THE HUGHSTON CLINIC

Sports Medicine Book

Champ L. Baker, Jr., M.D., Editor-in-Chief

Fred Flandry, M.D., Section Editor

John M. Henderson, D.O., Section Editor

Hughston Sports Medicine Foundation, Inc.

Williams & Wilkins

BALTIMORE • PHILADELPHIA • HONG KONG
LONDON • MUNICH • SYDNEY • TOKYO

A WAVERLY COMPANY

1995

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Printed in the United States of America

Library of Congress Cataloging in Publication Data
The Hughston Clinic sports medicine book / [edited by] Champ L. Baker.
p. cm.
"Hughston Sports Medicine Foundation, Inc."
Includes index.
ISBN 0-683-00397-6
1. Sports medicine. I. Baker, Champ L. II. Hughston Orthopaedic Clinic. III. Hughston Sports Medicine Foundation. IV. Title: Sports medicine book.
[DNLM: 1. Sports Medicine. 2. Athletic Injuries. QT 260 H8945 1995]
RC1210.H84 1995
617.1'027—dc20
DNLM/DLC
for Library of Congress 94-23442
CIP

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1 2 3 4 5 6 7 8 9 10

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Mary Lloyd Ireland and Craig C. McKirgan

Performance-Enhancing Drugs: Ergogenic Aids

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.[1] In the 19th century, caffeine, alcohol, nitroglycerin, ethyl ether, strychnine, and opium were commonly used by athletes.[2] In 1865, swimmers in a canal race in Amsterdam were charged with using ergogenic aids.[3] Cyclists used "speedballs" of heroine and cocaine in 1869.[4] 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.[5] Amphetamines replaced other stimulants in the competitive arena in the 1940s and 1950s.[4]

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 Lijenvall, a Swedish pentathlete who took alcohol to steady his trigger finger.[6] 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.[7] During the 1980s, human growth hormone and human chorionic gonadotropin entered the world of ergogenic aids.[4] 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 *gennan* (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."[8]

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."[9]

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

Drug	*Mechanisms of Action*	*Enhancing Effects*	*Adverse Effects*
Amphetamines and sympathomimetics	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	Increased alertness, increased self-confidence, elation, euphoria release of serum free fatty acids; decreased reaction time, appetite; fatigability, mood elevation	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
Caffeine	Translocation of intracellular calcium; increase available cAMP; competitive antagonism of adenosine receptors; glycogen sparing by increasing free fatty acid availability	Decreased fatigue; increased concentration and alertness, endurance, muscle contractility, performance	CNS: Anxiety, nervousness, insomnia, delirium seizures, coma, death CV: Tachycardia, arrhythmias, hypertension Genitourinary (GU): Diuresis
Cocaine	Increased concentrations of dopaminergic and noradenergic transmitters at the neural synapse; blocks reuptake antagonism	Euphoria; sense of enhanced mental prowess	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
Nicotine (tobacco)	Stimulation of CNS at low doses, depression of CNS (inhibition of catecholamine release) at high doses	CNS stimulation at low doses, decreased appetite, calming effect at high doses	CNS: Depression at high doses Respiratory (R): Chronic obstructive pulmonary disease, lung cancer CV: Hypertension, coronary artery disease Other: Periodontal disease
Beta blockers	Block beta receptors on end organs β_1: Heart, kidney, adipose tissue β_2: Liver, bronchi, and arteries	Relieving anxiety, decreased tremor and heart rate, improved hand-arm steadiness	CNS: Hallucinations, nightmares, insomnia, depression R: Increased airway resistance CV: Reduced blood pressure, congestive heart failure, retarded heart rate, atrioventricular block GI: Nausea, vomiting, diarrhea, constipation Other: Decreased endurance due to reduced Vo_2max; decreased muscle blood flow, O_2 uptake, and glucose concentration
Diuretics	*Types:* Osmotic diuresis; carbonic anhydrase inhibitor; increased concentration of Na^+ in urine; inhibition of electrolyte resorption; aldosterone antagonism	Weight reduction; dilution of drug concentration in urine	Dehydration, electrolyte imbalance, muscle cramps, orthostatic hypotension; decreased muscle strength, cardiac output, Vo_2max; poor temperature regulation
Blood doping and erythropoietin	Increased O_2 delivery to skeletal muscles via increased hemoglobin and hematocrit	Increased aerobic work; enhanced thermal regulation; increased cardiac output secondary to increased blood volume; increased buffering of lactic acid	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
Nutritional ergogenic aids: Amino acids, vitamin B_{15}, bee pollen, sodium, bicarbonate, baking powder, vitamins and minerals		Psychological benefit	Nutritional imbalance; vitaminosis; GI upset

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.[9] 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.[4] 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.[10] The therapeutic uses of amphetamines have included the treatment of obesity, narcolepsy, minimal brain dysfunction (hyperkinesis), attention deficit disorder, depression, and severe menstrual cramps.[11,12]

Amphetamines, sympathomimetic agents, mimic the endogenous catecholamines epinephrine, norepinephrine, and dopamine.[13] 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.[14] 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 amphetamine use include stimulation of the medullary respiratory center, the spinal cord, and the reticuloendothelial system.[14] 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.[15] 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% difference for weight throwers, this difference may be significant for a high-class or world-class athlete. Other studies have shown that amphetamines do *no* produce a positive effect.[16] 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 15–2. Amphetamines

Generic Name	*Trade Name*	*Street Name*
Amphetamine	Benzedrine	Uppers, bennies, peaches, greenies
Dextroamphetamine	Dexedrine	Dexies, oranges, greenies, orange heart caplets
Methamphetamine	Desoxynmethempex	Meth, crystal, whites, speed
Dextroamphetamine and amphetamine	Biphetamine	Footballs, black beauties

(Brill H., and Hirose, T.: The rise and fall of a methamphetamine epidemic: Japan 1945–55. Seminars in Psychiatry. *1*(2):179, 1969. Laties, V.G., and Weiss, B.: The amphetamine margin in sports. Fed. Proc. *40*:2689, 1981.)

vulsions, coma, and even death. Cardiovascular effects include headaches, chills, flushing, palpitations, angina, atrial or ventricular arrhythmias, hypertension, hypotension, bradycardia, tachycardia, cardiovascular collapse, necrotizing vasculitis, subarachnoid hemorrhage, and cerebral hemorrhage in doses as low as 20 mg. Gastrointestinal effects include gastrointestinal discomfort, weight loss, nausea, vomiting, abdominal pain, and decreased appetite. The sudden withdrawal from amphetamines, especially from long-term use, can result in chronic fatigue, lethargy, hypersomnia, hyperphagia, and depression.

Caffeine

Caffeine is one of the most commonly used drugs in the United States and Europe. Caffeine is a methylated xanthine similar to theophylline and theobromine.[12] Caffeine is found in the raw fruit of the coffee plant *(Coffea arabica)* as well as in 60 other species of plants, including tea leaves and coconuts. Today, caffeine is found principally in beverages such as coffee, tea, and soft drinks, and in some over-the-counter analgesics, cold medications, and antisomnolence drugs. The concentration of caffeine in an 8-ounce cup of coffee is roughly 100 to 150 mg. Tea has approximately 60 to 70 mg of caffeine per 8 ounce, cola drinks 40 to 60 mg in 12 ounce.[11,12]

Historically, caffeine was reported in France to cure smallpox, gout, and scurvy and in England to cure venereal disease and the common cold.[17] Today, the principal use of caffeine as an ergogenic aid is to increase endurance and increase alertness.[12] Some studies substantiate the hypothesis that the ingestion of caffeine may spare muscle glycogen use by mobilizing serum-free fatty acids.[18,19] The glycogen stores are then available for use later. There has also been evidence of an increase in muscle contractility associated with doses of caffeine.[20] Another primary effect of caffeine is its central nervous system stimulation, which may improve endurance, especially if an athlete is already fatigued. Studies of Vo_2max, an indicator of aerobic capacity, have shown no great effects after caffeine ingestion.

Because of conflicting reports about the ergogenic effects of caffeine, the IOC has changed its ban on caffeine and now allows it to be ingested in small amounts. The IOC banned the use of caffeine until 1972. At that time, any caffeine found in the urine was considered a banned substance. Just before the 1972 Olympics caffeine was removed from the banned list. This action reflected conflicting findings and the ubiquitous nature of caffeine. In the 1984 Olympic games in Los Angeles, the IOC returned caffeine to the "banned substance list," but only *restricted* the permissible level. Concentrations of caffeine greater than 15 μg/ml of urine were banned. In 1986, the IOC lowered the permissible caffeine level in the urine to less than 12 μg/ml.[9] The NCAA banned caffeine in urine concentrations greater than 15 μg/ml.[21] To reach a 12 μg/ml level, a person would have to drink six to eight cups of coffee over a short time and be tested within 2 or 3 hours.

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.[13] 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.[13,22,23] Sigmund Freud also performed studies using cocaine on himself and felt exhilarated and at ease.[11,24] 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.[23] 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.[11]

Cocaine and crack cocaine use by athletes has been principally recreational rather than for ergogenic effect.[25,26] In the general population, one in every six high school seniors has admitted to trying cocaine, one in 18 students has tried crack cocaine, and nearly 40% of adults in their late 20s have used cocaine.[27] A study in 1985 showed that nearly 20% of professional baseball players and 75% of professional basketball players have used cocaine.[28] 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.[25]

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 sniffing (snorting), smoking, chewing, and intravenous injection. The drug is usually snorted by illicit users.[11] 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.[11] 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,

anxiety, toxic psychoses, hallucinations, cardiac arrhythmias, sudden death, angina pectoris, myocardial infarction, and many others.

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.[29] Nicotine use in the athlete population is principally by smokeless tobacco, usually by baseball players. It is hoped the 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.[13,30]

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.[30]

The athlete uses nicotine for the stimulatory effect, calming effect, or appetite control.[12] 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 α_1- or α_2- and β_1- or β_2-receptor stimulation. Examples of the sympathomimetic amines include phenylpropanolamine, 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 β_2 agonists salbutamol and terbutaline are the only approved β_2 agonists for the treatment of asthma in the inhaled form.[9] Not only are these the only two β_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.[9] 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 phenylpropanolamine, the potential effects are still a concern.[31] 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

Generic Name	*Example*
Ephedrine	Tedral, Bronkotabs, Rynatuss, Primatene, Bronkaid, Nyquil Nighttime Cold Medicine, herbal teas and medicines containing Ma Huang (Chinese Ephedra) and related compounds
Pseudoephedrine	Actifed, Afrin tablets, Afrinol, Co-Tylenol, Deconamine, Novafed, Sudafed, Chlor-Trimeton-DC, Drixoral and related compounds
Phenylephrine	Dristan, Neo-Synephrine, Sinex, and related compounds
Desoxyephedrine	Vicks inhaler and related compounds
Phenylpropanolamine	Alka-Seltzer Plus, Allerest, Contact Dexatrim, Dietac, Sine-Aid, Sine-Off Sinutab, Trianinic, Sucrets Cold Decongestant, and related compounds
Isoetharine HCl	Bronkosol, Bronkometer, Numotac, Dilabron, and related compounds
Isoproterenol	Isuprel, Norisodrine, Metihaler-ISO, and related compounds
Metaproterenol	Alupent, Metaprel, and related compounds
Methoxyphenamine	Ritalin, Orthoxicol cough syrup, and related compounds
Methylphenidate HCl	Ritalin and related compounds

(Gilman, A. G., Goodman, L. S., Rall, T. W., et al. (eds.): Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan, 1985.)

uses for β blockers include the treatment of migraine headaches, essential tremors, overactivity, pheochromocytoma, thyroid toxicosis, and alcohol withdrawal. Beta blockers are agents that block β-adrenergic receptors on end organs. The β_1 receptors are found principally in heart, kidneys, and adipose tissue, and β_2 receptors in the liver, bronchi, and arteries. Beta-blocking agents, therefore, can be specific or nonspecific. Nonspecific agents block both the β_1 and β_2 receptor sites, whereas specific β blockers can block either the β_1 or the β_2 receptor sites. Although there is no pure β_1 or β_2 blocking without overlap, the agents principally affect either the β_1 or β_2 receptor sites.[13]

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.[9] 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]

Beta blockers decrease V_{O_2}max by 15% or greater and decrease muscle blood flow, muscle oxygen uptake, and blood glucose concentrations. The adverse effects of beta 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 V_{O_2}max, 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."[9] 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 increase 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 Ekbloom 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 V_{O_2}max by as much as 3.9 to 12.8% and an increase in endurance capacity from 2.5 to 35%.[8]

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.[35] Approximately 3% of all homologous blood transfusions cause immune reactions, including mild allergic reactions, fever, urticaria, and hemolytic transfusion reactions,

which can be fatal.[11] 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.[9,21]

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 B_{15}

Vitamin B_{15} (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 B_{15} 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 in 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 D. de Coubertin. This spirit should be the goal.

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SOURCES OF ADDITIONAL INFORMATION

National Collegiate Athletic Association (NCAA)
6201 College Boulevard
Overland Park, KS 66211-2422
(913) 339-1906

National Federation of State High School Association
11724 Plaza Circle
Kansas City, MO 64195
(816) 464-5400

United States Olympic Committee Drug Control Hotline
1750 East Boulder Street
Colorado Springs, CO 80909
(800) 233-0393