Summer 2013

The Effect of Shear Stress, Potassium, and Adenosine on α-1 Adrenergic Vasoconstriction of Rat Soleus Feed Arteries

Tanner J. Heckle  
*Pepperdine University*

Jeffrey Jasperse  
*Pepperdine University*

Follow this and additional works at: [http://digitalcommons.pepperdine.edu/sturesearch](http://digitalcommons.pepperdine.edu/sturesearch)

Part of the [Biology Commons](http://digitalcommons.pepperdine.edu/pepperdinecommons/biology), [Cardiovascular System Commons](http://digitalcommons.pepperdine.edu/pepperdinecommons/cardiovascular), and the [Exercise Science Commons](http://digitalcommons.pepperdine.edu/pepperdinecommons/exercise)

**Recommended Citation**

Heckle, Tanner J. and Jasperse, Jeffrey, "The Effect of Shear Stress, Potassium, and Adenosine on α-1 Adrenergic Vasoconstriction of Rat Soleus Feed Arteries" (2013). Pepperdine University, *All Undergraduate Student Research*. Paper 60.  
[http://digitalcommons.pepperdine.edu/sturesearch/60](http://digitalcommons.pepperdine.edu/sturesearch/60)

This Research Poster is brought to you for free and open access by the Undergraduate Student Research at Pepperdine Digital Commons. It has been accepted for inclusion in All Undergraduate Student Research by an authorized administrator of Pepperdine Digital Commons. For more information, please contact Kevin.Miller3@pepperdine.edu.
During exercise, blood flow increases to the working skeletal muscle primarily because of dilation of the arteries and arterioles feeding the muscle. Sympathetic nerve activity also increases during exercise, augmenting the release of the neurotransmitter norepinephrine (NE) at the arterial wall and into the blood. NE acts to constrict blood vessels; however, arteries and arterioles within contracting skeletal muscle dilate despite the increased NE present. This has led to the concept of functional sympathetic (3), the idea that a chemical released from contracting skeletal muscle interferes with NE signaling. NE acts by binding to adrenergic (alpha and beta) receptors, and it is alpha receptors in the arterial wall that cause vasoconstriction (8). While both alpha and beta adrenergic receptors have been found in some vascular beds of some species, there is significant evidence that in rat calf muscles, the response to norepinephrine is mediated solely by alpha receptors (5, 9). Because alpha receptors are the sole respondents to sympathetic signaling, we studied three proposed substances that may interfere with sympathetic signaling at the alpha receptors, thereby mediating sympathetic signaling. There is evidence to suggest that heat and acidosis may partially mediate sympatholysis of alpha receptors (1, 2). This study sought to determine whether increased levels of shear stress, potassium, or adenosine also contribute to sympatholysis. If shear stress, potassium, and adenosine are, in fact, sympatholytic agents, they may reduce the vasoconstriction mediated by the alpha receptors in rat soleus muscle feed arteres. We hypothesized that all three variables would be sympatholytic agents.

**RESULTS**

- **Figure 1:** Shear Stress did not reduce constriction to Phenylephrine. Estimated shear stress values of 0 dyne/cm², 25 dyne/cm², and 250 dyne/cm² were calculated for no, low, and high levels of shear stress, respectively (3). (N=12 arteres from 12 rats).

- **Figure 2:** Potassium did not reduce constriction to Phenylephrine. Potassium concentrations of 3 mM, 7.5 mM, and 10 mM were calculated for no, low, and high levels of potassium, respectively (5). (N=12 arteres from 12 rats).

- **Figure 3:** Adenosine (in vivo concentrations) did not reduce constriction to Phenylephrine. Adenosine concentrations of 0.1 µM, 0.5 µM, and 3 µM were calculated for no, low, and high levels of adenosine concentrations in skeletal muscle feed arteres. (N=12 arteres from 12 rats).

- **Figure 4:** Adenosine (high concentrations) did not reduce constriction to Phenylephrine. Due to the observations in the presence of a 6.6 µM adenosine, higher adenosine concentrations were used in another set of arteres. (N=9 arteres from 9 rats).

**DISCUSSION**

Our data show no significant difference between alpha meditated vasoconstriction in the absence and presence of shear stress, potassium, and adenosine. This data is not consistent with that of Ives et al., who found heat and acidosis attenuated sympathtic vasoconstriction of alpha-adrenergic receptors in human arteries (5, 9). However, this data is consistent with the findings of Thomas et al., who found that soleus muscle constriction did not attenuate sympathetic vasoconstriction. Thomas et al. hypothesized that the ability to fight sympathetic constriction may be dependent upon muscle fiber type (10). Because of this literature, our lab is furthering this study of the three proposed sympatholytic agents by carrying out the same procedures on arterioles from the rat gastrocnemius, a predominantly glycolytic muscle, for comparison with the rat soleus, a predominantly oxidative muscle. Furthermore, we have initiated confirmation of adenosine as a sympatholytic agent in soleus feed arteres to determine if the soleus muscle is capable of fighting sympathetic vasoconstriction.

**CONCLUSIONS**

- The presence of shear stress, potassium, and adenosine did not reduce vasoconstriction to phenylephrine, indicating that these factors are not sympatholytic in soleus feed arteres.
- Because there is evidence that predominantly glycolytic muscles fight sympathetic vasoconstriction, we are currently examining the effect of the three proposed sympatholytic agents on gastrocnemius arteres.

**REFERENCES**


**ACKNOWLEDGEMENTS**

This research was funded by the National Science Foundation, Research Experience for Undergraduates, REU-Site Grant, #DBI-1062721 and the Natural Science Division of Pepperdine University.

I would like to thank Dr. Jeffrey Jasperse for the opportunity and guidance of his lab. Additionally, I would like to thank Blanca Perez (MSU Billings ‘15) and Samara Jasperse (Pepperdine ’16) for their assistance and entertainment throughout this project.