The use of anabolic-androgenic steroids (AAS) among athletes is not new, nor is concern about their potential cardiac effects, but it has been difficult to definitively document deleterious cardiovascular effects from these drugs. There are case reports of unexpected myocardial infarctions and even sudden cardiac death in AAS users, but such reports are relatively rare given the reported widespread use of AAS. Moreover, their effects on cardiovascular risk factors are confusing. Oral synthetic steroids, such as stanozolol, reduce high-density lipoprotein and increase low-density lipoprotein cholesterol more than parenterally administered testosterone at similar androgenic doses, suggesting that oral AAS are more atherogenic, but both stanozolol and testosterone decrease lipoprotein (a), an important atherosclerotic risk factor. There is also concern that AAS increase blood pressure, but even the literature on this topic is equivocal, and some of the purported increase in blood pressure with AAS may be due to the use of undersized sphygmomanometer cuffs in subjects with increased arm circumference. Consequently, the overall clinical effect of AAS use on atherosclerotic risk and events is not clear.

AAS have more consistently been shown to impair left ventricular (LV) diastolic function, and these clinical studies are supported by pathological evidence of increased myocardial collagen content after exposure to AAS. Evidence of LV systolic dysfunction with AAS use has been evasive, but recent studies using measures of myocardial strain suggest that AAS also subtly impair cardiac systolic performance.

In contrast to these subtle effects of AAS on systolic function, Baggish et al in this issue of Circulation: Heart Failure present evidence that chronic, high-dose AAS use produces a dramatic impairment of LV systolic function. These authors recruited 19 male weightlifters, including 12 with prolonged AAS use. Recruitment was designed to minimize selection biases. Standardized questionnaires were used to determine participants’ lifetime AAS exposure as well as their use of illicit drugs and other ergogenic drugs including growth hormone.

The AAS users were remarkable for both their steroid dose and duration of use. For comparison, subjects in prior studies generally reported weekly AAS doses equivalent to 200 to 250 mg of testosterone, whereas subjects in the present study reported a median weekly dose equivalent to 675 mg of testosterone. The present subjects also reported a median of almost 9 years of steroid use.

These investigators confirmed the decreased early, and increased late, diastolic filling in the AAS users noted previously. LV hypertrophy did not differ between AAS users and control subjects. Both radial and longitudinal strain, measures of myocardial systolic function, were decreased in the AAS users, but more impressive was the decrease in LV ejection fraction (LVEF). Median LVEF was 51% in AAS users versus 59% in control subjects, and half of the AAS users had LVEF values below 50%.

This decreased in LVEF has not been observed in prior studies of AAS. There was no statistical association between dose and duration of AAS use and LVEF, but the small sample size precludes eliminating this possibility. We suspect that both the dose and duration of use are part of the explanation for the novelty of these findings, but this hypothesis awaits confirmation. It is difficult to separate duration of use from age, however, and the age (median, 40 years) of AAS users in the present study is older than in most prior reports.

There are limitations to the present report. The number of participants is small. The authors correctly note that this is more likely to cause a type II error, or false-negative finding, but this assumes that the effect is entirely due to the exposure being studied. Other causes of reduced LV systolic function, such as other drug use, could be clustered in the AAS subjects, confounding the results. Indeed, 9 of the AAS users reported prior growth hormone (GH) use and 6 had used GH for at least 3 months. Supraphysiologic levels of GH have been associated with diastolic and systolic dysfunction, although the later requires chronic exposure. Consequently, GH use may be a significant confounder. The authors tried to reduce selection bias, but the possibility remains that participants enrolled for a medical evaluation because of mild symptoms. Blood pressure, ECGs, and an assessment of overall atherosclerotic risk were not provided, so occult atherosclerotic disease cannot be excluded as a mechanism for the effect; however, none of the participants reported hypertension, atherosclerotic disease, or heart failure.

Despite such limitations, the present report by Baggish et al is the first to suggest that chronic AAS use can reduce LVEF. These results, if confirmed, require that AAS use be consid-
ered in the differential diagnosis of LV systolic dysfunction and suggest that the prevalence of this problem may increase as long-term AAS users reach middle age. These results also raise the haunting possibility that long-term AAS use can produce a clinically symptomatic cardiomyopathy. This report might spur the detection of such cases and the confirmation of this possibility.

Disclosures

None.

References


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Anabolic-Androgenic Steroids: Worse for the Heart Than We Knew?
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