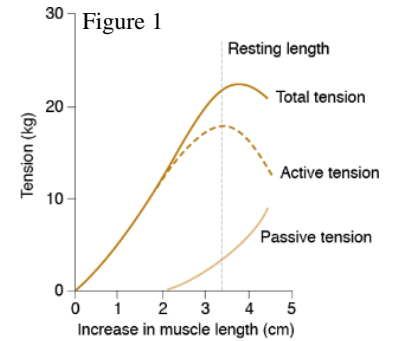


Cardiac Length Tension Curve of the American lobster, *Homerus americanus*

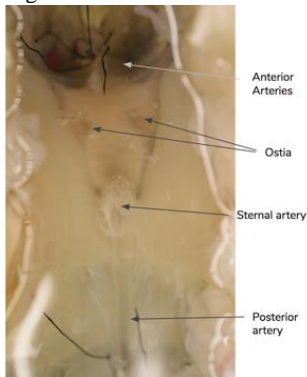
Grace Hamblen, Class of 2021

Hearts respond to increases and decreases in activity by greater or lesser filling with blood, which consequently increases and decreases stretch on the wall of the heart. As the heart wall stretches, the resistance to being stretched increases and the amplitude of the heartbeat increases up to a maximum (Fig. 1: passive and active tension, respectively). That maximum muscle length generally represents the longest functional heart length because at greater lengths the heart would decrease in ability to pump blood. Thus, cardiac muscles function on the ascending portion of the length-tension curve, thereby increasing the amount of blood, or hemolymph (in the case of *H. americanus*), pumped with increasing exertion.



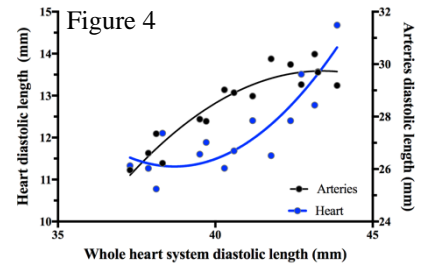
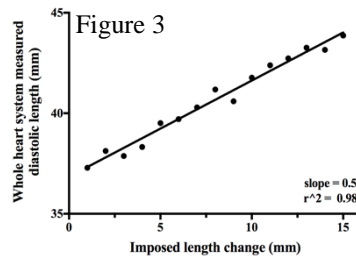
Source: Barrett KE, Barman SM, Boitano S, Brooks HL: Ganong's Review of Medical Physiology: www.accessmedicine.com Copyright © The McGraw-Hill Companies, Inc. All rights reserved.

Figure 2

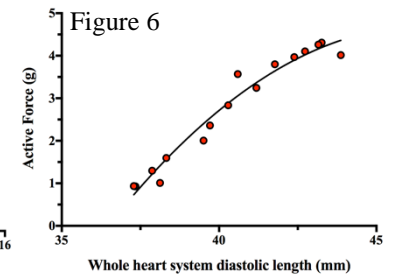
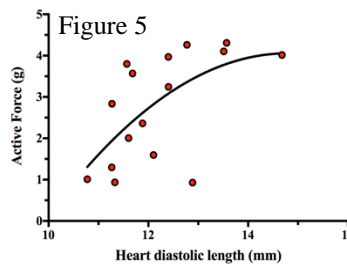


The Johnson lab has been studying the effects of stretch on the heart of *H. americanus* (Fig. 2) by pulling on the five anterior arteries (imposed length change), which lengthens the entire heart/artery system (whole heart system measured diastolic length). Pulling on this heart/artery system is mechanically similar to pulling on differentially stiff springs arranged in series: the least stiff spring will stretch first. To determine the actual stretch on the different parts of this system (anterior arteries, heart, posterior arteries), I videotaped the heart and measured passive and active forces while stretching it. I used Tracker Open Source Physics software to track the lengths of each part of the system during stretch. Example results are given below for one lobster (6/27/18).

While there was a linear relationship between imposed and actual stretch of the entire system (heart plus anterior and posterior arteries)(Fig. 3; linear regression analysis), most of the initial stretch was absorbed by the arteries (Fig. 4, black line and symbols, right axis), and most of the final stretch was absorbed by the heart (Fig. 4, blue line and symbols, left axis; data fit with 2nd order polynomials). The heart did not start stretching until the whole heart system reached about 40 mm diastolic length.



In terms of active force, both the heart length-tension curve (Fig. 5) and the whole heart system length tension curve (Fig. 6) follow the expected pattern for a cardiac muscle (compare Figs. 5 & 6 to Fig. 1; data fit with 2nd order polynomials): both show increasing active force with increasing length, up to a maximum of around 4 grams. Surprisingly, the increase in active force occurred at whole heart system lengths where only arteries were absorbing the imposed stretch (Compare Figs. 4 & 6). These data suggest (1) that at shorter lengths the arteries are less stiff than are the heart muscles (the same pattern is seen in systole), (2) that there is potentially a role for neural pressure receptors in the heart associated with the arteries, perhaps similar to the vertebrate arterial baroreceptors that regulate blood pressure.



Faculty Mentor: Amy Johnson and Olaf Ellers

Funded by: Bowdoin Life Sciences Research Fellowship and INBRE