

# Can Plasma Volume Affect Arterial Stiffness and Autonomic Modulation Response to Resistance Exercise?



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## Abstract

**Introduction:** Despite the numerous benefits of resistance exercise (RE), stress caused by muscle contractions may cause transient disturbances in the cardiovascular system including arterial stiffness increasing the temporary risk of cardiovascular events. One physiological response which can influence this process but has received little attention from the scientific community is plasma volume (PV).

**Discussion:** Recent reviews point to a complex interaction between autonomic modulation, central and peripheral adjustments triggered by a RE session. After the end of a moderate to high intensity RE session there may be a reduction in PV caused by its influx into the interstitial tissue and consequent decline in stroke volume and venous return. Moreover, there is an increase in sympathetic modulation, and a decrease in parasympathetic modulation. Arterial stiffness is one of the first signs of pathological changes in the arterial wall leading to an increased risk of cardiovascular events. The stiffening of the main arteries of the body can also be negatively affected by PV reduction. The PV reduction would lead to elevation of sympathetic nerve modulation, cardiac workload and reduction in cardiac perfusion. However, depending of the nature of the PV reduction contradictory results emerges and decrease in arterial stiffness was also reported.

**Conclusion:** There is no agreement as to why PV reduction may affect arterial stiffness, and several questions regarding hydration status and RE influence remains to be addressed. Future studies should standardize water intake and control hydration status before and during experimental sessions utilizing simple measures of hydration status.

**Keywords:** Arterial stiffness; Plasma volume; Cardiopulmonary; Heart rate

**Abbreviations:** RE: Resistance Exercise; PV: Plasma Volume; HR: Heart Rate; SNS: Sympathetic Nervous System

## Introduction

It is well established in the literature that the benefits of resistance exercise (RE) for the general physical health of people of all ages go beyond the increase in muscle mass [1]. RE can improve cardiovascular function, reduce risk factors associated with diabetes mellitus, coronary heart disease [2], and high blood pressure [3]. Besides, higher levels of muscle strength are associated with lower risks of mortality [4].

However, despite the numerous benefits of RE, the stress caused by muscle contractions (mainly moderate to high intensity), may cause transient disturbances in the cardiovascular system and arterial stiffness. This response can increase the risk of cardiovascular events such as myocardial infarction and sudden death [5].

In this sense, many of the cardiovascular responses to RE have not been fully elucidated. One physiological response which can

influence this relationship but has received little attention from the scientific community is a reduction of plasma volume (PV) [6].

## Discussion

Plasma volume is an important variable, affecting a vast array of physiological functions during exercise, and some studies indicate that changes in its composition may influence cardiovascular system adjustments and autonomic modulation after the exercise.

After the end of a moderate to high intensity RE session, there may be a reduction in plasma volume caused by its influx into the interstitial tissue [7] with a consequent reduction in stroke volume and venous return. This process would result in a deactivation of cardiopulmonary receptors with subsequent increase in heart rate (HR).

These changes in PV can at least partially explain the behavior of autonomic modulation. Rezk et al. [8] for instance, observed increased HR and stroke volume reduction after high and low intensity RE sessions. According to the authors, reduction in PV was responsible for triggering this response accompanied by an increase in sympathetic modulation, and a decrease in parasympathetic modulation.

In this context, recent reviews point to a complex interaction caused by a RE session between autonomic modulation, central (heart rate and myocardial contractility) and peripheral (i.e., vascular resistance) adjustments [9].

According to Buchheit et al. [10], arterial baroreceptors are dependent on the levels of atrial pressure and stroke volume with on the other hand is affected by plasma volume. A decrease in plasma volume would exert an inhibitory influence on the receptors of the parasympathetic cardiovascular control centers in the medulla and cause an excitatory effect on the sympathetic nervous system (SNS) with consequent elevation of the HR [11,12].

It is worth mention that during a typical RE session in thermoneutral conditions (temperature 25o C and humidity 50%), the loss of fluids due to perspiration/sweat and respiration is considered negligible. The body weight changes caused by dehydration after most RE studies are far less than those observed during aerobic exercise studies [13]. Therefore, the mechanisms for the changes in PV during resistance exercise are not due to loss of body water to the degree as seen with aerobic exercise.

Rodriguez et al. [14] hypothesized that during high intensity RE the influx of plasma to the active muscle seems to occur due to an imbalance in hydrostatic and osmotic pressure between the capillary terminal and the interstitial tissue of the active muscles. That process can result in an elevated capillary filtration pressure, which facilitates outward filtration of plasma from the intravascular space into the surrounding tissues [15] resulting in reduction of plasma volume in the circulation.

Arterial stiffness typically observed from arterial pulse wave velocity (PWV) evaluation can also be negatively affected by RE [9] and hydration status [16]. PWV is considered an independent predictor of coronary heart disease, stroke, and mortality [17], reduced arterial stiffness is typically perceived as beneficial because it indicates a more compliant artery conversely, increased arterial stiffness is one of the first signs of pathological changes in the arterial wall leading to an increased risk of cardiovascular events [18].

There have been numerous consensus guidelines proposing protocols to standardize the measurement of arterial stiffness/PWV and thereby ensure high-quality research, nonetheless, no one up to date has considered the influence of plasma volume.

Figuroa et al. [9], pointed out a linear relationship between exercise intensity x PWV during and shortly after the end of

a RE session. As the intensity of exercise increases, there is a concomitant increase in blood pressure and PWV. This is important because arterial stiffness increase is strongly associated with elevated sympathetic nerve modulation, cardiac workload and reduction in cardiac perfusion thus increasing the future risk of left ventricular hypertrophy and myocardial ischemia.

Regarding arterial stiffness and hydration, there are inconsistent findings. In a recent study from our group, water intake hindered arterial stiffness caused by high intensity RE [14]. Alternatively, demonstrated that mild dehydration (reduction of 2% of body mass) caused by passive heat, provoked a reduction in central PWV when compared to euhydration.

The contradictory results can be partially explained by the different nature of plasma volume reduction, as mentioned before, during a RE session there is an increase in blood pressure that would lead to PV reduction. On the other hand, during heat stress, vasodilation occurs in the periphery to increase blood flow to the skin in attempt to maximize heat loss. Overall, heat stress acts as an arterial vasodilator, much like nitrates, which reduces stiffness of the peripheral, but not central, arteries.

There is no agreement as to why PV reduction may affect arterial stiffness; however, Arnaoutis et al. [19] verified that in healthy young adults, dehydration reduced endothelial function, and reductions in endothelial function are strongly correlated with increased arterial stiffness. Furthermore, blood pressure also seems to influence the measurement of arterial stiffness; hypovolemia can acutely reduce blood pressure [12] and thereby reduce arterial stiffness.

Despite those contradictions, reduction of PV seems to play an important role as one of the mechanisms of arterial stiffness changes and as well as an intervening variable to cardiovascular response to RE.

### Conclusion

Up to the present date several questions regarding hydration status and RE influence on arterial stiffness remains to be addressed. In that sense, Future investigations are necessary to elucidate the mechanisms and variables influencing this process. Also, it is desirable to develop strategies to hinder the increased autonomic modulation and arterial stiffness response to RE exercise minimizing the risk of cardiovascular events. We strongly suggest that future studies should standardize water intake and control hydration status before and during experimental sessions utilizing simple measures such as urine specific gravity and body weight.

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