

Active Voice: High Blood Pressure During Weight Lifting – Damaging to Your Blood Vessels?

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Matthew J. Durand, Ph.D., is an assistant professor in the Department of Physical Medicine and Rehabilitation at the Medical College of Wisconsin in Milwaukee. He learned techniques to measure peripheral vascular function in humans during his postdoctoral fellowship and applied those techniques to examine how exercise-induced increases in blood pressure influenced vasodilator function in athletes and non-athletes. Dr. Durand is currently conducting studies examining how reduced peripheral blood flow impacts recovery of motor and muscle function in post-stroke subjects.

*This commentary presents the authors' views on the topic of their research article that they had published with other colleagues. Their article appears in the April 2017 issue of *Medicine & Science in Sports & Exercise*® (MSSE).*

In 1985, J.D. MacDougall and his co-investigators published the remarkable finding in the [Journal of Applied Physiology](#) that systolic blood pressure surpasses 320 mmHg in body builders who perform a maximal effort leg bench press. Incredibly, in one subject, a systolic blood pressure of 480 mmHg was observed. Considering these stunning findings, and given the well-known relationship between blood pressure and vascular endothelial function, our group has conducted a series of studies to examine the effects of similar maximal effort resistance exercises on peripheral vascular function both in athletes and non-athletes.

[The first of these studies](#), by J.W. Jurva, M.D., S.A. Phillips, Ph.D., and others in the laboratory of David D. Gutterman, M.D., surprisingly showed that a single session of maximum effort leg bench press had no effect on peripheral vascular function in weight lifters. They measured peripheral vascular function as a change in brachial artery flow-mediated dilation (FMD) before and after weight lifting. Conversely, when young, healthy, sedentary subjects performed the same maximal-effort exercise task a dramatic reduction in FMD was observed despite a similar increase in blood pressure as recorded in the athletes. This finding indicated that the blood vessels of athletes are conditioned to the detrimental effects of maximum effort weight lifting, while peripheral vascular function is compromised in non-athletes who perform the same task.

In a series of follow-up studies that our group published more recently, we demonstrated that the weight lifting-induced reductions in vascular function also extend to the small blood vessels. These assessments were made in arterioles obtained from gluteal fat pad biopsies, and cannulated vessel studies in the laboratory demonstrated that the microvessels (100-200 micrometers in diameter) also have overt endothelial dysfunction in sedentary subjects following weight lifting.

In [our study, published in the April 2017 issue of *MSSE*](#), we sought to determine if the reduction in vascular function observed in sedentary subjects after weight lifting is actually due to the high systolic pressure itself, or if it could be due to an increase in a circulating neurohumoral factor caused by the exercise task. To make this determination, we protected the brachial artery from the exercise-induced hypertension by having sedentary subjects perform the leg bench press exercise with a blood pressure cuff inflated to 100 mmHg on their upper arm proximal to the site of the brachial artery measurements. By design, the artery would still be exposed to the same circulating factors as the rest of the body, but the distal blood pressure in the cuffed arm would be approximately 100 mmHg lower than the uncuffed arm. We also performed these same studies in conditioned weight lifters.

As the results of our study showed, FMD was maintained in sedentary subjects when the protective cuff was present, indicating it is the high blood pressure which is responsible for reducing FMD in these subjects. Interestingly, augmented FMD was observed in the conditioned weight lifters when the cuff was present, suggesting the presence of an unknown pro-dilatory factor that is masked by the high arterial pressure.

The results of our study further underscore that caution should be undertaken when a previously sedentary person begins a resistance exercise training program. While it remains unclear how long the exercise-induced impairment in vascular function lasts, or how long it takes a person to become “conditioned” to the high pressure stress. Thus, a slow progression toward performing maximum effort resistance exercise is advisable to reduce stress on the cardiovascular system and peripheral vasculature.