Femoral Blood Flow and Cardiac Output during Blood Flow Restricted Leg Press Exercise

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Abstract

Low load blood flow restricted resistance exercise (LBFR) causes muscle hypertrophy that may be stimulated by the local ischemic environment created by the cuff pressure. However, local blood flow (BF) during such exercise is not well understood. **PURPOSE:** To characterize femoral artery BF and cardiac output (CO) during leg press exercise (LP) performed at a high load (HL) and low load (LL) with different levels of cuff pressure. **METHODS:** Eleven subjects (men/women 4/7, age 31.4±12.8 y, weight 68.9±13.2 kg, mean±SD) performed 3 sets of supine left LP to fatigue with 90 s of rest in 4 conditions: HL (%1-RM/cuff pressure: 80%/0); LL (20%/0); $LBFR_{DBP}$ (20%/1.3 x diastolic blood pressure, BP); $LBFR_{SBP}$ (20%/1.3 x supine systolic BP). The cuff remained inflated throughout the LBFR exercise sessions. Artery diameter, velocity time integral (VTI), and stroke volume (SV) were measured using Doppler ultrasound at rest and immediately after each set of exercise. Heart rate (HR) was monitored using a 3-lead ECG. BF was calculated as VTI x vessel cross-sectional area. CO was calculated as HR x SV. The data obtained after each set of exercise were averaged and used for analyses. Multi-level modeling was used to determine the effect of exercise condition on dependent variables. Statistical significance was set *a priori* at $p < 0.05$. **RESULTS:** Artery diameter did not change from baseline. BF increased ($p<0.05$) after exercise in each condition except $LBFR_{SBP}$ in the order of HL (12.73±1.42 cm^3,mean±SE) > LL (9.92±0.82 cm^3) > $LBFR_{DBP}$ (6.47±0.79 cm^3) > $LBFR_{SBP}$ (3.51±0.59 cm^3). Blunted exercise induced increases occurred in HR, SV, and CO after LBFR compared to HL and LL. HR increased 45% after HL and LL and 28% after LBFR ($p<0.05$), but SV increased ($p<0.05$) only after HL. Consequently, the increase ($p<0.05$) in CO was greater in HL and LL (~3 L/min) than in LBFR (~1 L/min). **CONCLUSION:** BF during $LBFR_{SBP}$ was ⅓ of that observed in LL, which supports the hypothesis that local ischemia stimulates the LBFR hypertrophic response. As the cuff did not compress the artery, the ischemia may have occurred because of the blunted rise in CO or because arterial BP cannot overcome the cuff pressure. As $LBFR_{DBP}$ effectively reduced BF and CO with cuff pressures less than systolic BP, future studies should investigate the hypertrophic potential of LBFR at even lower cuff pressures.