Duration and Amplitude Decay of Acute Arterial Leg Inflow Enhancement with Intermittent Pneumatic Leg Compression: An Insight into the Implicated Physiologic Mechanisms

Konstantinos T. Delis, MD, MS, PhD and Alison L. Knaggs
St Mary's Hospital, Imperial College School of Medicine, London, UK and the Division of Vascular Surgery, Mayo Clinic, Rochester, MN

J Vasc Surg 2005;42:717-25

Purpose: By acutely enhancing the arterial leg flow, intermittent pneumatic leg compression (IPC) improves the walking ability, arterial hemodynamics, and quality of life of claudicants. We quantified the duration of acute leg inflow enhancement with IPC of the foot (IPC_{foot}), calf (IPC_{calf}), or both ($IPC_{foot+calf}$) and its amplitude decay in claudicants and controls in relation to the pulsatility index, an estimate of peripheral resistance. These findings are cross-correlated with the features of the three implicated physiologic mechanisms: (1) an increase in the arteriovenous pressure gradient, (2) suspension of peripheral sympathetic autoregulation, and (3) enhanced release of nitric oxide with flow and shear-stress increase.

Methods: Twenty-six limbs of 24 claudicants with superficial femoral artery occlusion or stenoses (>75%) and 24 limbs of 20 healthy controls matched for age and sex, meeting stringent selection criteria, had their popliteal volume flow and pulsating index (peak-to-peak velocity/mean velocity) measured with duplex scanning at rest and upon delivery of IPC. Spectral waveforms were analyzed for 50 seconds after IPC per 5-second segments. The three IPC modes were applied in a true crossover design. Data analysis was performed with the Page, Friedman, Wilcoxon, Mann-Whitney and $_X^2$ tests.

Results: The median duration of flow enhancement in claudicants exceeded 50 seconds with IPC_{foot}, IPC_{calf}, and $IPC_{foot+calf}$ but was shorter ($P < .001$) in the controls (32.5 to 40 seconds). Among the three IPC modes, the duration of flow enhancement differed ($P < .05$) only between IPC_{foot} and $IPC_{foot+calf}$. After reaching its peak within 5 seconds of IPC, flow enhancement decayed at rates decreasing over time (trend, $P < .05$, Page test), which in both groups were highest at 5 to 20 seconds, moderate at 20 to 35 seconds, and lowest at 35 to 50 seconds ($P < .05$, Friedman test). Baseline and peak flow with all IPC modes was similar between two groups. Pulsatility index attenuation in claudicating limbs lasted a median 32.5 seconds with IPC_{foot}, 37.5 seconds with IPC_{calf}, and 40 seconds with $IPC_{foot+calf}$, duration of pulsatility index attenutaion was shorter in the control limbs with IPC_{foot} (30 seconds), IPC_{calf} (32.5 seconds), or $IPC_{foot+calf}$ (35 seconds), yet differences, as well as those among 3 IPC modes, were not significant.

Conclusion: Leg inflow enhancement with IPC exceeds 50 seconds in claudicants and lasts 32.5 to 40 seconds in the controls. Peak flow occurs concurrently with maximal pulsatility index attenuation, within 5 seconds of IPC. Irrespective of group or IPC mode, the decay rate (%) of flow enhancement is highest within 5 to 20 seconds of IPC, moderate at 20 to 35 seconds, and lowest at 35 to 50 seconds. Since attenuation in

peripheral resistance terminates with the mid time period (20 to 35 seconds) of flow decay, and nitric oxide has a half-life of <7 to 10 seconds, the study's data indicate that all implicated physiologic mechanisms (1, 2 and 3) are likely active immediately after IPC delivery (0 to 20 sec) and all but nitric oxide are effective in the mid time period (20 to 35 seconds). As the pulsatility index returned to baseline, the late phase of flow enhancement (35 to 50 seconds) could be attributable to the declining arteriovenous pressure gradient alone.