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Cardiac and Metabolic Effects of Anabolic-Androgenic Steroid Abuse on Lipids, Blood Pressure, Left Ventricular Dimensions, and Rhythm

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Abstract

Recent surveys and reports suggest that many athletes and bodybuilders abuse anabolic-androgenic steroids (AAS). However, scientific data on the cardiac and metabolic complications of AAS abuse are divergent and often conflicting. A total of 49 studies describing 1,467 athletes were reviewed to investigate the cardiovascular effects of the abuse of AAS. Although studies were typically small and retrospective, some associated AAS abuse with unfavorable effects. Otherwise healthy young athletes abusing AAS may show elevated levels of low-density lipoprotein and low levels of high-density lipoprotein. Although data are conflicting, AAS have also been linked with elevated systolic and diastolic blood pressure and with left ventricular hypertrophy that may persist after AAS cessation. Finally, in small case studies, AAS abuse has been linked with acute myocardial infarction and fatal ventricular arrhythmias. In conclusion, recognition of these adverse effects may improve the education of athletes and increase vigilance when evaluating young athletes with cardiovascular abnormalities.

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone that were originally developed in the late 1930s.[1] At present, the United States Food and Drug Administration has approved a variety of AAS to treat wasting syndrome in human immunodeficiency virus infection, hypogonadism, anemia accompanying renal and bone marrow failure, endometriosis, and cancer.[2,3] Unfortunately, AAS are frequently abused and have recently been linked to the tragic deaths of celebrated professional athletes in the United States.[4,5] Indeed, recent estimates suggest that >3 million individuals in the United States abuse AAS, including nandrolone decanoate, methandienone, stanozolol, androsterone, and androstane.[6,7] This rampant abuse led Congress to enact the Anabolic Steroids Control Act in 1990, requiring that anabolic steroids be added to Schedule 3 of the Controlled Substances Act.[8,9] All major professional sports organizations ban the use of AAS. Regardless, a recent report by Mitchell et al[10] showed that >29 major league baseball players tested positive for AAS abuse within the past 4 years. Many effects of AAS abuse are unclear. Although side effects are rare at therapeutic doses, abusers typically use 5 to 15 times the recommended clinical doses of AAS.[6,11,12] At such doses, general adverse effects include dose-dependent suppression of testicular function, gynecomastia, hepatotoxicity, and

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psychologic disorders.[6,11,12] Cardiac and metabolic effects of AAS abuse are particularly unclear, although there are alarming reports of cardiac morbidity and mortality. Moreover, athletes often abuse AAS for years, prolonging the potential for harm.[13–15] The purpose of this review is to synthesize the recent published reports on the cardiac and metabolic effects of AAS abuse in athletes.

Methods

We reviewed human studies retrieved from the PubMed, eMedicine, Heart Online, and Cochrane Databases in the English language. Inclusion terms were “anabolic steroid,” “body builder,” “athlete,” and “steroid user,” used alone or in combination with the terms “ventricular hypertrophy,” “hypertension,” “lipoprotein,” “sudden death,” “myocardial infarction” (MI), “cardiac,” “arrhythmia,” “tachycardia,” and “fibrillation.” The only exclusion term was “animal.” In turn, a review of primary sources for each report was also conducted to find additional sources pertaining to their parent topics. Review of published reports was limited to the period from January 1, 1987, to December 31, 2009, because widespread testing became available in the United States and Europe at the end of 1986.

Results

We retrieved a total of 49 reports describing a total of 1,467 athletes (median 15 subjects/study). In aggregate, studies evaluated lipoprotein concentrations in 643 subjects, blood pressure in 348, left ventricular (LV) dimensions in 561, and sudden death in 102. We also report 4 key animal studies whose results shed insights into potential mechanisms linking AAS abuse with cardiovascular disease.

Clinical pharmacology of AAS

AAS include many agents with chemical structures derived from cholesterol that are synthesized in the liver and then metabolized in the adrenal glands and testes to AAS. Their structure resembles that of corticosteroids, explaining some similarities in actions in terms of renal sodium retention and hypertension.

The public health problem: prevalence of AAS abuse

AAS are abused by athletes primarily to increase lean muscle mass, enhance appearance, and improve performance.[16–18] Self-reported rates of abuse in bodybuilders range from 29% to 67%.[19–21] In a 1996 British survey of steroid abuse in competitive gymnasiums (albeit with few women), 29% of respondents admitted using AAS.[19] In an American study of 380 competitive bodybuilders in 1989, 54% of men and 10% of women admitted using AAS on a regular basis,[20] while 10 of 15 bodybuilders from an American power-lifting team admitted to taking AAS in a more recent study.[21]

Mortality in AAS abuse: the importance of cardiovascular causes

Mortality appears to be significantly higher in AAS abusers than in nonabusing athletes. In a retrospective case-cohort study of 248 AAS users and 1,215 controls (average age 23 years), 12 AAS users died during the study period,[22] providing a standard mortality ratio of 20.43

(95% confidence interval 10.56 to 35.70). Of the 1,215 athletes who did not abuse AAS, 22 died during the study period, resulting in a standard mortality ratio of 6.02 (95% confidence interval 3.77 to 9.12).[22] Although the exact causes of death were difficult to ascertain, a postmortem study of male Caucasian AAS abusers (aged 20 to 45 years) suggested primary cardiac pathology in 1/3,[23] while a recent case-control study[24,25] suggested cardiac causes in 2/3 of deaths, with others being attributed to suicide, hepatic coma, and malignancy. Many mechanisms have been proposed to explain potential adverse cardiovascular events of AAS.

Potential mechanisms

The physiologic and pharmacologic mechanisms of action of AAS on vascular structure and function are incompletely understood. AAS bind to androgen receptors in the heart and major arteries,[26] and physiologic levels (e.g., of testosterone) may have a beneficial effect on coronary arteries via endothelial release of nitric oxide and inhibition of vascular smooth muscle tone.[27,28] Conversely, animal studies show that abused AAS such as nandrolone at appropriately high doses may reverse this vasodilator response and lead to growth-promoting effects on cardiac tissue, as seen in hypertrophic cardiomyopathy, followed by apoptotic cell death.[29–31] These effects are likely mediated by membrane receptor–second messenger cascades that increase intracellular Ca^{2+} influx and Ca^{2+} mobilization from the sarcoplasmic reticulum.[32] Increases in Ca^{2+} affect mitochondrial permeability, leading to the release of apoptogenic factors such as holocytochrome c, apoptosis-inducing factor, and caspase-9.[33] Notably, AAS dosing associated with sudden cardiac death, MI, ventricular remodeling, and cardiomyopathy is related to apoptosis.[30] These findings may explain clinical observations that AAS can lead to myocardial death without coronary thrombosis or atherosclerosis.[34,35]

AAS and abnormal plasma lipoproteins

AAS abuse has been linked with abnormal plasma lipoproteins (Table 1). Several studies suggest that AAS abuse in athletes increase low-density lipoprotein (LDL) levels by >20%[14,36–38] and decrease high-density lipoprotein (HDL) levels by 20% to 70%.[13,14,37,39–44] More generally, steroid hormones alter serum lipoprotein levels via the lipolytic degradation of lipoproteins and their removal by receptors through modification of apolipoprotein A-I and B synthesis. Although some studies have shown an association between AAS and elevated LDL, no definitive mechanism has been established. Baldo-Enzi et al[39] suggested that serum LDL levels may increase through the induction of the enzyme hepatic triglyceride lipase and catabolism of very low density lipoprotein. Hepatic triglyceride lipase induction may also catabolize HDL and reduce its serum levels.[45] By some estimates, these lipoprotein abnormalities increase the risk for coronary artery disease by three- to sixfold.[14,45]

In a study of 88 bodybuilders who tested positive for AAS, Lenders et al[14] showed that AAS abusers had significantly higher LDL and lower HDL levels than nonabusers. Other studies have confirmed these effects (Table 1). Although actual lipoprotein levels vary among studies, LDL levels as high as 596 mg/dl[46] and HDL levels as low as 14 mg/dl[46] have been noted in otherwise healthy athletes who abuse AAS. We have observed a markedly low HDL level of 5 mg/dl in a bodybuilder who admitted to AAS abuse (unpublished

observations). Abnormalities of HDL and LDL may arise within 9 weeks of AAS self-administration (Table 1).[14] This time of onset and duration is supported in numerous studies.[13,36–42,44,47–57] Fortunately, lipid effects seem to be reversible (Table 1)[14,42,55] and may normalize 5 months after discontinuation.[14] Nevertheless, further studies are warranted, because the duration of effect is longer than would be expected from the terminal half-lives of these agents (typically 7 to 12 days).[58]

AAS may elevate blood pressure

The relation between AAS abuse and blood pressure is controversial. A link between AAS abuse and elevated blood pressure has been observed in some studies,[14,43,59,60] whereas others have shown no association.[13,38,42,59,61–67] When hypertension is observed, it likely follows renal retention of sodium from AAS.[12,68] Blood pressure response to AAS abuse typically shows a dose-response relation. In a retrospective study, Urhausen et al[43] reported that mean arterial pressure in AAS users was elevated, in the prehypertensive and stage I hypertensive range as defined in the "Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure," compared to former users or nonusers. Other studies support these data (Table 2). Although actual elevations vary (Table 2), blood pressures as high as 195/110 mm Hg have been recorded in otherwise healthy athletes with no other identifiable cause.[69] Again, the effects of AAS abuse on blood pressure may persist for long periods; some studies have shown persistent elevations for 5 to 12 months after discontinuing AAS.[14,43] Although this may reflect the prolonged half-lives of depot AAS preparations,[58] it may also reflect the fact that self-reporting of discontinuation is unreliable. In some studies, AAS remain detectable after self-reported discontinuation.[14,43,58] Furthermore, there is variability in dosing regimens and supplemental substance use in numerous studies.[14,43,59]

Importantly, the link between AAS abuse and elevated blood pressure is not seen in all studies.[13,38,42,59,61–67] In a small cross-sectional study, Palatini et al[38] did not find any difference in blood pressure between 10 AAS users and 14 age-matched controls. Measurements were made when users were taking AAS and during the withdrawal stage of cycling. Misclassification of athletes was minimized by measuring gonadratropin levels, follicle-stimulating hormone, and luteinizing hormone, which decreased significantly in users. In another cross-sectional study by D'Andrea et al,[61] blinded blood pressure measurements in 20 AAS-abusing athletes did not differ significantly from those in 25 age-matched AAS-free bodybuilders using the same exercise protocol, although blood pressure was non-significantly elevated ($p > 0.05$). Moreover, Lenders et al[14] did not show elevated blood pressure in AAS users compared with nonusers in a larger population. Possible explanations include lower AAS doses in those abusers or, speculatively, undeclared abuse in the "control" population in whom occult AAS abuse was not tested for. Another confounding variable that some investigators neglect to report relates to cuff size. In athletes with larger arms, regular sized blood pressure cuffs could overestimate blood pressure. Analysis is complicated further by variability in exercise regimens, variability in dosing and duration of AAS use, and potential biases in unblended studies. Clearly, additional studies are necessary to definitively reveal a link between AAS and blood pressure.

AAS and LV hypertrophy (LVH)

Athletes abusing AAS often exhibit LVH (Table 3).[13,15,43,64,65,70,71] However, because the hypertrophy may relate to increased afterload from isometric exercise,[72] the interpretation of LVH in elite athletes who admit to AAS abuse is complex. Possible associations between AAS and LVH may be explained as secondary to hypertension or as a direct effect on the myocardium. Notably, studies in isolated human myocytes have shown that AAS bind to androgen receptors and may directly cause hypertrophy,[73–75] potentially via tissue up-regulation of the renin-angiotensin system.[76] Indeed, clinical studies suggest a distinct form of LVH in AAS abusers, suggested by textural changes in the myocardium on echocardiography before the onset of overt LVH.[59]

In a retrospective case-control study, Krieg et al[65] performed nonblinded echocardiographic measurements on 14 AAS users, 11 age-matched nonuser strength athletes, and 15 age-matched sedentary controls. Self-reported use of oral and injectable AAS was confirmed by luteinizing hormone and follicle-stimulating hormone assay and elevated testosterone/epitestosterone ratios. AAS users had significantly enlarged interventricular septal wall thickness on echocardiography compared to nonusers and controls ($p < 0.05$), although posterior wall thickness was only slightly larger. In contrast, Dickerman et al[15] and others[77] have reported that AAS abuse is associated with increased LV posterior wall and interventricular septal thickness compared to non-abusing athletes (Table 3). A variety of agents and doses were noted in the abusers. However, these studies are somewhat difficult to interpret, because most studies did not account for differences in exercise protocols, and most were not blinded.[13,44,61,65,70]

Several studies have confirmed that LVH may directly follow resistance training in the absence of AAS use.[71,77–79] D'Andrea et al[80] used nonblinded echocardiographic measurement to find that the thickness of the interventricular septum (12.3 ± 2.4 vs 9.7 ± 3.1 mm, $p < 0.01$) and posterior LV wall (11.6 ± 1.6 vs 9.2 ± 2.1 mm, $p < 0.05$) were greater in 130 strength-trained compared to 160 endurance-trained athletes. As always, however, an important caveat is the lack of control for occult AAS abuse.[38,44,47,59,61–63,67,81]

The few randomized clinical trials investigating the association between AAS use and LVH have similar limitations. Chung et al[82] conducted a randomized, double-blind, placebo-controlled study in which 30 healthy men were separated into 3 groups receiving weekly testosterone 200 mg, nandrolone decanoate 200 mg, or matching placebo injections for 4 weeks. After 4 weeks, blinded echocardiography showed that only the testosterone group had a significant increase in LV end-systolic diameter, although this remained within the normal range. In the only other randomized prospective study designed to evaluate the impact of AAS on cardiac dimensions, no changes were noted between strength athletes receiving 8 weeks of nandrolone at 200 mg/week compared to controls.[63] There is a need for a closely monitored observational echocardiographic study over an extended period to compare cardiac morphology between AAS abusers and strength athletes, with appropriate control for occult AAS use.

Studies observing LVH related to AAS abuse reported that it likely reverses after discontinuing the agent, although with persistent effects for a prolonged period. In 1 study,

echocardiography showed that LVH persisted for 9 ± 2 weeks after AAS withdrawal,[62] although most AAS have half-lives <12 days.[58] Although some investigators have suggested that concentric LVH may persist for years after discontinuing AAS,[43] it is also possible that this observation reflects LVH secondary to strength training (or, again, potential occult AAS use).

AAS, acute MI, and sudden death

Alarming data have linked AAS with fatal events, although these are mostly case-control studies and case reports of acute coronary syndromes, MIs, and ventricular arrhythmias.[24,46,83–88]

In the absence of carefully conducted animal experiments, it is felt that AAS abuse may cause cardiac ischemia by exaggerating oxygen demand at peak exercise, potentially precipitated by accelerated atherosclerosis from lipoprotein abnormalities over years of abuse.[89] As 1 potential direct mechanism, AAS also enhance platelet aggregation and thrombus formation by increasing platelet production of thromboxane A_2 (a potent platelet aggregator), decreasing production of prostacyclin (prostaglandin I_2, an inhibitor of platelet aggregation) and increasing fibrinogen levels.[90] As stated previously, these detrimental effects must be weighed against the potentially beneficial effect of low-dose AAS on vasoreactivity.

The relative clinical contributions of these mechanisms are unclear. Nevertheless, their combination may plausibly explain MIs or ventricular arrhythmias in young athletes with no cardiac risk factors,[83,91] in many of whom AAS abuse has been ruled the primary cause of death.[92–94] McNutt et al[46] reported an acute MI in a 22-year-old bodybuilder who admitted to AAS abuse but lacked cardiac risk factors. The patient presented with markedly elevated LDL (596 mg/dl) and depressed HDL (14 mg/dl) yet had no family history of premature atherosclerosis or cardiac events. Within a month of discontinuing AAS, his LDL decreased to 220 mg/dl and his HDL increased to 35 mg/dl. Similar cases have been reported by others.[69] Figure 1 shows the presenting electrocardiogram of a 25-year-old male athlete who abused nandrolone and presented with an acute MI without traditional cardiac risk factors. Acute angiography revealed extensive left anterior descending coronary artery thrombosis, which was managed by thrombolysis. Angiography in the subacute phase confirmed very mild luminal irregularity at the site of previous thrombosis.

Finally, there are numerous anecdotes of potentially lethal ventricular arrhythmias in AAS-abusers. The most commonly observed arrhythmias, typically occurring during physical exertion, include atrial fibrillation, ventricular fibrillation, ventricular tachycardia, and supraventricular and ventricular ectopic beats.[95] Hausmann et al[84] describe a 23-year-old male bodybuilder who abused AAS and experienced sudden cardiac arrest. Postmortem examination revealed ventricular hypertrophy, myocardial fibrosis, and acute MI, and the cause of death was attributed to arrhythmic sudden death secondary to AAS abuse. Dickerman et al[96] reported a 20-year-old male bodybuilder who self-administered AAS 700 mg/week and had sudden cardiac arrest. Autopsy indicated LVH with a cardiac mass >2 times the upper limit of normal. More organized case series are needed to define to what

extent such cases represent occult forms of hypertrophic cardiomyopathy or other arrhythmogenic conditions.

Discussion

Limitations

This review included 1,467 subjects, but the major variables discussed included only a fraction of these subjects. Many studies that were reviewed were observational, cross-sectional studies with small sample sizes and single case reports that may explain variability in the reported data. Although prospective, randomized controlled studies would be ideal, such studies are also difficult to conduct because they must control for occult AAS use and recruit large samples of competitive athletes willing to disclose their (illegal) use of AAS. Many studies were not blinded, particularly with regard to blood pressure and echocardiographic assessment. Others did not account for variability in AAS dosage and cumulative duration of use or compared dissimilar exercise regimens (e.g., those that involve more resistance training can accentuate LVH). Many studies also did not specify the reasons for the discontinuation of AAS and whether such athletes continued exercising to the same degree. In addition, most studies followed AAS abusers for short periods, and many were too small to perform multivariate corrections for other risk factors.

Some studies on the adverse cardiometabolic effects of AAS may also reflect publication bias. Finally, AAS are often coabused with growth hormone, erythropoietin, and agents including creatine, ephedra alkaloids, and herbal supplements, although most AAS reports neither documented nor controlled for these agents. This is important because growth hormone may lead to cardiomyopathy, abnormal lipoprotein profiles,[97,98] and LVH.[64] Erythropoietin abuse is similarly linked to hypertension and increased risk for thromboembolic events.[99] Such effects may be difficult to separate from the results of AAS abuse alone and motivate the need for more rigorous clinical screening. Nevertheless, AAS alter lipids, may affect LV dimensions, and have an unclear role in blood pressure elevation in athletes. Physicians should consider the possibility of AAS abuse when treating young athletes with abnormal lipids, LV dimensions, and potentially even blood pressure elevation.

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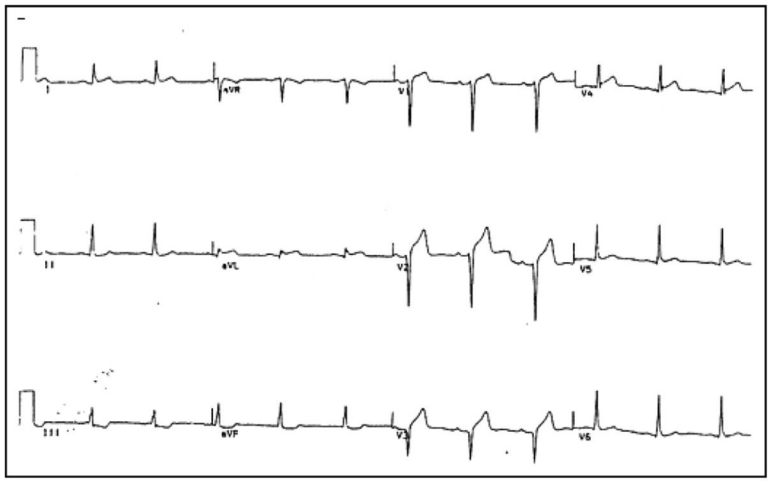

Figure 1.

Electrocardiogram of a 25-year-old male athlete presenting with acute MI, showing anterior precordial Q waves and ST-segment elevation. The patient, who admitted to recent nandrolone abuse, had been previously healthy and without cardiac risk factors. Angiography revealed thrombosis in the left anterior descending coronary artery but no significant atherosclerotic narrowing. Reproduced from *Recent Prog Horm Res*.[74]

Table 1

Effects of anabolic-androgenic steroid abuse on lipoprotein concentration

Study	Abused Agent	Dosage of AAS (mg/ week)	Subjects, Age (years)			
			Users Ex-Users Controls	Users LDL (mg/dl) HDL (mg/dl)	Ex-users LDL (mg/dl) HDL (mg/dl)	Controls LDL (mg/dl) HDL (mg/dl)
Baldo-Enzi et al[39],¶	Methenolone enanthate	100–300	14, 27 ± 5	129 ± 37	—	119 ± 17
	Testosterone cypionate	200–300	17, 25 ± 4	27 ± 11‖	—	48 ± 6
Fröhlich et al[40]	—	—	13, 27 ± 4	154 ± 58	—	121 ± 22
			11, 27 ± 7	23 ± 16‡	—	34 ± 7
Hartgens et al[41],¶	Stanozolol	30–140	19, 31 ± 7	—	—	—
	Nandrolone decanoate	8–250	—	17 ± 9‖	—	47 ± 22
			16, 33 ± 5			
Lajarin et al[36]	Stanozolol	50–100	2, 27 ± 3	238 ± 8	—	—
	Methenolone enanthate	100	—	14 ± 0.4	—	—
			—			
Lane et al[42]	Testosterone	—	10, 26 ± 7	113 ± 27	86 ± 23	82 ± 12
	Nandrolone		8, 32 ± 7	27 ± 16†,‖	51 ± 16	51 ± 12
	Stanozolol		10, 24 ± 4			
Lenders et al[14],¶	Methenolone	385–690	20, 26 ± 8	206 ± 21*,‡	156 ± 9	130 ± 13
	Testosterone	310–355	42, 28 ± 7	27 ± 3†,‖	42 ± 2	46 ± 2
	Oxymetholone	580–650	13, 28 ± 5			
McKillop and	Stanozolol	280	8, 25 ± 4	243 ± 50‖	—	122 ± 27
Ballantyne[37]	Nandrolone decanoate	200	—	16 ± 11‖	—	43 ± 12
			8, 25 ± 3			
Palatini et al[38],¶	Testosterone enanthate and propionate	50–1,500	10, 27 ± 8	153 ± 34§	—	107 ± 41
	Stanozolol	50–150	—	30 ± 10	—	57 ± 13
			14, 28 ± 5			
Sader et al[13]	Stanozolol	—	10, 37 ± 3	—	—	—
	Nandrolone		—	23 ± 4‖	—	55 ± 4
	Creatine		10, 34 ± 3			

Study	Abused Agent	Dosage of AAS (mg/week)	Subjects, Age (years)			
			Users Ex-Users Controls	Users LDL (mg/dl) HDL (mg/dl)	Ex-users LDL (mg/dl) HDL (mg/dl)	Controls LDL (mg/dl) HDL (mg/dl)
Urhausen et al[43]	Oral (i.e., mesterolone) and intramuscular	1,030	17, 31 ± 5	139 ± 37	119 ± 30	—
			15, 38 ± 7	17 ± 11†	43 ± 11	—
	AAS (i.e., stanozolol, nandrolone)		—			
Zuliani et al[44]	Testosterone enanthate and propionate	750–1,500	6, 28 ± 2	—	—	—
			—	19 ± 8‖	—	49 ± 6
	Human growth hormone		8, 26 ± 2			

Data are expressed as ranges, numbers, or mean ± SD. The control group included bodybuilders who denied AAS abuse.

*p <0.05 and

†p <0.001 versus ex-users;

‡p <0.05,

§p <0.01, and

‖p <0.001 versus controls.

¶Other unspecified AAS abused.

Table 2

Effects of anabolic-androgenic steroids on blood pressure

Study	Abused Agent	Dosage (mg/week)	Subjects, Age (years) Users Ex-Users Controls	Blood Pressure (mm Hg)		
				Users Systolic Diastolic	Ex-Users Systolic Diastolic	Controls Systolic Diastolic
D'Andrea et al[61],‖	—	525 (90)	20, 35 ± 3	140 ± 7	—	131 ± 9
			—	84 ± 4	—	81 ± 5
			25, 34 ± 3			
De Piccoli et al[62]	—	—	14, 26 ± 5	142 ± 11	140 ± 10	136 ± 12
			9, 26 ± 5	83 ± 5	83 ± 5	87 ± 9
			14, 26 ± 4			
Di Bello et al[59]	Testosterone propionate	300–500	10, 33 ± 3	135 ± 19	—	138 ± 8
	Methenolone enanthate	300–600	—	89 ± 12‡	—	87 ± 8
	Testosterone cypionate	200–350	10, 30 ± 7			
Hartgens et al[63],‖	Nandrolone decanoate	20–250	17, 32 ± 7	139 ± 13	—	134 ± 8
	Stanozolol	30–140	—	85 ± 12	—	81 ± 7
			15, 33 ± 5			
Karila et al[64],‖	—	770 (310)	16, 30 ± 5	131 ± 13	—	131 ± 13
			—	76 ± 10	—	77 ± 9
			15, 26 ± 3			
Krieg et al[65],‖	—	820 (620)	14, 36 ± 7	135 ± 10	—	130 ± 5
			—	85 ± 5	—	85 ± 5
			11, 36 ± 11			
Kuipers et al[66]	Nandrolone decanoate	200–400	7	134 ± 14	—	127 ± 11
	Testosterone	2000	—	86 ± 14	—	74 ± 8
	Stanozolol	150–300	6			
Lane et al[42]	Testosterone		10, 27 ± 7	119 ± 7	121 ± 7	125 ± 13
	Nandrolone		8, 32 ± 7	81 ± 4	67 ± 18	72 ± 14
	Stanozolol		10, 24 ± 4			
Lenders et al[14],‖	Methenolone	385–690	20, 26 ± 8	121 ± 2†	119 ± 2†	114 ± 2
	Testosterone	310–360	42, 28 ± 7	74 ± 2	72 ± 1	71 ± 2
	Oxymetholone	580–650	13, 28 ± 5			

Study	Abused Agent	Dosage (mg/week)	Subjects, Age (years) Users Ex-Users Controls	Blood Pressure (mm Hg)		
				Users Systolic Diastolic	Ex-Users Systolic Diastolic	Controls Systolic Diastolic
Nottin et al[67]	—		6, 41 ± 6	132 ± 10	—	122 ± 11
			—	87 ± 8	—	77 ± 12
			9, 38 ± 6			
Palatini et al[38],‖	Testosterone enanthate and propionate	50–1,500	10, 27 ± 8	124 ± 14	—	128 ± 11
	Stanozolol	50–150	—	80 ± 14	—	74 ± 7
			14, 28 ± 5			
Riebe et al[60],‖	Stanozolol	110–200	9, 25 ± 4	133 ± 8†	—	
	Testosterone	250–400	—	83 ± 7	—	123 ± 10
	Nandrolone decanoate	200–400	10, 28 ± 4			77 ± 7
Sader et al[13]	Stanozolol		10, 37 ± 3	127 ± 3	—	119 ± 4
	Nandrolone		—	74 ± 5	—	71 ± 5
	Creatine		10, 34 ± 3			
Urhausen et al[43],‖	—	1,030	17, 31 ± 5	140 ± 10*,§	130 ± 5	125 ± 10
			15, 38 ± 7	85 ± 10	85 ± 5	80 ± 10
			15, 28 ± 5			

Data are expressed as ranges, numbers, or mean ± SD. The control group included bodybuilders who denied AAS abuse.

*p <0.05 versus ex-users;

†p <0.05,

‡p <0.01, and

§p <0.001 versus controls.

‖Other unspecified AAS abused.

Table 3

Effects of anabolic-androgenic steroid abuse on cardiac dimensions

Study	Abused Agent	Dosage (mg/week)	Subjects, Age (years) Users Ex-Users Controls	IVS (mm)			PW (mm)		
				Users	Ex-Users	Controls	Users	Ex-Users	Controls
D'Andrea et al[61],‖	—	525 (91)	20, 35 (3)	12.3 (1.3)	—	11.2 (2.1)	11.8 (1.4)	—	10.4 (2.1)
			—						
			25, 34 (3)						
De Piccoli et al[62]	—	—	14, 26 (5)	11 (0.8)	10.6 (1.0)	10.5 (0.8)	10.3 (0.8)	9.8 (0.9)	9.8 (0.7)
			9, 26 (5)						
			14, 26 (4)						
Di Bello et al[59]	Testosterone propionate	300–500	10, 33 (3)	12.3 (0.7)	—	12.2 (0.4)	11.6 (0.5)	—	11.7 (0.4)
	Methenolone enanthate	300–600	—						
	Testosterone cypionate	200–350	10, 30 (7)						
Dickerman et al[15]	—	—	8	11.27 (0.2)†	—	8.74 (2.5)	12.1 (1.0)†	—	10.3 (2.0)
			—						
			8						
Hartgens et al[63],‖	Nandrolone decanoate	20–250	17, 32 (7)	8.8 (1.1)	—	8.3 (1.0)	8.9 (0.7)	—	8.6 (0.8)
	Stanozolol	30–140	—						
			15, 33 (5)						
Karila et al[64],‖	—	770 (310)	16, 30 (5)	11.2 (1.0)‡	—	8.9 (1.1)	11.3 (1.1)‡	—	9.1 (1.0)
			—						
			15, 26 (3)						
Krieg et al[65],‖	—	820 (620)	36 (7)	12 (1.5)†	—	10.5 (1.0)	10.5 (1.5)	—	10 (0.5)
			—						
			36 (11)						
Nieminen et al[47],‖	Testosterone	2,860¶	4, 30 (3)	12.75 (1.5)	—	—	13.75 (1.3)	—	—
	Testosterone undecanoate	2,660¶	—						
			—						
Nottin et al[67]	—	—	6, 41 (6)	10.8 (1.3)	—	9.7 (1.7)	10.0 (1.4)	—	10.3 (0.9)
			—						
			9, 38 (6)						

Study	Abused Agent	Dosage (mg/week)	Subjects, Age (years) Users Ex-Users Controls	IVS (mm)			PW (mm)		
				Users	Ex-Users	Controls	Users	Ex-Users	Controls
Palatini et al[38],‖	Testosterone enanthate and propionate	50–1,500	10, 27 (8)	10.8 (2.3)	—	9.6 (0.8)	10.4 (2.3)	—	10.1 (1.3)
	Stanozolol	50–150	—						
			14, 28 (5)						
Sachtleben et al[70]	Stanozolol	—	11, 27 (6)	11.1 (1.2)*	—	9.3 (1.2)	11.2 (1.5)*	—	9.5 (1.6)
	Methandrostenolone nandrolone		—						
	Testosterone cypionate		13, 27 (6)						
Sader et al[13]	Stanozolol	—	10, 37 (3)	10 (0.3)‡	—	8.7 (0.2)	9/8 (0.4)†	—	8.7 (0.3)
	Nandrolone		—						
	Creatine		10, 34 (3)						
Thompson et al[81],‖	Nandrolone decanoate	—	12, 23 (4)	10 (2.0)	—	9.0 (1.0)	8.0 (1.0)	—	8 (1.0)
	Testosterone cypionate		—						
	Stanozolol		11, 26 (7)						
Urhausen et al[43],‖	—	1,030	17, 31 (5)	12.3 (1.4)§	11.5 (1.2)†	10.3 (1.0)	11.4 (1.3)*,§	10.2 (0.8)	9.4 (1.5)
			15, 38 (7)						
			15, 28 (5)						
Urhausen et al[71]	Methandione, stanozolol	630	14, 28 (6)	12.6 (1.7)	—	11.6 (0.9)	12.5 (1.2)‡	—	10.3 (1.8)
	Testosterone depot		—						
				7, 26 (5)					
Zuliani et al[44],‖	Testosterone enanthate and propionate	750–1,500	6, 28 (2)	11.8 (0.8)	—	11.2 (0.7)	10.8 (0.7)	—	10.3 (0.5)
	Human growth hormone		—						
			8, 26 (2)						

Data are expressed as ranges, numbers, or mean ± SD. The control group included bodybuilders who denied AAS abuse.

*p <0.05 versus ex-users;

†p <0.05,

‡p <0.01, and

§p <0.001 versus controls.

‖Other unspecified AAS abused.

¶Milligrams per international unit.

IVS = interventricular septum; PW = posterior wall.