Acute metabolic effects of exercise in bodybuilders using anabolic steroids

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Four male bodybuilders who had started taking anabolic steroids were monitored during exercise. Most metabolic indicators were similar to bodybuilders not taking steroids; i.e. metabolic acidosis with little change in glucose. However, there is a marked elevation of creatine kinase.

Keywords: Anabolic steroids, exercise, bodybuilders, acute metabolic effects

Introduction

The acute metabolic effects of strenuous exercise have been studied in several groups of athletes because of the stressful nature of such activity. We have previously reported the acute effects in bodybuilders who train specifically to develop maximum muscle hypertrophy using weightlifting and diet. However, some bodybuilders also use anabolic steroids believing that they enhance muscle hypertrophy. Initially we studied bodybuilders who were not taking steroids¹. When these athletes decided to start taking anabolic steroids, they asked us to monitor their performance. We assessed the metabolic changes of acute exercise while they were taking anabolic steroids.

Subjects and methods

Four male bodybuilders (age range 24–31 yr) were studied. All had been training regularly for at least eight hours per week for more than four years. None had previously taken anabolic steroids, but all had decided to use them and requested medical supervision while taking these drugs. The subjects were all informed of the known and theoretical complications of anabolic steroids use by a registered medical practitioner before obtaining the drugs from a source unknown to us.

All drugs, both oral and intramuscular, were examined by medical staff and an attempt was made to verify that these were genuine by comparing them with pharmacy stores and relevant drug company literature. All subjects decided to take the same dose schedule of drugs i.e. 175 mg of stanozolol orally plus 2000 mg nandrolone decanoate intramuscularly per week for 12 weeks. All drugs were self administered.

The four subjects attended the medical department during week 12 to undergo an exercise programme and physical examination. The programme consisted of exercising all the major muscle groups in a 'super set' fashion. The same series of exercises was performed by all four subjects on a Universal Multigym with which they were familiar.

The exercise programme was the same as that car-

ried out in a previously reported study, although all of the subjects reported that the weights they used increased by between five and ten per cent. However, we did not objectively validate this, nor did we have access to a dynamometer to assess changes in muscular function.

The training programme was completed within 60 minutes and the subjects were then monitored for a recovery period of two hours. Venesection was carried out before exercise and at 15, 30, 45 and at 60 minutes (the end of the exercise). Recovery bloods were taken at 63, 66, 70, 90, 120 and 180 minutes. At each time 15 ml of blood was withdrawn. One millilitre was preserved in a fluoride/oxalate container for glucose and lactate. The remainder of the blood was allowed to clot in a plain container and the serum used for all other analyses.

Glucose was measured by a glucose oxidase method. Lactate was measured enzymatically by a change in the absorbance of NAD/NADH. Sodium, potassium, chloride, CO₂, urea, creatinine, calcium, phosphate and albumin were analysed by a Technicon SMAC II. Alkaline phosphatase (ALP) was measured by the change in absorbance of substrate (4-nitrophenyl phosphate). Aspartate transaminase (AST), alanine transaminase (ALT), lactate dehydrogenase (LD) and creatine kinase (CK) were measured by change in the absorbance of NAD/NADH. Transferrin and caeruloplasmin were estimated by immunoturbidimetry, zinc and copper by atomic absorption spectrophotometry. Iron was measured by the absorbance of the complex formed with ferrozine.

Due to the small sample size, assumptions required for statistical analysis could not be met. We therefore present a descriptive analysis.

Results

All analytes, except AST and CK, were initially within appropriate reference ranges. There was no change in sodium, potassium, chloride, glucose or urea. As shown in Table 1 creatinine increased during exercise. Lactate rose markedly during exercise and returned to within the reference range after 30 minutes. This was mirrored by a fall in CO₂ and entirely accounted for the rise in the cation-anion gap. Calcium, albumin, protein and phosphate all rose during exercise, in addition phosphate fell to below resting values after exercise.

The initial AST level was elevated, but did not rise during or after exercise. The pre-exercise CK level was markedly elevated and rose during exercise.

The zinc and caeruloplasmin levels fell steadily from 15 minutes of exercise and the former was still falling

Table 1. Metabolic changes in bodybuilders using anabolic steroids showing median plus range

Time (min)	Exercise					Recovery					
	0	15	30	45	60	63	66	70	90	120	180
CO ₂ (mmol.l ⁻¹)	19.5 (18–21)	13 (13–14)	10.5 (9–11)	11 (10–13)	12 (9–12)	12 (10–13)	13.5 (11–14)	14.5 (14–16)	17.5 (17–19)	19 (17–22)	20 (20–22)
Creatinine (μmol.l ⁻¹)	110 (100–120)	120 (110–140)	130 (130–140)	130 (130–150)	135 (120–140)	130 (120–140)	130 (120–130)	125 (120–140)	115 (110–130)	115 (110–130)	100 (90–130)
PO ₄ (mmol.l ⁻¹)	1.07 (1.05–1.20)	1.37 (1.3–1.65)	1.35 (1.2–1.55)	1.15 (1.1–1.25)	1.02 (0.9–1.15)	0.95 (0.85–1.05)	0.93 (0.7–1.00)	0.8 (0.75–0.90)	0.68 (0.65–0.80)	0.8 (0.75–0.80)	1.0 (0.95–1.05)
Albumin (g.l ⁻¹)	44 (40–46)	48 (44–51)	48.5 (45–53)	48.5 (44–51)	46.5 (43–50)	45.5 (42–50)	45 (41–49)	43.5 (41–47)	42.5 (40–45)	43.5 (40–45)	43.5 (39–45)
AST (U.l ⁻¹)	74 (43–136)	81 (48–124)	80.5 (48–141)	79 (44–129)	80 (43–140)	80 (51–131)	76 (46–145)	78 (45–135)	72 (45–114)	70.5 (44–113)	71.5 (42–119)
ALT (U.l ⁻¹)	45 (29–72)	49 (36–83)	51.5 (37–84)	50.5 (35–84)	48 (33–79)	48 (31–78)	49 (41–78)	48 (33–75)	48 (29–74)	49 (29–73)	49 (28–74)
CK (U.l ⁻¹)	1210 (690–4300)	1395 (790–4900)	1475 (840–5300)	1485 (850–5200)	1525 (840–5000)	1500 (840–4900)	1470 (830–4700)	1430 (830–4600)	1400 (810–4500)	1420 (820–4900)	1430 (820–4400)
Lactate (mmol.l ⁻¹)	2.35 (1.4–2.6)	9.6 (9.2–12.1)	12.9 (11.1–17.1)	13.1 (11.8–15.1)	10.6 (9.8–14.6)	9.4 (8.8–12.9)	7.8 (6.3–12.1)	6.2 (5.9–8.9)	2.9 (2.6–4.5)	1.85 (1–4)	1.0 (0.7–1)
Zinc (μmol.l ⁻¹)	14.5 (12.5–16.5)	13.5 (13–17.5)	15.2 (11–16.5)	13.5 (10.5–14.5)	13.5 (10.5–14.5)	12.8 (11.5–14)	12.5 (11–13.5)	12.0 (10.5–14)	11.5 (9.5–12.5)	11 (10.5–12)	11 (8.5–11.5)
Caeuloplasmin (mg.l ⁻¹)	294 (288–443)	300 (247–336)	281 (230–467)	308 (242–436)	306 (232–445)	295 (234–451)	271 (229–455)	286 (217–427)	244 (215–412)	236 (195–429)	261 (213–423)
Iron (μmol)	6.0 (5–10)	8.0 (5–13)	8.5 (6–13)	8.0 (6–13)	7.5 (6–14)	7.5 (6–13)	9.0 (6–13)	7.5 (5–14)	6.0 (3–13)	5.5 (3–15)	6.5 (3–12)
Transferrin (g.l ⁻¹)	3.15 (3.0–3.5)	3.5 (2.3–3.8)	3.5 (3.3–3.8)	3.45 (3–3.8)	3.45 (3–3.8)	3.45 (3.1–3.5)	3.25 (2.9–3.5)	3.2 (3–3.4)	3.05 (3–3.2)	3.15 (2.8–3.3)	3.1 (2.9–3.3)

at the time of the last sample i.e. two hours post-exercise. Serum iron rose during exercise and peaked six minutes post-exercise before returning to within the reference range. There was no marked change in transferrin.

Discussion

As Table 1 shows there were changes in metabolic parameters during and after the exercise period. Most of these findings were similar to those seen in our previous study of these bodybuilders when they were not taking steroids. The rise in lactate and fall in CO₂ reflected anaerobic metabolism which is sustained for at least 45 minutes of exercise.

However, the major differences pre- and post-anabolic steroids are in the basal levels of AST and CK. The elevation of AST has been previously reported in both normal subjects and athletes who take anabolic steroids², and we were unable to demonstrate any acute effect of exercise over and above this.

The marked elevation of CK before exercise has not been previously reported in athletes using anabolic steroids. Creatine kinase is the most sensitive enzyme index of muscle damage. It is an intracellular enzyme with three main isoenzymes and is found mainly in skeletal and cardiac muscle. It is commonly measured to assess damage to cardiac muscle post myocardial infarction or to skeletal muscle in myositis.

Studies on marathon runners^{3,4} show that plasma CK peaks 6–24 hours after the end of the run^{3,4} but that CK may remain elevated for at least 96 hours³. All four bodybuilders had last trained about 72 hours before the test exercise. Their markedly increased CK at the start of the test may therefore be due to the previous

period of muscle trauma induced by exercise. This marked elevation of CK before exercise (median concentration nine times the upper limit of reference range) may be a direct effect of the anabolic steroids. When they were not taking steroids, their initial CK was only about one and a half times the upper limit of reference range.

The steroids may have affected the integrity of muscle cell membranes, and the stress of exercise may have exacerbated the loss of intracellular substances such as CK and AST. Another possibility is that the effect of steroid abuse may be indirect and due to an increase in the user's aggression. The bodybuilders may then be able to sustain a higher level of exercise which could cause muscle damage.

This study shows that essentially most metabolic changes in bodybuilders who use steroids are similar to those who do not with a metabolic acidosis but little change in glucose. However, the striking feature is the marked elevation of CK. This phenomenon merits further investigation.

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