.\(^{42}\)

The possible adverse consequences of using amino acids and protein powders include dehydration, gout, calcium loss, and increased urea production.\(^{40}\) There is no overwhelming evidence that protein powders or amino acids in supplemental form provide any benefit over those of a balanced adequate diet.

**Vitamin B₁₃**

Vitamin B₁₃ (pangamic acid), though not a true vitamin, has been theorized to enhance aerobic endurance performance by improving oxidative metabolism by enzyme stimulation of succinate dehydrogenase and cytochrome oxidase. The evidence to date does not support vitamin B₁₃ as an ergogenic aid.\(^{40}\)

**Bee Pollen**

Bee pollen, a mixture of microspores from flowers and nectar from the beehive, has historically been considered an ergogenic aid. Some believe that it may help the athlete recover faster during workouts. Available scientific evidence does not support bee pollen as a true ergogenic aid.\(^{40}\)

**Soda Doping**

The use of sodium bicarbonate or baking soda to increase the normal alkaline reserve of the body has been termed soda doping or buffer boosting.\(^{35,40}\) In theory, fatigue may be reduced. By increasing pH by soda loading, lactic acid production may be reduced.

This would be most beneficial for athletes who use anaerobic metabolism, such as sprinters, as opposed to aerobic metabolism, such as endurance athletes. A decrease in subjective fatigue ratings and perceived exertion during exercise has been reported. Complications of soda doping are principally gastrointestinal, like gastrointestinal upset and diarrhea.

**General Nutrition**

The best advice is sound nutrition in all phases: training phase, performance, and recovery. Unlike pharmacologic ergogenic aids, no one specific nutritional source has been shown to enhance performance.

**CONCLUSION**

"Higher, faster, stronger." Although these are the words of the Olympic motto, they by no means reflect or condone achieving these goals by any means, including ergogenic aids. The use of ergogenic aids is evident at all levels of competition, including high school, college, and international competition. Our roles as health care givers to athletes is one of education, support, and providing quality health care and safety. Although not paramount in the minds of present-day athletes, The important thing is that the Olympic games is not to win but to take
part; the important thing in life is not the triumph but the struggle. The essential thing is not to have conquered but to have fought well," as stated in 1908 by the founder of the modern Olympic games Baron Pierre de Coubertin. This spirit should be the goal.

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6201 College Boulevard
Overland Park, KS 66211-2422
(913) 339-1906

National Federation of State High School Associations
11724 Plaza Circle
Kansas City, MO 64195
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United States Olympic Committee Drug Control Hotline
1750 East Boulder Street
Colorado Springs, CO 80909
(800) 233-0393