Resistive Training Improves Insulin Sensitivity after Stroke

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Background: Insulin resistance is highly prevalent after stroke, contributing to comorbid cardiovascular conditions that are the leading cause of death in the stroke population. This study determined the effects of unilateral resistive training (RT) of both the paretic and nonparetic legs on insulin sensitivity in stroke survivors. *Methods:* We studied 10 participants (mean age 65 ± 2 years; mean body mass index 27 ± 4 kg/m^2) with hemiparetic gait after remote (>6 months) ischemic stroke. All subjects underwent 1-repetition maximum (1-RM) strength testing, 9 had an oral glucose tolerance test (OGTT), and 7 completed a 2-hour hyperglycemic clamp (with glucose elevation targeted at 98 mg/dL above baseline fasting level) before and after 12 weeks (3×/week) of progressive, high repetition, high-intensity RT. Body composition was assessed by dual-energy x-ray absorbtiometry in all participants. *Results:* Leg press and leg extension 1-RM increased in the paretic leg by 22% ($P < .05$) and 45% ($P < .01$), respectively. Fasting insulin decreased 23% ($P < .05$), with no change in fasting glucose. The 16% reduction in OGTT insulin area under the curve (AUC) across training was not statistically significant ($P = .18$). There was also no change in glucose AUC. First-phase insulin response during the hyperglycemic clamp (0-10 minutes) decreased 24% ($P < .05$), and second-phase insulin response (10-120 minutes) decreased 26% ($P < .01$). Insulin sensitivity increased by 31% after RT according to clamp calculations ($P < .05$). *Conclusions:* These findings provide the first preliminary evidence that RT may reduce hyperinsulinemia and improve insulin sensitivity after disabling stroke. **Key Words:** Diabetes—energy metabolism—exercise—rehabilitation—stroke recovery.
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Insulin resistance[1] and abnormal glucose metabolism[2] are prevalent during the subacute and chronic phases of stroke recovery, predisposing survivors to macrovascular[3,4] and microvascular complications[5] that worsen morbidity and mortality.[6] Treadmill training decreases hyperinsulinemia and improves glucose tolerance in stroke survivors with impaired glucose tolerance.[7] Although aerobic exercise rehabilitation in stroke may improve metabolic outcomes and other factors linked to cardiovascular and recurrent stroke risk,[7-10] no studies have assessed the effects of resistive training (RT) on glucose metabolism in chronically disabled stroke participants.

We and others have previously shown that RT improves insulin action in nonstroke populations,[12-15] with RT having recently been recognized by the American College of Sports Medicine for this purpose.[16] Quantitative[17,18] and qualitative changes[19,20] occur in skeletal muscle with RT and partially account for the observed

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metabolic effects. We recently reported that RT results in both paretic and nonparetic thigh skeletal muscle hypertrophy after stroke.[11] Importantly, relative adaptation in skeletal muscle quantity after stroke[11] was comparable to that observed in age-matched individuals from previous investigations.[18] Therefore, it is reasonable to hypothesize that systemic metabolic adaptations are possible after stroke with this form of intervention.

The purpose of the current study was to assess for the first time whether progressive lower-body RT reduces hyperinsulinemia and improves insulin sensitivity in chronically disabled stroke survivors.

Methods

Subjects

Participants were recruited from the University of Maryland Medical System and the Baltimore Veterans Affairs (VA) Medical Center referral networks. Chronic hemiparetic stroke patients (>6 months) had completed all conventional physical therapy and had mild to moderate hemiparetic gait deficits, defined as asymmetry of gait with reduced stance, or reduced stance and increased swing in affected limb, with preserved capacity for ambulation with assistive device (e.g., a walker or cane) or standby aid. Baseline evaluations with medical history and examination excluded those with heart failure, unstable angina, peripheral arterial occlusive disease, diabetes, and aphasia, operationally defined as the incapacity to follow 2-point commands. Patients were also excluded for dementia, untreated major depression, and orthopedic or chronic pain conditions. This study was approved by the Institutional Review Board of the University of Maryland and the Baltimore VA Research and Development committee. Written informed consent was obtained from each participant.

Testing

Of the 10 stroke subjects completing the intervention, 9 had pre– and post–oral glucose tolerance tests (OGTT), 7 completed both hyperglycemic clamp tests, and all had 1-repetition maximum (1-RM) strength, peak oxygen consumption (VO_2 peak), self-selected walking speed (SSWS), and dual-energy x-ray absorptiometry (DXA) tests before and after the intervention. One subject completed the hyperglycemic clamp testing but not OGTT testing.

Strength

A 1-RM strength test was conducted for leg press and leg extension on each leg. Two familiarization sessions were included before baseline 1-RM testing to avoid the confounding effects of learning on baseline strength measures. Strength in the paretic and nonparetic legs was tested separately using pneumatic RT equipment built for single-leg movement (Keiser, Fresno, CA).

VO_2 Peak

Exercise testing with open-circuit spirometry was conducted to measure VO_2 peak during a graded treadmill test as previously described.[21]

SSWS

The 30-foot walk test is widely recognized as a valid index of mobility recovery after stroke and simulates the distance required for many home-based activities of daily living functions. Gait velocity was determined from a self-selected pace, with participants using the same assistive device and or/orthoses as normally used to walk across a room at home. Before the test, participants were positioned several steps behind an orange cone to avoid timing the acceleration period. The tester then initiated the walks using the "ready, set, go" command. The stopwatch was started when the participant's toe crossed the first (closest) cone. The timer was stopped when the participant's heel crossed the end of the cone that was lined up with the measured distance. Participants were instructed to walk several steps beyond the second cone to avoid the confounding effects of deceleration in the timing.

DXA

Fat mass, lean tissue mass, and percentage of body fat were determined by DXA scans (Prodigy LUNAR GE, version 7.53.002; GE Healthcare, Little Chalfont, UK).

Glucose Metabolism

The OGTT was performed after a 12-hour overnight fast to measure glucose tolerance and the total glucose and insulin response to an oral glucose load. After 2 baseline venous blood samples (5 mL each) were drawn from an antecubital catheter, participants consumed 75 g of glucose. Subsequent blood samples were drawn every 30 minutes for 2 hours. All samples were analyzed for glucose (glucose oxidase method; YSI Analyzer, YSI Life Sciences, Yellow Springs, OH) and insulin by radioimmunoassay (Millipore, Billerica, MA). Plasma samples were frozen (−70°C) and analyzed in duplicate in the same assay to avoid interassay variability.

Insulin Sensitivity

Peripheral tissue sensitivity to endogenously secreted insulin and β-cell sensitivity to glucose were measured before the intervention and 24 hours after the last exercise session using the hyperglycemic clamp technique.[22] One intravenous catheter was placed in a vein in the antecubital fossa to infuse the dextrose (maintaining plasma glucose within 10% of the targeted glucose level, which was 98 mg/dL above baseline fasting level). A second catheter was inserted in a retrograde fashion into a dorsal

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