

Bodybuilding's dark side: Clues to anabolic steroid use

Watch for telltale behavioral and physical signs of this most-secretive substance abuse

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Anabolic steroid use by athletes and body-builders has captured public attention but remains poorly understood by most physicians. This is not surprising because users of anabolic-androgenic steroids (AAS):

- rarely seek treatment or disclose their drug use
- frequently distrust professionals.

If you are a clinician who regularly sees male adolescents and young men, you need to become familiar with—and watch for—this often-secret form of substance abuse. This article provides the groundwork for that understanding, starting with the story of “Aaron”—a composite patient whose case represents experiences and verbatim quotes from AAS users known to the authors.

CASE REPORT: 'I FEEL INVINCIBLE'

At his first visit, Aaron, age 18, told the psychiatrist he had no complaints but was coming to please his parents. “I have a lot of arguments with my Dad,” he said, “and they keep thinking something’s wrong with me.”

The patient was very muscular and dressed in baggy sweats that masked his body proportions. He was appropriately groomed and darkly tanned but displayed some acne. The clinician guessed he weighed about 175 lbs and stood at about 65 inches, with very low body fat. Although superficially confident, he seemed restless, somewhat anxious, and guarded as the interview progressed.

Aaron admitted he experienced prominent mood swings. During rage outbursts, he had damaged objects and put his fist through the wall. “There’s holes all over the wall of my room,” he joked.

He also had assaulted a motorist in a traffic altercation, then left the scene. “Did you hurt him?” the clinician asked. Somewhat sheepishly, Aaron responded, “Well, I bought the newspaper and kept checking the obituaries for about 2 weeks afterwards.”

He spoke with pride about his weightlifting, which was the focus of his life. He revealed that he was preparing for a bodybuilding contest in 2 months. The psychiatrist asked him about use of supplements—protein shakes, creatine, and “andro” (androstenedione)—all of which Aaron acknowledged. The psychiatrist then gently asked about anabolic steroid use ([Box 1](#)).

Initially, Aaron strongly denied using AAS. The psychiatrist persisted, pointing out that no information would be disclosed to his parents, and asked again using colloquial terms from the AAS subculture: “Anybody who is prepping for an untested contest in a couple of months is going to be on a cycle. Come on, what are you taking?”

What are anabolic-androgenic steroids?

Anabolic-androgenic steroids (AAS) are hormones that include testosterone—nature’s own AAS—and more than 100 synthetically developed testosterone relatives. All AAS possess anabolic (muscle-building) and androgenic (masculinizing) properties; no known compound can produce one of these effects without the other.

Because of their masculinizing effects, AAS are rarely used by women—and even then in much lower doses than those used by men. Thus, this article focuses on evaluating and treating male adolescents and men.

AAS are not:

- **Corticosteroids** (such as cortisol) are often called “steroids” but possess no muscle-building properties. Corticosteroids’ prominent but idiosyncratic psychiatric effects are usually seen in consultation-liaison settings where patients have been prescribed these drugs, rather than among substance abusers.
- **Androstenedione** (“andro”) and its relatives are adrenal steroids that are weakly metabolized into testosterone or other AAS. These substances were sold legally without prescription in the United States for many years but were banned by federal law in October 2004. Their anabolic and psychiatric effects are much weaker than those of AAS.
- **Athletic “supplements”** with names designed to sound like AAS (such as beginning with “Ana...”) or supplements claimed to be “testosterone-releasers” or the like. If sold legally in supplement stores, an athletic supplement is not an AAS. Psychiatric effects are extremely unlikely.

Eventually it emerged that Aaron had taken five 8- to 20-week AAS “cycles” (courses), during which he had “stacked” (combined) various “injectables” such as IM testosterone and “orals” such as methyltestosterone ([Table 1](#)). His current cycle included:

- a blend of testosterone esters (Sustanon), 500 mg IM once a week
- boldenone (Equipose), a veterinary AAS normally used for horses, 200 mg IM per week
- oxymetholone (Anadrol), 50 mg orally per day.

Table 1

Commonly used anabolic-androgenic steroids ‘Injectables’ (usually administered only by injection)

Boldenone (Equipose)*
Methenolone (Primobolan depot)
Nandrolone (Deca-Durabolin, Durabolin, Laurabolin, others)
Stanozolol (Winstrol-V)*
Testosterone esters (Depo-testosterone, Sten, Sustanon, others)
Trenbolone (Finajet, Parabolan)
‘Orals’
Methandienone (formerly called methandrostenolone) (Dianabol, others)
Methenolone (Primobolan)
Methyltestosterone (Android, others)
Mibolerone (Checque Drops)*
Oxandrolone (Anavar, Lipidex)
Oxymetholone (Anadrol, Anapolon)

Stanozolol (Stromba, Winstrol)	
Other anabolic substances sold on the 'black market'	
Human growth hormone (HGH)	Possesses anabolic properties
	Extremely expensive
	Almost impossible to detect by testing
	Lacks androgenic effects
	Psychiatric effects appear negligible
	Large doses can cause acromegaly
Clenbuterol	Beta-adrenergic agonist with stimulant and anabolic properties
	Used less commonly than AAS
	Lacks androgenic effects and assists fat loss
	Can produce psychiatric effects similar to those of amphetamine abuse (rare, in the authors' experience)
Human chorionic gonadotropin (HCG)	Stimulates testes to produce more testosterone, creating an AAS effect
	Most commonly used near the end of an AAS "cycle" to "jump-start" the hypothalamic-pituitary-testicular axis and minimize AAS withdrawal
* Veterinary preparation	
AAS: anabolic-androgenic steroid	

His friends had taught him to self-inject AAS at age 15; he admitted that he was also occasionally self-injecting the opioid analgesic nalbuphine intravenously because of "pain in my 'deltis' from military presses."

During his cycles, Aaron experienced hypomanic symptoms, including euphoria, prominent irritability, increased libido, decreased need for sleep, and grandiosity. "I feel invincible," he said. His aggressive outbursts had worsened with increasing AAS doses; in addition to attacking the motorist, he also had been physically violent with his girlfriend. "She's scared of me when I'm on juice," he conceded.

During the withdrawal phase after stopping each cycle, Aaron described prominent depression with anhedonia, hypersomnia, loss of libido, and suicidal ideation. "I once almost jumped off a bridge after my fourth cycle," he admitted. "I couldn't wait to get on my next cycle to feel good again." His depressions were also characterized by body-image obsessions; he would regularly spend at least 1 hour a day examining his musculature in a mirror, and sometimes refused to go out in public because he "was getting too small."

Perhaps most disturbing was his increasing use of opioids. In addition to self-injecting nalbuphine, he also ingested oral opioids such as oxycodone almost daily. He mentioned that several of his friends in the gym had progressed from injecting nalbuphine to injecting morphine or heroin, and he knew two bodybuilders who had died from apparently unintentional opioid overdoses.

Aaron said his parents, teachers, and non-bodybuilding friends were unaware of this history. He claimed his parents were proud that their son had apparently eschewed drugs and alcohol to pursue a healthy athletic lifestyle.

RECOGNIZING AAS USE

In our experience with treating substance abusers, we find that AAS users may be the least likely to disclose their drug use to clinicians. In a recent study,¹ 20 of 36 AAS users (56%) reported they had never revealed their AAS use to any physician. When asked to rate their trust in sources of information about AAS, 17 of 42 AAS users (40%) said they trusted information from their drug dealers at least as much as information from any physician they had seen.

Some expressed contempt for physicians as “geeks” or “pencil-necks” who could not comprehend the body-building lifestyle. They gave doctors high marks on knowledge of tobacco, alcohol, and ordinary “street drugs” but much lower ratings on AAS knowledge. Other investigators have shown that many clinicians are unfamiliar with AAS.^{2,3}

AAS users embrace these beliefs for two other reasons. First, to admit to AAS use is to admit that one’s muscularity and physical prowess is the result of taking a drug; there is no comparable motivation to withhold information about, say, one’s use of marijuana or cocaine.

Second, AAS users are much less likely than other substance abusers to view their behavior as pathologic. We have argued that our culture is partially to blame.⁴ Americans pay to watch 300-lb football linemen and AAS-using movie stars. Makers of cars, computers, and electronics do not hesitate to advertise their products as “on steroids,” but they would never claim their products were “on cocaine.” In this climate, it is easy to forget that AAS use is an illicit substance abuse.

How to calculate fat-free mass index (FFMI)

Formula: $FFMI = (W \times (100 - BF)/100)H^2 + 6.1 \times (1.8 - H)$

W = weight in kilograms

BF = body fat percentage

H = height in meters

Obtain height in meters and weight in kilograms. Ideally, measure body fat using calipers, electrical impedance, or some other method. Alternately, estimate body fat by visual inspection:

- 20% = average 30-year-old American man
- 10% = quite lean
- 5% = approaching lowest body fat normally attainable

FFMI values for American men:

20 = average

22 = visibly muscular

25 = approximate maximum attainable by a lean individual without using drugs

Example 1

Young male weightlifter is 69 inches tall, weighs 175 lbs, and is moderately lean, with body fat of 10%; he denies AAS use

$H = 69 \text{ inches} \times .0254 \text{ meters/inch} = 1.75 \text{ m}$

$H^2 = 1.75 \times 1.75 = 3.06 \text{ m}^2$

$W = 175 \text{ pounds} \times 0.454 \text{ kilograms/lb} = 79.5 \text{ kg}$

Therefore, $FFMI = (79.5 \times (100-10)/100)/3.06 + 6.1 \times (1.8 - 1.75) = 23.7$

This degree of muscularity can be attained without using AAS

Example 2

Young male weightlifter is 66 inches tall, weighs 175 lbs, and is very lean, with body fat of 6%; he also denies AAS use

$$H = 66 \text{ inches} \times .0254 \text{ meters/inch} = 1.68 \text{ m}$$

$$H^2 = 1.68 \times 1.68 = 2.82 \text{ m}^2$$

$$W = 175 \text{ pounds} \times 0.454 \text{ kilograms/lb} = 79.5 \text{ kg}$$

$$\text{Therefore, FFMI} = (79.5 \times (100-6)/100) / 2.82 + 6.1 \times (1.8 - 1.68) = 27.2$$

This level of muscularity is extremely unlikely without drugs. Patient is almost certainly lying and should be gently confronted, especially if other symptoms (Table 2) suggest AAS use

Table 2

Clues to possible AAS use in men Muscularity

Estimated fat-free mass index (FFMI) >26 (see [Box 2](#))

Recent rapid muscle gains (>8 lb/month)

Striae over pectoralis muscles caused by rapid hypertrophy of underlying muscle

Other physical signs

Acne

Gynecomastia

Testicular atrophy

Psychiatric signs

Uncharacteristically aggressive behavior

Uncharacteristic hypomanic symptoms

- Grandiosity
- Decreased sleep
- Hypersexuality
- Recklessness

Uncharacteristic depressive symptoms

- Sudden depression onset
- Anhedonia
- Hypersomnia

To overcome these treatment obstacles, we recommend that you:

- Become as knowledgeable about AAS use as you are about other forms of substance abuse (see [Related resources](#)).
- Approach AAS users as you would any other substance abusers—as individuals at risk for potentially serious medical and psychiatric consequences.
- Maintain a high index of suspicion when evaluating any muscular young male patient, even if he initially denies AAS use.

AAS use can often be suspected by looking at the patient as he walks in the door. Using what we call the “fat-free mass index” (FFMI) to calculate muscularity (*Box 2*), we have shown that a lean man can achieve only a certain amount of muscularity without using drugs.⁵ Although this finding needs to be replicated elsewhere, in our experience a man is almost certainly lying if he:

- is relatively lean (with approximately 10% body fat)
- displays an FFMI >26
- and claims he has not used drugs.

If a patient has an elevated FFMI and other cues suggesting AAS use (*Table 2*), gently but persistently question him if he denies using these drugs.

TREATING AAS-ASSOCIATED SYNDROMES

When you have established a history of AAS use, you will be far better prepared to anticipate and possibly treat its associated syndromes. Discussion of these effects is beyond the scope of this paper; for details, see reviews of AAS-associated medical effects,^{3,6} psychiatric effects,^{6,7} and general treatment principles.⁸ We focus here on the four scenarios clinicians encounter most often in practice and offer some pragmatic suggestions.

Forensic cases. AAS users almost never voluntarily seek help to stop their drug use. Such a request would be somewhat analogous to a girl with anorexia nervosa voluntarily asking for help to gain weight. We are unaware of any rehabilitation centers, clinics, 12-step programs, or the like for AAS users—there is no demand for them.

Thus, an AAS user may first come to clinical attention through legal channels. For example, if an AAS user committed a violent crime while experiencing hypomanic effects from these drugs, he might be required to undergo random urine testing as a condition of probation. This may be reasonable, provided that the tests are unannounced and urine is always collected under direct observation.

Monitoring clinicians may serve as little better than policemen, although sometimes it is possible to forge an alliance with the patient.

Depression. Exogenous AAS administration suppresses endogenous testosterone production through feedback mechanisms involving the hypothalamic-pituitary-testicular axis.^{3,6} Thus, during a long cycle, the user’s testes may shrink to half their normal size and stop producing testosterone and spermatozoa.

If the user then stops AAS rapidly, he may plunge into a profoundly hypogonadal state associated with symptoms of major depression. In a field study of 77 steroid users (71 male and 6 female), 6 (7.8%) reported they attempted suicide during AAS withdrawal.⁹ Depression associated with AAS withdrawal may prompt users to resume AAS quickly, triggering a syndrome of AAS dependence.^{6,10,11}

Fortunately, AAS-withdrawal depression is usually self-limited and responds—in our experience and that of others¹²—to standard antidepressants. We recommend aggressively treating such depressions, as doing so may prevent resumption of AAS use and eventual AAS dependence.

Body-image disorders. AAS users often report body-image disorders, especially muscle dysmorphia—a form of body dysmorphic disorder where individuals become preoccupied with the belief that they are not adequately muscular.^{13,14} Anxieties about muscularity are a risk factor for subsequent AAS use¹⁵ and a major contributor to AAS dependence.^{8,11}

Body dysmorphic disorder responds to pharmacologic and cognitive-behavioral interventions.^{3,16} Young men showing pathologic concerns about their muscularity or displaying related body-image pathology may benefit from prompt treatment before they are tempted to use AAS.

Progression to opioid dependence. An ominous development among American¹⁷ and British¹⁸ AAS users is a growing tendency to use opioids. In two studies of individuals with opioid dependence,^{19,20} 7% to 9% reported beginning as AAS users, then learning about opioids from fellow bodybuilders and often buying their first illicit opioids from the person who had

sold them AAS. Most learned as teenagers to use needles to inject AAS intramuscularly, so beginning to using opioids intravenously was only a small step.

In the last 5 years, we have become anecdotally aware of numerous AAS users who developed heroin addiction requiring repeated inpatient detoxification or who died of unintentional opioid overdoses. We suspect this phenomenon is under-recognized and urge clinicians to watch for it.

Related resources

- Pope HG Jr, Brower KJ. Anabolic-androgenic steroid abuse. In: Sadock BJ, Sadock VA (eds). *Comprehensive textbook of psychiatry (8th ed)*. Philadelphia: Lippincott Williams & Wilkins (in press).
- Yesalis CE (ed). *Anabolic steroids in sport and exercise (2nd ed)*. Champaign, IL: Human Kinetics, 2000.
- The Taylor Hooton Foundation. Started by the father of a high school athlete who committed suicide during a depressive episode apparently precipitated by AAS withdrawal. Includes links to related Web sites. <http://www.taylorhooton.org/about.asp>. Accessed Nov. 10, 2004.

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References

1. Pope HG Jr, Kanayama G, Ionescu-Pioggia M, Hudson JI. Anabolic steroid users' attitudes towards physicians. *Addiction* 2004;99:1189–94.
2. Dawson RT. Drugs in sport—the role of the physician. *J Endocrinol* 2001;170:55–61.
3. Kutscher EC, Lund BC, Perry PJ. Anabolic steroids: a review for the clinician. *Sports Med* 2002;32:285–96.
4. Pope HG Jr, Phillips KA, Olivardia R. *The Adonis complex: the secret crisis of male body obsession*. New York: Free Press, 2000.
5. Kouri EM, Pope HG Jr, Katz DL, Oliva PS. Fat-free mass index in users and non-users of anabolic-androgenic steroids. *Clin J Sport Med* 1995;5:223–8.
6. Brower KJ. Anabolic steroid abuse and dependence. *Curr Psychiatry Rep* 2002;4:377–83.
7. Pope HG Jr, Katz DL. Psychiatric effects of exogenous anabolic-androgenic steroids. In: Volkowitz OM, Rothschild AJ (eds). *Psychoneuroendocrinology: the scientific basis of clinical practice*. Washington, DC: American Psychiatric Publishing, 2003;331–58.
8. Pope HG Jr, Brower KJ. Anabolic-androgenic steroids. In: Galanter M, Kleber HD (eds). *American Psychiatric Publishing textbook of substance abuse treatment (3rd ed)*. Washington DC: American Psychiatric Publishing, 2004;257–64.
9. Malone DA Jr, Dimeff R, Lombardo JA, Sample BRH. Psychiatric effects and psychoactive substance use in anabolic-androgenic steroid users. *Clin J Sports Med* 1995;5:25–31
10. Kashkin KB, Kleber HD. Hooked on hormones? An anabolic steroid addiction hypothesis. *JAMA* 1989;262:3166–70.
11. Brower KJ, Eliopoulos GA, Blow FC, et al. Evidence for physical and psychological dependence on anabolic androgenic steroids in eight weight lifters. *Am J Psychiatry*. 1990;147(4):510–2.
12. Malone DA Jr, Dimeff RJ. The use of fluoxetine in depression associated with anabolic steroid withdrawal: a case series. *J Clin Psychiatry*. 1992;53:130–2.
13. Pope HG Jr, Gruber AJ, Choi PY. Muscle dysmorphia: an underrecognized form of body dysmorphic disorder. *Psychosomatics* 1997;38:548–57.
14. Olivardia R, Pope HG Jr, Hudson JI. 'Muscle dysmorphia' in male weightlifters: a case-control study. *Am J Psychiatry* 2000;157:1291–6.

15. Kanayama G, Pope HG Jr, Cohane G, Hudson JI. Risk factors for anabolic-androgenic steroid use among weightlifters: a case-control study. *Drug Alcohol Depend* 2003;71:77–86.
16. Phillips KA. Pharmacologic treatment of body dysmorphic disorder: a review of empirical data and a proposed treatment algorithm. *Psychiatr Clin North Am* 2000;7:59–82.
17. Wines JD Jr, Gruber AJ, Pope HG Jr, Lukas SE. Nalbuphine hydrochloride dependence in anabolic steroid users. *Am J Addictions* 1999;8:161–4.
18. McBride AJ, Williamson K, Petersen T. Three cases of nalbuphine hydrochloride dependence associated with anabolic steroid abuse. *Br J Sports Med* 1996;30:69–70.
19. Kanayama G, Cohane G, Weiss RD, Pope HG Jr. Past anabolic-androgenic steroid use among men admitted for substance abuse treatment: an underrecognized problem? *J Clin Psychiatry*. 2003;64:156–60.
20. Arvary D, Pope HG Jr. Anabolic steroids: a possible gateway to opioid dependence. *N Engl J Med* 2000;342:1532.