Anabolic Steroid Abuse and Dependence

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Current Psychiatry Reports 2002, **4:**377–387
Current Science Inc. ISSN 1523-3812
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Anabolic-androgenic steroids (AAS) are mainly used to treat androgen deficiency syndromes and, more recently, catabolic states such as AIDS-associated wasting. There is no evidence in the reviewed literature that AAS abuse or dependence develops from the therapeutic use of AAS. Conversely, 165 instances of AAS dependence have been reported among weightlifters and bodybuilders who, as part of their weight training regimens, chronically administered supraphysiologic doses, often including combinations of injected and oral AAS as well as other drugs of abuse. A new model is proposed in which both the "myoactive" and psychoactive effects of AAS contribute to the development of AAS dependence. The adverse consequences of AAS are reviewed, as well as their assessment by means of a history and physical, mental status examination, and laboratory testing. When patients with AAS use disorders are compared with patients with other substance use disorders, both similarities and differences become apparent and have implications for treatment.

Introduction

Anabolic-androgenic steroids (AAS) are synthetic derivatives of the male hormone, testosterone, which bind androgen receptors to produce both anabolic (body building) and androgenic (masculinizing) effects (Table 1). Anabolic-androgenic steroids are primarily indicated and prescribed to treat androgen deficiency syndromes [1], some types of anemia, and hereditary angioedema (a rare skin condition) [2]. Increasingly, they are used to treat AIDS-associated wasting (malnutrition) [3,4] and other catabolic conditions [5–8]. Less common medical uses of AAS have been reviewed elsewhere [1–3,9]. Importantly, no cases of abuse or dependence have been described in men or women who received or self-administered therapeutic doses of AAS for legitimate medical indications. Nevertheless, AAS are classified as Schedule III controlled substances, because using them for nonmedical purposes, such as enhancing physical appearance, can lead to abuse and dependence in both men and women [10,11•,12].

Patterns of nonmedical use

Illicit AAS are usually administered orally or by injection, although transdermal skin patches are also available pharmaceutically. Injection occurs into large muscle groups (buttocks, thigh, or shoulder) or subcutaneously, but not intravenously. Needle sharing is reportedly uncommon, but does occur [13]. Users typically take steroids in "cycles" of 6 to 12 weeks, followed by 6 to 12 weeks off AAS, although longer cycles of use occur. At the beginning of cycles, small doses are taken with the intent to build to larger doses, which are then tapered at the cycle's end. Initial patterns of cycling on and off AAS, however, may develop into continuous patterns without AAS-free intervals, as users try to secure muscle gains while avoiding withdrawal symptoms. Typical AAS doses reach 10 to 100 times the amounts ordinarily prescribed for medical purposes, and users often combine multiple oral and injected AAS to achieve the desired amount. Because illicit steroids commonly contain falsely labeled contents as well as veterinary preparations [14,15], the actual human dosage is usually unknown.

Table 2 lists drugs that are commonly combined with AAS to augment their effects, to manage unpleasant side effects, or to avoid detection by urine testing. Anabolic-androgenic steroid users are also more likely to use alcohol and other drugs of abuse than AAS nonusers are [16–19]. Consistent with elevated alcohol intake in human AAS users, AAS-treated rats increased their voluntary alcohol consumption compared with control-treated rats [20]. Anabolic-androgenic steroids may even serve as a gateway drug to other illicit drug use [21]. Combining AAS with other drugs may lead to adverse interactions. Amphetamines, for example, may increase the overdose potential of AAS due to cardiotoxicity [22•]. Anabolic-androgenic steroids may also increase the reinforcing properties of amphetamine [23], an effect that could favor increased self-administration of both drug classes together. Bromocriptine, used in combination with AAS to reduce body fat, was recently associated with syncopal episodes and atrial fibrillation [24]. In animals, AAS increase cocaine-related seizures [25] and potentiate cocaine-induced increases in heart rate [26]. Autopsies involving human AAS users commonly reveal mixed substance use [22•].

Table 1. Commonly used anabolic-androgenic steroids

Injected testosterone esters (C-17-β ester derivatives)*
Testosterone cypionate
Testosterone enanthate
Testosterone ester mixtures
Testosterone propionate
Testosterone undecanoate
Other injected compounds
Boldenone undecylenate†
Methenolone enanthate
Nandrolone decanoate
Nandrolone phenpropionate
Stanozolol†
Trenbolone acetate†
Trenbolone hexahydrobencylcarbonate
Oral agents (c-17-α alkyl derivatives)‡
Ethylestrenol
Fluoxymesterone
Methandrostenolone or methandienone
Methenolone
Methyltestosterone
Oxandrolone
Oxymetholone
Oxymesterone
Stanozolol

*Metabolized to testosterone and estradiol; less toxic to liver and cholesterol levels.
†Veterinary compound.
‡More toxic to liver and cholesterol levels.

Medical consequences of use

Adverse consequences of using AAS for nonmedical purposes have been reviewed elsewhere [27•,28••–30•], and will be briefly summarized here. Unfortunately, the long-term health risks of nonmedical AAS use are poorly studied [30]. Therefore, neither safety nor catastrophe can be reliably predicted with long-term use, and longitudinal investigations of AAS users are needed. Many common side effects of AAS are reversible at discontinuation. Other reported consequences such as myocardial infarction are infrequent, but obviously irreversible.

Endocrine effects are gender specific. Men may manifest male-pattern baldness, gynecomastia, testicular atrophy, prostatic hypertrophy, and low sperm counts resulting in sterility [31]. Gynecomastia may require surgical intervention in some cases, because of irreversibility and tenderness [32]. Women may manifest menstrual irregularities and masculinizing effects such as increased hair growth, breast tissue atrophy, deepened voice, and clitoral hypertrophy [14]. The latter two effects are often irreversible. Both genders manifest acne in response to hormonal stimulation of sebacious glands.

The cardiovascular and hepatic consequences AAS receive much attention, because of reported fatalities from myocardial infarction, cardiac arrest, stroke, and liver tumors. Anabolic-androgenic steroids, especially the oral (C-17-alkylated) forms (Table 1), alter the cholesterol profile by decreasing high-density lipoproteins and increasing low-density lipoproteins, an effect that favors coronary artery disease, but is fortunately reversible after drug discontinuation. Animal studies suggest that AAS are also toxic to myocardial cells [33]. These observations support an association between AAS and heart disease, and epidemiologic studies are needed to confirm this. The C-17 alkyl forms of AAS are also more likely to cause liver dysfunction than the injected testosterone esters. Adverse hepatic effects include cholestatic jaundice, benign and malignant tumors, and peliosis hepatis (blood filled cysts that may rupture and cause death). Some tumors are reversible after discontinuing AAS.

Anabolic-androgenic steroids may predispose people engaged in strength training to tendon injuries and neuropathies, and animal studies indicate that AAS can damage and weaken collagen fibers in tendons [34]. Other adverse effects attributed to AAS include sleep apnea, exacerbation of tic disorders, polycythemia, and isolated cases of nonhepatic neoplasias. Children who use AAS before and during puberty may suffer from short stature resulting from premature closure of bone plates. Finally, premature mortality has been demonstrated in AAS-treated rats and suggested in humans [35].

Psychiatric effects

Anabolic-androgenic steroids have been associated with depression, mania, psychosis, suicide, and marked aggression leading to violence and homicide [22,29,36,37•,38]. Conversely, they have been used therapeutically to improve mood and alleviate depression [39,40]. Either way, AAS are generally recognized by psychiatric researchers to have psychoactive properties [40].

In a post-mortem study of 34 deaths among users of AAS [22•], mortality resulted from suicide in 11 cases, homicide in nine cases, automobile accidents following reckless driving in two cases, and polysubstance-related causes in 11 cases (*eg*, mixing AAS with heroin, amphetamines, or alcohol). Clearly, psychologic factors were involved in all 34 cases.

The prevalence of AAS-induced psychiatric disorders and effects has been difficult to determine, because of sampling biases inherent with both clinical case reports and convenience samples of AAS users. Pope *et al.* [37•] reviewed four prospective, placebo-controlled trials and conservatively estimated that at least 5% of AAS users will have manic or hypomanic reactions, effects that appeared to be substance induced and dose dependent [29]. However, experimental studies of AAS administration cannot ethically mimic the extreme doses and combinations of AAS taken by nonmedical users, so rates of AAS-induced mental disorders are likely higher. Indeed, rates of psychiatric disorders noted in several diagnostic studies of selected AAS users have been more than 5%. However, a number of factors can increase the likelihood of psychiatric effects including prior psychiatric history, alcohol and

Table 2. Drugs used in combination with anabolic-androgenic steroids

Drug	Reasons for use	Comments
Amphetamines	Increase endurance, burn fat	Stimulant
Androstenedione*	Increase muscle mass and strength	May be purchased over the counter
Bromocriptine	Burn fat	Dopamine agonist
Caffeine	Increase endurance	Over the counter stimulant
Clenbuterol	Increase muscle mass, burn fat	β-agonist
Clomiphene	Prevent gynecomastia; increase gonadotrophins	Blocks estrogen receptors
Clonidine	Increase muscle mass and strength	Via increased growth hormone
Creatine	Increase muscle mass and strength	Over the counter food supplement
Dehydroepiandrosterone	Increase muscle mass and strength	May be purchased over the counter
Diuretics	Decrease anabolic-androgenic steroid-induced edema; dilute urine	Hydrochlorothiazide, furosemide
Ephedrine	Increase endurance, burn fat	Over the counter stimulant
Erythropoietin	Increase endurance	Injected
Gamma-hydroxybutyrate	Increase muscle mass and strength	Via increased growth hormone
Human chorionic gonadotrophin	Increase endogenous testosterone; prevent testicular atrophy from anabolic-androgenic steroids	Injected
Human growth hormone	Increase muscle mass and strength	Injected
Insulin-like growth factor	Increase muscle mass and strength	Injected
Levodopa	Increase muscle mass and strength	Via increased growth hormone
Levothyroxine	Increase endurance, burn fat	Thyroid hormone
Opioids	Decrease pain from workouts and injuries	Supplied by dealers of anabolic-androgenic steroids
Probenicid	Mask urine testing	Decreased renal excretion of drugs
Tamoxifen	Prevent gynecomastia	Blocks estrogen receptors
Testolactone	Prevent gynecomastia	Inhibits estrogen synthesis

*The immediate precursor of testosterone [2].
Some over the counter ergogenic drugs [68,69] have been associated with heavy drinking, driving after drinking, and physical fights [70].

other drug use [41], and comorbid medical conditions. For example, a recent report described a man with Axis II psychopathology who developed psychosis after receiving therapeutic doses of an anabolic steroid for burn injuries in combination with lorazepam and opioids [6]. Expectancy effects may also play a role in mental status changes; for example, it has been suggested that people who expect to become aggressive on AAS do become aggressive.

Some investigators point out that many of the psychiatric effects that are attributed to AAS might result from the type of person who uses them or the effects of weight training itself. For example, the large number of nightclub doormen in one sample [38] confounded high levels of aggression in a recent study of AAS users. Moreover, weight training without AAS use can improve mood [42]. Nevertheless, psychiatric effects following high doses of AAS have been reported in healthy volunteers without a prior history of weight training or mental disorders [37,43••].

Sharing needles or syringes, having unprotected sex with multiple partners, reckless driving, and using other harmful drugs have been noted in some AAS users [13,22•,44]. One explanation is that AAS may induce mental states characterized by impulsivity and poor judgment. Another explanation is that people who accept the risks of taking AAS may exhibit generalized risk-taking tendencies.

The mechanisms underlying the psychiatric effects of AAS remain poorly understood. A recent experimental study found significantly higher levels of 5-hydroyindoleacetic acid (5-HIAA) in the cerebrospinal fluid of methyltestosterone- versus placebo-treated men. Moreover, the increased 5-HIAA levels were significantly correlated with AAS-related effects such as increased energy, diminished sleep, and sexual arousal [43••]. Other studies suggest that AAS-induced increases in substance P and vasopressin [45,46], as well as alterations in central opioid systems [20,47], may be related to aggressive behavior.

Addiction potential

In a previous review of the scientific literature [11•] published between 1988 and 1998, evidence was cited that AAS dependence is a diagnosable mental disorder (Table 3). Between 1999 and 2000, two more diagnostic studies of AAS dependence were published [10,12] including the second known instance of dependence in women. Altogether, the medical literature contains a total of at least 165 AAS users who met criteria for dependence (Table 3). Therefore, AAS dependence is readily identifiable if one samples the right population and asks the usual diagnostic questions.

A withdrawal syndrome from AAS has been described that can last for weeks to months, and consists of depressed

Table 3. Medically published instances of anabolic-androgenic steroid dependence

Authors	Users, *n*	Dependent, *n*
Case reports and series		
Tennant *et al.* [49]	1 man	1
Brower *et al.* [71]	1 man	1
Brower *et al.* [72]	8 men	6
Hays *et al.* [73]	1 man	1
Copeland *et al.* [74]*	1 woman	1
DSM Diagnostic Surveys		
Brower *et al.* [75]	49 men	28
Gridley and Hanrahan [76]	21 men	12
Pope and Katz [77]	88 men	22
Malone *et al.* [78]	71 men; 6 women	11†
Midgley *et al.* [10]	50 men	13
Copeland *et al.* [12]	94 men; 6 women	23 (21 men; 2 women)
Addiction Treatment Center Survey		
Clancy and Yeats [79]	64 men; 4 women	47†
Total instances of anabolic-androgenic steroid dependence		165

DSM—Diagnostic and Statistical Manual of Mental Disorders.
*Case report by Copeland *et al.* [74] is not included in the total, because it is double-counted in the study by Copeland *et al.* [12].
†Number of women not specified.

mood, fatigue, a desire to take more AAS (craving), restlessness, anorexia, insomnia, and decreased libido [11•,48]. Many of these symptoms are the opposite of effects observed during AAS administration (hypomania and increased energy, appetite, and libido). In the 1980s a biphasic course of withdrawal was proposed, based on a single case report [49], with an initial phase lasting for 1 week or less that resembled opioid withdrawal, and a second phase characterized by depressive symptoms and craving. No other observations of an opioid-like withdrawal state have appeared in the literature, and a recent study in rhesus monkeys failed to find naloxone-induced withdrawal phenomena following high-dose AAS administration [50]. Even though AAS do interact with opioid systems in several discrete areas of the brain [20,51,52], the evidence for opioid-like withdrawal and a biphasic course is extremely weak.

A Proposed Model of Anabolic-androgenic Steroid Dependence

All 165 cases of AAS dependence reported to date have occurred in dedicated weight trainers who were motivated to either compete athletically, achieve an extremely muscular appearance, or intimidate and fight potential rivals. All cases have also occurred in a societal context that highly rewards and values people who win sport competitions and appear physically fit and attractive [53]. It is possible, then, that AAS are predominantly reinforcing due to their *muscle-active effects*, in contrast to traditional drugs of abuse (*eg,* heroin and cocaine) that have immediate *psychoactive effects* related to their rapid stimulation of brain reward mechanisms. Indeed, the notion that AAS are immediately reinforcing because of their psychoactive effects has reasonably been challenged [54], even though users may report euphoric and antidepressant effects with continual high-dose administration [27•]. A model of AAS dependence, therefore, must account for the actions of AAS on both muscle and brain tissue in the development of dependence.

A new model of AAS dependence is proposed in which its development occurs primarily in a sociocultural context that motivates certain individuals, particularly men, to attain large and strong muscles for specific purposes. Attaining the desired increases in musculature requires frequent and intensive weight training sessions that become time-consuming and driven, but may also improve mood and self-esteem [42]. Weight training is accompanied by strict dietary regimens. These rigorous regimens of diet and weight training when combined with AAS remain highly goal-directed, compared with the activities of "traditional" drug addicts, which appear to be directed mainly at obtaining and using more drugs for their temporary euphoric effects. At this stage, the reinforcing actions of AAS derive mainly from their muscle-active effects [10], and the compulsive patterns of AAS use parallel the somewhat compulsive patterns of training and diet. Professional treatment for addiction is unlikely to be required at this stage even if users appear to fulfill *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DMS-IV) criteria for dependence on AAS. Rather, users can be expected to discontinue AAS on their own when the goals of increased muscularity are no longer required (such as when a professional athlete retires from competition). This could explain why so few AAS users present for addiction-specialized treatment [55•].

Weight trainers typically and sometimes correctly believe that chronic, high-dose exposure to AAS (in addition to their intensive training regimens) is required to maximize their gains in muscularity. Such high-dose

administration of AAS increases the likelihood of psychoactive effects [37] that could possibly reinforce repeated drug administration. Recent animal studies support the hypothesis that supraphysiologic doses of AAS act on brain-mediated reward systems[51,56–60]. For example, increased levels of beta-endorphin have been reported in the ventral tegmental area [61] and midline thalamus [52] following chronic administration of AAS. Other work implicates dopamine including AAS-induced changes in dopamine receptors in the nucleus accumbens and ventral tegmental area [60]. Recently, the first demonstration of self-administration of AAS by animals was reported [57]. Another animal model of drug dependence is the conditioned place preference paradigm. Recent studies indicate that 3-alpha-androstanediol, a neurosteroid and testosterone metabolite, may mediate conditioned place preference through its actions in the nucleus accumbens [58,59]. Therefore, a second stage is hypothesized during which chronic, high-dose exposure to AAS activates brain-mediated psychoactive effects that augment the "myoactive" dependence on AAS.

In summary, a two-stage model of AAS dependence is proposed. In Stage 1, high-dose AAS are used for their muscle-active effects in conjunction with strict dietary and intensive weight training regimens. The combination of goal-directed activities (weight training, diet, and AAS use) consumes large amounts of time and replaces other activities, continues despite medical and social problems, and is greatly missed and desired if temporarily interrupted. In addition, the AAS user may sometimes feel that the activities are "out of control" when expressing doubts about their value and doing them. At this stage in the process, the AAS user will appear to meet criteria for substance dependence when diagnostic questionnaires and interviews are applied. However, this stage of dependence is greatly confounded by the compulsive quality of weight training [42], and the reinforcing value of achieving large muscle size and increased strength [10]. In Stage 2, chronic, high-dose administration of AAS activates brain-mediated reward systems, similar to other drugs of abuse. Subjectively experienced and objectively observed psychoactive effects such as mood changes and increases in aggressive behaviors characterize this stage of dependence. *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* criteria for AAS dependence are met, and users are not able to discontinue AAS despite their desires and attempts to do so. Addiction treatment may be required at this stage, especially when accompanied by other substance use disorders such as alcohol dependence, opioid dependence, or amphetamine abuse.

Identification and Assessment

The process of evaluating potential, suspected, and known AAS users should include a comprehensive history, physical examination, mental status examination, and laboratory testing [55•]. Young men who engage in weight training or sports that require strength or power are at highest risk to use AAS. Thus, a high index of suspicion for these patients is warranted as the evaluation begins.

History

Inquiry may begin with the use and reasons for use of nutritional supplements and over-the-counter ergogenic aids (Table 2), because the use of legal aids commonly precedes and accompanies the use of AAS. The clinician may then ask if the patient knows other people who use AAS, because people who use or are thinking about using AAS are more likely to know other users than low-risk nonusers [62]. Finally, the clinician should ask if the patient has ever tried AAS. If not, the discussion that ensues may help to prevent initiation of use.

If yes, an inquiry into both the benefits perceived as well as the side effects experienced (both medical and psychosocial) is important. It is generally accepted that AAS can increase muscle mass and strength when combined with proper training and nutrition [27•]. When asking about dependence, the clinician must distinguish between the effects of AAS use and weight training [42]. For example, AAS users may spend large amounts of time training with weights, which can interfere with other important activities. However, only time actually spent on obtaining, using, and recovering from the effects of AAS meets the DSM-IV criterion for spending large amounts of time on drug-related activities.

Physical examination

Table 4 lists the potential findings of AAS use that are observable during the physical examination, based on the adverse medical (as well as desired) consequences discussed. Although high blood pressure typically appears in published lists of adverse events with AAS, several studies have failed to find it.

Mental status examination

A broad range of mental status findings has been associated with AAS. Although causation is sometimes debated in the literature for specific findings such as aggressiveness [38], there is no doubt that such symptoms require careful assessment and therapeutic attention when they do occur. Moreover, discontinuing AAS in patients with adverse mental status changes will help to assess and eliminate the role of AAS.

> Appearance—marked enlargement of musculature with disproportionate development of the neck, shoulders, and chest. Oversized clothes may be worn to hide the extent of muscular development, especially in self-conscious patients who may suffer from muscle dysmorphia. Facial acne is also seen.

Table 4. Physical signs of using anabolic-androgenic steroids for nonmedical purposes

Vital signs	Increased blood pressure (relatively uncommon)
Skin	Acne, needle marks in large muscles, male pattern baldness, hirsutism (especially in women), jaundice with liver disease
Head and neck	Jaundiced eyes with liver disease; deepening of the voice in women
Chest	Gynecomastia with tenderness in men; breast tissue atrophy in women
Abdominal	Right upper quadrant tenderness and hepatomegaly with liver disease
Genitourinary	Testicular atrophy and prostatic hypertrophy in men; clitoral hypertrophy in women
Musculoskeletal	Generalized muscle hypertrophy with disproportionately large upper body mass (especially neck, shoulders, arms, and chest)
Extremeties	Edema due to water retention for which diuretics may be used

Behavior—observe for psychomotor agitation or retardation as consistent with manic or depressive states.

Cooperation—a variable that is dependent on the degree of irritability and defensiveness about drug use. Cooperation may be influenced by traits (or AAS-induced states) of competitiveness and aggressiveness.

Speech—usually normal, although deepened voice is heard in women.

Sensorium—usually clear unless frankly manic or psychotic.

Mood—individual may display mania or hypomania with elevated mood or irritability; dysphoria, depression, or marked anxiety may also be seen.

Affect—observe for lability and intensity of expression when mania or hypomania are present.

Thought process—slowed with depressive features; rapid or disorganized with manic features.

Thought content—assess for suicidal, homicidal, and paranoid ideation. Grandiose or persecutory thoughts may progress to delusions.

Hallucinations—not common, but may occur with states of psychotic depression or mania.

Laboratory examination

Anabolic-androgenic steroids users may manifest abnormalities on their urine drug screens, blood work, semen analyses, and cardiac function tests (Table 5). Although urine tests for AAS are commonly employed at different levels of sport, they are not included in the usual drug screens conducted for clinical purposes by most hospital laboratories. Therefore, urine testing for AAS requires a special order in most clinical settings, necessitating shipment to an outside laboratory. Because of the known association between using AAS and other drugs of abuse, the usual urine drug tests for amphetamines, benzodiazepines, cannabinols, cocaine, and opioids should also be ordered

Blood work should include skeletal muscle enzymes, although they can be elevated in both steroid users and nonusers after intensive weight training. Among users, muscle damage may also result from intramuscular injections. In a few case reports involving AAS users, extremely high levels of creatine kinase resulted from rhabdomyolysis [63,64]. Several muscle enzymes are also present in the liver (Table 5). Liver specific tests (*eg,* bilirubin, gamma-glutamyltransferase) and creatine kinase (not released from the liver in clinically significant amounts) may help to distinguish muscle from hepatic damage [63].

Blood concentrations of testosterone and estrogen vary depending on the specific metabolic pathways of ingested AAS. For example, testosterone esters are metabolized to testosterone and secondarily to estrogen, so increased levels of these hormones may be observed. However, testosterone and estrogen levels are decreased by other AAS.

Treatment

It must emphasized that no controlled trials of treatment for either AAS abuse or dependence have been published [48]. Therefore, the material that follows derive mostly from what little appears in the literature and the author's own experiences [55•]. The goals of treatment are abstinence from AAS and other drugs, restoration of health, and improved psychosocial functioning. Needle-exchange programs and medical monitoring of AAS users are well established in Great Britain and Australia [12,38,55•]. Nevertheless, abstinence should be the ultimate goal of treatment, because of the known risks of short-term use (several months to several years) and the unknown risks of long-term use (10 to 20 years or more) [30•].

The treatment of AAS use disorders may be compared with the treatment of other substance use disorders, particularly when AAS use occurs in the context of polysubstance dependence. As with other drugs of abuse, the treatment of AAS use disorders involves the initiation and maintenance of abstinence, as well as the treatment of comorbid disorders. Nevertheless, there are unique considerations when treating AAS users. First, AAS users focus frequently,

Table 5. Laboratory abnormalities in anabolic-androgenic steroid users

Blood work	
Muscle enzymes	Increased ALT, AST, LDH, and CK
Liver function tests	Increased ALT, AST, LDH, GGT, and total bilirubin
Cholesterol levels	Increased HDL-C; decreased LDL-C
Hormonal levels	Increased testosterone and estradiol (with use of testosterone esters); decreased testosterone (without use of testosterone esters or during withdrawal); decreased LH and FSH
Complete blood count	Increased RBC count, hemoglobin, and hematocrit
Urine testing	
Anabolic-androgenic steroids	Positive
Other drugs of abuse	May be positive
Cardiac testing	
Electrocardiogram	Left ventricular hypertrophy (seen in intensive weight trainers also)
Echocardiogram	Impaired diastolic function
Semen analysis	Decreased sperm count and motility; abnormal morphology

ALT—alanine aminotransferase; AST—aspartate aminotransferase; CK—creatine kinase; FSH—follicle-stimulating hormone; GGT—gamma-glutamyltransferase; HDL-C—high-density lipoprotein cholesterol; LDH—lactate dehydrogenase; LDL-C—low-density lipoprotein cholesterol; LH—luteinizing hormone; RBC—red blood cell.

if not excessively, on their physical attributes, compared with other substance abusers who may disregard their physical attributes and sometimes appear unkempt as drugs increasingly dominate their lives. For some users, physical attributes are a way of defining themselves, deriving self-esteem, and competing successfully in a world perceived as populated with winners and losers. Other users suffer from a form of body dysmorphic disorder, labeled muscle dysmorphia [65••], in which obsessively focusing on muscular appearance becomes an organizing force that, in psychodynamic terms, deflects conscious attention away from other psychosocial problems and underlying conflicts. Recent studies suggest that selective serotonin reuptake inhibitor (SSRI) antidepressants have efficacy in treating body dysmorphic disorder [66], as well as for the depression following discontinuation of AAS [55•].

Second, when abstinence from AAS is initiated, users respond to their perceived and real losses of physical attributes with negative mood states. Anabolic-androgenic steroids can facilitate gains in muscle size and strength that are not necessarily achievable by so-called natural methods alone. Therefore, patients consider their losses irreversible without resuming use of AAS, and they may benefit from therapy that helps them to accept and mourn the loss of both idealized and realized physical attributes. Of course, users must move forward as well as giving due attention to past losses. Patients may be viewed as undergoing a life-cycle transition in which old values and activities that emphasized physical attributes are replaced with newly fulfilling and a balanced array of alternatives that will vary with each individual.

Third, AAS users often embrace prevailing cultural values of physical fitness, success, victory, and appearing attractive. Although people may debate how attractive one becomes after using AAS, there is no doubt that AAS users strive to accomplish something with drugs other than simply getting high or escaping from generally shared cultural values. Rather, their goals of winning competitions and achieving a particular physical appearance require the hard work ordinarily associated with delayed gratification. Indeed, AAS are not immediately euphorigenic and reinforcing in ways that cocaine, heroin, and alcohol are.

For conceptual purposes, treatment may be divided into the following three (admittedly artificial) phases: a post-assessment phase, a withdrawal phase, and a post-withdrawal phase.

Post-assessment phase

The goals of this phase are to treat AAS-associated consequences that require immediate attention and to motivate the patient for treatment of abuse or dependence. Immediate treatment with antipsychotic medication, for example, may be required for marked states of agitation, aggression, and mania. In terms of motivation, AAS users may present for treatment reluctantly as do other patients with substance use disorders. The need of AAS users to see themselves as big and strong may contribute to their denial about having a drug problem. Motivational interviewing techniques may be applied to treatment-averse AAS users [67]. For example, providing feedback regarding laboratory abnormalities and other physical findings is a helpful technique to motivate AAS users who already have strong concerns about their bodies. Involving family and friends in the assessment and motivation of patients for treatment is also useful.

Withdrawal phase

Withdrawal symptoms, discussed previously, are typically depressive in nature. Supportive psychotherapy during withdrawal is essential and consists of reassurance, education, and directive guidance or coaching. Patients are

reassured when they perceive those who treat them as nonjudgmental of their use, understanding of their motivations, and knowledgeable about the effects of AAS. Patients should be educated about the symptoms and course of AAS withdrawal so they may knowingly anticipate what will happen. Whether by trait or drug-induced state, AAS users can be competitive and aggressive, which may confer advantages in some settings, but not in the therapeutic relationship. The therapist who assumes a position like a coach or teammate—that is, someone on the same side as the patient—may effectively diffuse these challenges to treatment.

Pharmacotherapy is viewed as adjunctive to psychotherapy during AAS withdrawal [48]. Endocrine pharmacotherapy is targeted at the syndrome of hypogonadotropic hypogonadism that results from chronic, high-dose administration of AAS. In the absence of any controlled clinical trials, endocrine pharmacotherapy is reserved for persistently severe symptoms that 1) do not respond to supportive psychotherapy, and 2) jeopardize the goal of initiating abstinence because patients find them intolerable [55•]. Endocrine pharmacotherapy is only recommended in consultation with an endocrinologist, especially when considering the treatment of women and children who abuse AAS.

The goals of endocrine therapy are to restore functioning of the hypothalamic-pituitary-gonadal (HPG) axis, and to alleviate associated symptoms such as sterility and fatigue. Tests of endocrine function (Table 5) should be performed both before and during treatment. Endocrine pharmacotherapies for hypogonadotropic hypogonadism include the testosterone esters, human chorionic gonadotropin, antiestrogens, and synthetic forms of luteinizing hormone-releasing hormone [48,55•]. Sample regimens are detailed elsewhere and will not be reviewed here [48,55•].

Depressive symptoms are common during AAS withdrawal, and the use of antidepressants is indicated when symptoms persist and meet criteria for major depression. Serotonin selective reuptake inhibitors are preferred, because of successful case reports (reviewed elsewhere [55•]) and a recent study suggesting that AAS alter serotonergic functioning [43••]. Serotonin selective reuptake inhibitors also have a low potential for overdose, adverse cardiac effects, and anticholinergic side effects, all of which must be considered when treating AAS users who are already at risk for suicide, cardiotoxicity, and prostatic hypertrophy. Antipsychotic drugs may be necessary for treating marked irritability, aggressiveness, or agitation that persists into the withdrawal phase. Although previous reviews by the author suggested a potential role for using clonidine to treat opioid-like withdrawal signs and symptoms [48,55], the lack of evidence that opioid-like withdrawal occurs with AAS dependence precludes any such recommendation [50].

Post-withdrawal phase

Following the immediate withdrawal period, treatment should focus on factors that may increase the risk for relapse including comorbid psychiatric disorders such as major depression and muscle dysmorphia [65••]. Antidepressant treatment that was started during the withdrawal phase should be monitored for effectiveness and any necessary adjustments. If antidepressants were previously withheld to assess the possibility of remission with abstinence and psychotherapy alone, then patients should be reevaluated for medication during this phase if clinically significant depressive symptoms remain present. Antidepressants should also be considered to treat muscle dysmorphia [66]. Antipsychotic medication can usually be tapered and discontinued during this phase unless a persisting comorbid disorder (*eg*, bipolar disorder) requires it.

When motivation to maintain abstinence is high enough and a therapeutic alliance is established, psychotherapy may proceed. Internal and external triggers for resuming AAS use should be identified, and new thoughts and behaviors acquired to counter such triggers and provide alternatives to using AAS. Examples of internal triggers include thinking one is not big enough, feeling inadequate in social situations or during workouts, and perceiving one's body as too small. External triggers include the people, places, and things that were associated with using AAS such as working out in the same gym where AAS were purchased.

Conclusions

This literature review provides no evidence that AAS abuse or dependence develops with the therapeutic use of AAS. Therefore, AAS should not be withheld for legitimate medical indications. However, at least 165 instances of AAS dependence have been reported among weightlifters and bodybuilders who took supraphysiologic doses and engaged in intensive weight training activity. Nonmedical users sometimes take AAS despite adverse endocrine, cardiac, hepatic, and psychiatric consequences and, thus, meet DSM-IV criteria for abuse.

A new model of AAS dependence is proposed consisting of two stages to account for both the muscle-active and psychoactive effects of AAS. In Stage 1, high-dose AAS are used for their muscle-active effects in conjunction with strict dietary and intensive weight training regimens. At this stage, a diagnosis of DSM-IV dependence at this stage is greatly confounded by the compulsive quality of weight training and the reinforcing value of achieving large muscle size and increased strength [10,42]. In Stage 2, it is hypothesized that chronic, high-dose administration of AAS activates brain-mediated reward systems. No cases of AAS dependence in the absence of weight training and muscle-active effects have been described. Thus, Stage 2 users are a subset of Stage 1 users. Latter stage users may meet DSM-IV criteria for dependence and require

professional treatment for addiction, especially in the presence of coexisting substance use disorders.

The history, physical, and laboratory examinations are used to identify new cases of AAS users and to assess for adverse consequences of use and dependence. The mental status examination is a key part of the assessment because multiple reports of suicide and homicide have been associated with nonmedical AAS use. Treatment for withdrawal symptoms involves supportive psychotherapy and sometimes medications. Psychosocial therapy, although similar in some respects to treatment of other substance use disorders, must also address the over-reliance on physical attributes for identity and self-esteem.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- • Of importance
- •• Of major importance

1. Conway AJ, Handelsman DJ, Lording DW, *et al.*: **Use, misuse and abuse of androgens. The Endocrine Society of Australia consensus guidelines for androgen prescribing.** *Med J Aust* 2000, **172:**220–224.
2. Shahidi NT: **A review of the chemistry, biological action, and clinical applications of anabolic-androgenic steroids.** *Clin Ther* 2001, **23:**1355–1390.
3. Basaria S, Wahlstrom JT, Dobs AS: **Clinical review 138: Anabolic-androgenic steroid therapy in the treatment of chronic diseases.** *J Clin Endocrinol Metab* 2001, **86:**5108–5017.
4. Polsky B, Kotler D, Steinhart C: **HIV-associated wasting in the HAART era: guidelines for assessment, diagnosis, and treatment.** *AIDS Patient Care STDS* 2001, **15:**411–423.
5. Langer CJ, Hoffman JP, Ottery FD: **Clinical significance of weight loss in cancer patients: rationale for the use of anabolic agents in the treatment of cancer-related cachexia.** *Nutrition* 2001, **17(suppl):**S1-S20.
6. Morton R, Gleason O, Yates W: **Psychiatric effects of anabolic steroids after burn injuries.** *Psychosomatics* 2000, **41:**66–68.
7. Hart DW, Wolf SE, Ramzy PI, *et al.*: **Anabolic effects of oxandrolone after severe burn.** *Ann Surg* 2001, **233:**556–564.
8. Ferreira I, Brooks D, Lacasse Y, Goldstein R: **Nutritional intervention in COPD: a systematic overview.** *Chest* 2001, **119:**353–363.
9. Morley JE: **Testosterone replacement in older men and women.** *J Gend Specif Med* 2001, **4:**49–53.
10. Midgley SJ, Heather N, Davies JB: **Dependence-producing potential of anabolic-androgenic steroids.** *Addict Res* 1999, **7:**539–550.
11. • Brower KJ: **Anabolic steroids: potential for physical and psychological dependence.** In *Anabolic Steroids in Sport and Exercise,* edn 2. Edited by Yesalis CE. Champaign: Human Kinetics; 2000:280–304.

A detailed and comprehensive review of the literature on AAS dependence, including evidence, course, predictors, and mechanisms.

12. Copeland J, Peters R, Dillon P: **Anabolic-androgenic steroid use disorders among a sample of Australian competitive and recreational users.** *Drug Alcohol Depend* 2000, **60:**91–96.
13. Midgley SJ, Heather N, Best D, *et al.*: **Risk behaviors for HIV and hepatitis infection among anabolic-androgenic steroid users.** *AIDS Care* 2000, **12:**163–170.
14. Gruber AJ, Pope HG Jr: **Psychiatric and medical effects of anabolic-androgenic steroid use in women.** *Psychother Psychosom* 2000, **69:**19–26.
15. O'Sullivan AJ, Kennedy MC, Casey JH, *et al.*: **Anabolic-androgenic steroids: medical assessment of present, past and potential users.** *Med J Aust* 2000, **173:**323–327.
16. Nilsson S, Baigi A, Marklund B, Fridlund B: **Trends in the misuse of androgenic anabolic steroids among boys 16–17 years old in a primary health care area in Sweden.** *Scand J Prim Health Care* 2001, **19:**181–182.
17. Irving LM, Wall M, Neumark-Sztainer D, Story M: **Steroid use among adolescents: findings from Project EAT.** *J Adolesc Health* 2002, **30:**243–252.
18. Wichstrom L, Pedersen W: **Use of anabolic-androgenic steroids in adolescence: winning, looking good or being bad?** *J Stud Alcohol* 2001, **62:**5–13.
19. Kindlundh AM, Hagekull B, Isacson DG, Nyberg F: **Adolescent use of anabolic-androgenic steroids and relations to self-reports of social, personality and health aspects.** *Eur J Public Health* 2001, **11:**322–328.
20. Johansson P, Lindqvist A, Nyberg F, Fahlke C: **Anabolic androgenic steroids affects alcohol intake, defensive behaviors and brain opioid peptides in the rat.** *Pharmacol Biochem Behav* 2000, **67:**271–279.
21. Arvary D, Pope HG Jr: **Anabolic-androgenic steroids as a gateway to opioid dependence.** *N Engl J Med* 2000, **342:**1532.
22. • Thiblin I, Lindquist O, Rajs J: **Cause and manner of death among users of anabolic androgenic steroids.** *J Forensic Sci* 2000, **45:**16–23.

This article well documents the influence of psychiatric factors on premature mortality in AAS users. Suicide, homicide, reckless driving, and mixed drug abuse accounted for all 34 cases described.

23. Clark AS, Lindenfeld RC, Gibbons CH: **Anabolic-androgenic steroids and brain reward.** *Pharmacol Biochem Behav* 1996, **53:**741–745.
24. Manoharan G, Campbell NP, O'Brien CJ: **Syncopal episodes in a young amateur body builder.** *Br J Sports Med* 2002, **36:**67–68.
25. Long SF, Wilson MC, Davis WM: **The effects of nandrolone decanoate on cocaine-induced kindling in male rats.** *Neuropharmacology* 2000, **39:**2442–2447.
26. Phillis BD, Irvine RJ, Kennedy JA: **Combined cardiac effects of cocaine and the anabolic steroid, nandrolone, in the rat.** *Eur J Pharmacol* 2000, **398:**263–272.
27. • Kutscher EC, Lund BC, Perry PJ: **Anabolic steroids: a review for the clinician.** *Sports Med* 2002, **32:**285–296.

An up to-date overview of the topic for practicing physicians.

28. •• Yesalis CE: *Anabolic Steroids in Sport and Exercise,* edn 2. Champaign: Human Kinetics; 2000.

An excellent compilation of well-referenced chapters that cover the full spectrum of AAS-related topics including history, epidemiology, effects on performance, medical and psychiatric consequences, drug testing, prevention, assessment and treatment, and legal aspects. Another chapter is devoted to the use and effects of AAS in women.

29. Pope HG Jr, Brower KJ: **Anabolic-androgenic steroid abuse.** In *Comprehensive Textbook of Psychiatry,* edn 7. Edited by Sadock BJ, Sadock VA. Philadelphia: Lippincott Williams & Wilkins; 2000:1085–1095.
30. • Parssinen M, Seppala T: **Steroid use and long-term health risks in former athletes.** *Sports Med* 2002, **32:**83–94.

Details information that is useful for informing and counseling potential and current users of AAS.

31. Torres-Calleja J, Gonzalez-Unzaga M, DeCelis-Carrillo R, *et al.*: **Effect of androgenic anabolic steroids on sperm quality and serum hormone levels in adult male bodybuilders.** *Life Sci* 2001, **68:**1769–1774.
32. Babigian A, Silverman RT: **Management of gynecomastia due to use of anabolic steroids in bodybuilders.** *Plast Reconstr Surg* 2001, **107:**240–242.
33. Zaugg M, Jamali NZ, Lucchinetti E, *et al.*: **Anabolic-androgenic steroids induce apoptotic cell death in adult rat ventricular myocytes.** *J Cell Physiol* 2001, **187:**90–95.
34. Haupt HA: **Upper extremity injuries associated with strength training.** *Clin Sports Med* 2001, **20:**481–490.

35. Parssinen M, Kujala U, Vartiainen E, *et al.*: **Increased premature mortality of competitive powerlifters suspected to have used anabolic agents.** *Int J Sports Med* 2000, **21:**225–227.
36. Bahrke MS: **Psychological effects of endogenous testosterone and anabolic-androgenic steroids.** In *Anabolic Steroids in Sport and Exercise,* edn 2. Edited by Yesalis CE. Champaign: Human Kinetics; 2000:247–278.
37.• Pope HG Jr, Kouri EM, Hudson JI: **Effects of supraphysiologic doses of testosterone on mood and aggression in normal men: a randomized controlled trial.** *Arch Gen Psychiatry* 2000, **57:**133–140.

One of few controlled studies investigating the psychiatric effects of high-dose AAS. The authors go on to estimate the prevalence of mania and hypomania among AAS users, and argue why their estimate is the lower limit.

38. Midgley SJ, Heather N, Davies JB: **Levels of aggression among a group of anabolic-androgenic steroid users.** *Med Sci Law* 2001, **41:**309–314.
39. Rabkin JG, Wagner GJ, Rabkin R: **A double-blind, placebo-controlled trial of testosterone therapy for HIV-positive men with hypogonadal symptoms.** *Arch Gen Psychiatry* 2000, **57:**141–147.
40. Yates WR: **Testosterone in psychiatry: risks and benefits.** *Arch Gen Psychiatry* 2000, **57:**155–156.
41. Dean CE: **Prasterone (DHEA) and mania.** *Ann Pharmacother* 2000, **34:**1419–1422.
42. Bahrke MS, Yesalis CE: **Weight training: a potential confounding factor in examining the psychological and behavioral effects of anabolic-androgenic steroids.** *Sports Med* 1994, **18:**309–318.
43.•• Daly RC, Su TP, Schmidt PJ, *et al.*: **Cerebrospinal fluid and behavioral changes after methyltestosterone administration: preliminary findings.** *Arch Gen Psychiatry* 2001, **58:**172–177.

One of the first studies to make a correlation between AAS-induced neurochemical effects and behavioral changes in humans.

44. Aitken C, Delalande C, Stanton K: **Pumping iron, risking infection? Exposure to hepatitis C, hepatitis B and HIV among anabolic-androgenic steroid injectors in Victoria, Australia.** *Drug Alcohol Depend* 2002, **65:**303–308.
45. Hallberg M, Johansson P, Kindlundh AM, Nyberg F: **Anabolic-androgenic steroids affect the content of substance P and substance P(1–7) in the rat brain.** *Peptides* 2000, **21:**845–852.
46. Harrison RJ, Connor DF, Nowak C, *et al.*: **Chronic anabolic-androgenic steroid treatment during adolescence increases anterior hypothalamic vasopressin and aggression in intact hamsters.** *Psychoneuroendocrinology* 2000, **25:**317–338.
47. Schlussman SD, Zhou Y, Johansson P, *et al.*: **Effects of the androgenic anabolic steroid, nandrolone decanoate, on adrenocorticotropin hormone, corticosterone and proopiomelanocortin, corticotropin releasing factor (CRF) and CRF receptor1 mRNA levels in the hypothalamus, pituitary and amygdala of the rat.** *Neurosci Lett* 2000, **284:**190–194.
48. Brower KJ: **Withdrawal from anabolic steroids.** *Curr Ther Endocrinol Metab* 1997, **6:**338–343.
49. Tennant F, Black DL, Voy RO: **Anabolic steroid dependence with opioid-type features.** *N Engl J Med* 1988, **319:**578.
50. Negus SS, Pope HG Jr, Kanayama G, *et al.*: **Lack of evidence for opioid tolerance or dependence in rhesus monkeys following high-dose anabolic-androgenic steroid administration.** *Psychoneuroendocrinology* 2001, **26:**789–796.
51. Johansson P, Hallberg M, Kindlundh A, Nyberg F: **The effect on opioid peptides in the rat brain, after chronic treatment with the anabolic androgenic steroid, nandrolone decanoate.** *Brain Res Bull* 2000, **51:**413–418.
52. Harlan RE, Brown HE, Lynch CS, *et al.*: **Androgenic-anabolic steroids blunt morphine-induced c-fos expression in the rat striatum: possible role of beta-endorphin.** *Brain Res* 2000, **853:**99–104.
53. Yesalis CE, Bahrke MS: **Doping among adolescent athletes.** *Baillieres Best Pract Res Clin Endocrinol Metab* 2000, **14:**25–35.
54. Fingerhood MI, Sullivan JT, Testa M, Jasinski DR: **Abuse liability of testosterone.** *J Psychopharmacol* 1997, **11:**59–63.
55.• Brower KJ: **Assessment and treatment of anabolic steroid abuse, dependence, and withdrawal.** In *Anabolic Steroids in Sport and Exercise,* edn 2. Edited by Yesalis CE. Champaign: Human Kinetics; 2000:305–332.

This book chapter contains the authors' most comprehensive review of clinical assessment and treatment issues, and served as the guide to relevant sections of the current article.

56. Arnedo MT, Salvador A, Martinez-Sanchis S, Gonzalez-Bono E: **Rewarding properties of testosterone in intact male mice: a pilot study.** *Pharmacol Biochem Behav* 2000, **65:**327–332.
57. Johnson LR, Wood RI: **Oral testosterone self-administration in male hamsters.** *Neuroendocrinology* 2001, **73:**285–292.
58. Rosellini RA, Svare BB, Rhodes ME, Frye CA: **The testosterone metabolite and neurosteroid 3alpha-androstanediol may mediate the effects of testosterone on conditioned place preference.** *Brain Res Brain Res Rev* 2001, **37:**162–171.
59. Frye CA, Park D, Tanaka M, *et al.*: **The testosterone metabolite and neurosteroid 3alpha-androstanediol may mediate the effects of testosterone on conditioned place preference.** *Psychoneuroendocrinology* 2001, **26:**731–750.
60. Kindlundh AM, Lindblom J, Bergstrom L, *et al.*: **The anabolic-androgenic steroid nandrolone decanoate affects the density of dopamine receptors in the male rat brain.** *Eur J Neurosci* 2001, **13:**291–296.
61. Johansson P, Ray A, Zhou Q, Huang W, *et al.*: **Anabolic androgenic steroids increase beta-endorphin levels in the ventral tegmental area in the male rat brain.** *Neurosci Res* 1997, **27:**185–189.
62. Brower KJ, Blow FC, Hill EM: **Risk factors for anabolic-androgenic steroid use in men.** *J Psychiatr Res* 1994, **28:**369–380.
63. Pertusi R, Dickerman RD, McConathy WJ: **Evaluation of aminotransferase elevations in a bodybuilder using anabolic steroids: hepatitis or rhabdomyolysis?** *J Am Osteopath Assoc* 2001, **101:**391–394.
64. Braseth NR, Allison EJ Jr, Gough JE: **Exertional rhabdomyolysis in a body builder abusing anabolic androgenic steroids.** *Eur J Emerg Med* 2001, **8:**155–157.
65.•• Olivardia R, Pope HG Jr, Hudson JI: **Muscle dysmorphia in male weightlifters: a case-control study.** *Am J Psychiatry* 2000, **157:**1291–1296.

This article from the authors who coined the term, "muscle dysmorphia," examines a variant of body dysmorphic disorder characterized by preoccupation with the perception that one's musculature is too small. Previously labeled "reverse anorexia nervosa" by the same investigators, muscle dysmorphia may possibly underlie many cases of Stage I dependence on AAS.

66. Phillips KA, Albertini RS, Rasmussen SA: **A randomized placebo-controlled trial of fluoxetine in body dysmorphic disorder.** *Arch Gen Psychiatry* 2002, **59:**381–388.
67. Brower KJ, Rootenberg JH: **Counseling for substance abuse problems.** In *Counseling in Sports Medicine.* Edited by Ray R, Wiese-Bjornstal D. Champaign: Human Kinetics; 1999:179–204.
68. Ahrendt DM: **Ergogenic aids: counseling the athlete.** *Am Fam Physician* 2001, **63:**913–922.
69. Kanayama G, Gruber AJ, Pope HG Jr, *et al.*: **Over-the-counter drug use in gymnasiums: an underrecognized substance abuse problem?** *Psychother Psychosom* 2001, **70:**137–140.
70. Stephens MB, Olsen C: **Ergogenic supplements and health risk behaviors.** *J Fam Pract* 2001, **50:**696–699.
71. Brower KJ, Blow FC, Beresford TP, Fuelling C: **Anabolic-androgenic steroid dependence.** *J Clin Psychiatry* 1989, **50:**31–33.
72. Brower KJ, Eliopulos GA, Blow FC, *et al.*: **Evidence for physical and psychological dependence on anabolic androgenic steroids in eight weight lifters.** *Am J Psychiatry* 1990, **147:**510–512.
73. Hays LR, Littleton S, Stillner V: **Anabolic steroid dependence.** *Am J Psychiatry* 1990, **147:**122.
74. Copeland J, Peters R, Dillon P: **Anabolic-androgenic steroid dependence in a woman.** *Aust N Z J Psychiatry* 1998, **32:**589.

75. Brower KJ, Blow FC, Young JP, Hill EM: **Symptoms and correlates of anabolic-androgenic steroid dependence.** *Br J Addict* 1991, **86:**759–768.
76. Gridley DW, Hanrahan SJ: **Anabolic-androgenic steroid use among male gymnasium participants: dependence, knowledge, and motives.** *Sport Health* 1994, **12:**11–14.
77. Pope HG Jr, Katz DL: **Psychiatric and medical effects of anabolic-androgenic steroid use: a controlled study of 160 athletes.** *Arch Gen Psychiatry* 1994, **51:**375–382.
78. Malone DA Jr, Dimeff RJ, Lombardo JA, Sample RH: **Psychiatric effects and psychoactive substance use in anabolic- androgenic steroid users.** *Clin J Sport Med* 1995, **5:**25–31.
79. Clancy GP, Yates WR: **Anabolic steroid use among substance abusers in treatment.** *J Clin Psychiatry* 1992, **53:**97–100.