Cardiovascular Response to Isometric Handgrip Efforts

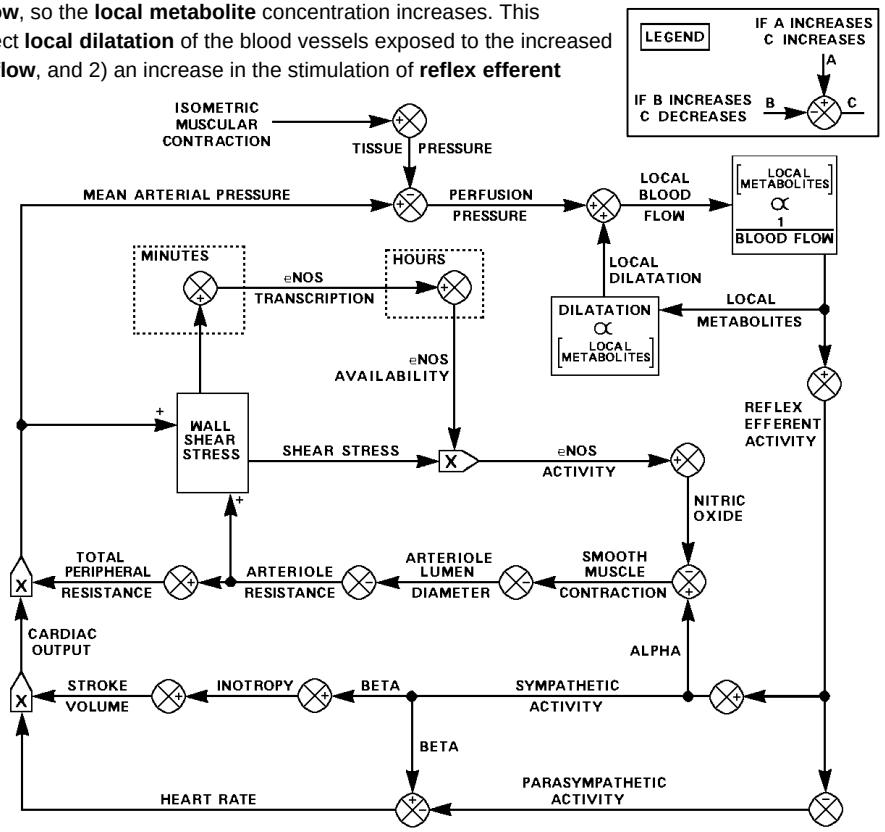
When you squeeze and hold an ***isometric*** hand contraction, you restrict blood flow to your forearm. Your body responds to this by driving up both systolic and diastolic pressure, to try to get the blood flowing in that arm again. But it can't get it flowing, because you are still holding the effort, and so blood pressures continue to rise as long as you hold the effort. The rest of your body, that does not need the extra blood flow from the extra pressure, shuts down; the blood vessels constrict to help divert the blood flow to the one muscle that needs it the most. So, both pressures are driven up, so the average blood pressure goes up, and your whole body is exposed to this cardiovascular stress/exercise.

This is different from ***dynamic or rhythmic*** exercise, where systolic pressure is driven up, but diastolic pressure stays the same, or goes down because your blood vessels dilate enough at each muscle to maintain proper blood flow. During this type of exercise your average blood pressure stays about the same during the effort.

It is the increase in average blood pressure, during an ***isometric*** effort, that causes an *increase* in the average shear stress in your arteries during the effort. This increase in shear stress is what conditions the body, over time, to be more efficient in regulating blood flow.

The principal mechanisms available for blood pressure regulation in response to an isometric muscular contraction

When an **isometric muscular contraction** is applied, and held, **tissue pressure** increases; this causes a decrease in **perfusion pressure** of blood to that working muscle. **Blood flow** to the working muscle also decreases, since it depends positively on the **perfusion pressure**. The concentration of **local metabolites** depends on the reciprocal of **blood flow**, so the **local metabolite** concentration increases. This increase in **metabolites** causes two actions to occur: 1) direct **local dilatation** of the blood vessels exposed to the increased **metabolites**, therefore causing an increase in **local blood flow**, and 2) an increase in the stimulation of **reflex efferent activity** of nerves from the local muscle bed. This stimulation to the central nervous system is an indication of a lack of blood flow to the particular working muscle. The central nervous system responds in two ways: 1) by increasing **sympathetic activity** and 2) by decreasing **parasympathetic activity**. The decrease in **parasympathetic activity** causes an increase in **heart rate**, since **heart rate** depends negatively on **parasympathetic activity**.



The increase in **sympathetic activity** causes three changes: 1) an increase in **smooth muscle contraction**, 2) an increase in heart **inotropy** (muscle contractility), and 3) an additional increase in **heart rate**. The increase in **inotropy** increases the **stroke volume** (volume of blood ejected on each heartbeat). The **cardiac output** (volume of blood pumped per unit of time) depends on the product of the **stroke volume** and the **heart rate**, both of which have been increased, and so the **cardiac output** necessarily increases. The increase in **smooth muscle contraction** causes a decrease of **arteriole lumen diameters,** which causes an increase in **arteriole resistance** throughout the entire body, which increases **total peripheral resistance** (**TPR**).

Mean arterial pressure (**MAP**) is the product of the **cardiac output** and the **TPR**. Since both **cardiac output** and **TPR** are increased, **mean arterial pressure** necessarily increases. The increased **arterial pressure** then aids in increasing (restoring) the **perfusion pressure** to the working muscle which was being restricted in the isometric effort. This completes the 'feed back loop' for regulating blood flow to the working muscle, in an attempt to keep the concentration of **metabolites** at a minimum level. However, the increase in **blood flow** provided by the increase in **mean arterial pressure**, and due to the direct **local dilatation**, is not sufficient to reduce the **metabolite** concentration during a muscular contraction greater than 15% of maximum voluntary contraction (MVC). In this case, the feedback loop continues, and arterial pressure keeps increasing for as long as the muscular contraction is held. This continuing increase in arterial pressure is termed "pressor response".

During a pressor response, the **wall shear stress** in arterioles is driven up due to the constriction of the diameter and the increased **mean arterial pressure** forcing blood through the vessels. The **wall shear stress** is a measure of the frictional force exerted on the moving blood by the endothelial cells, which line the inner surface of blood vessels. **Endothelial nitric oxide synthase** (**eNOS**) is **activated** by this increased **shear stress**, and causes the release of **nitric oxide** (**NO**) from the endothelial cells. The **NO** acts as a vasodilator, which means that it reduces **smooth muscle contraction**. This in turn increases the **lumen diameter**, reduces **arteriole resistance**, and causes a decrease in **wall shear stress**, thus forming another 'feed back loop'. This activity of **eNOS** attempts to maintain the **wall shear stress** at a constant level.

When the **wall shear stress** is elevated for prolonged periods, minutes, signaling is initiated which leads to an increase in **eNOS transcription**. This means that the endothelial cells begin the genetic transcription process, to increase production of the enzyme **eNOS**. After many hours, the quantity of eNOS begins to increase, and the **eNOS availability** is thus higher. Since the **eNOS activity** is the product of the **wall shear rate** and the **eNOS availability**, more **NO** is released in response to **wall shear stress**. This increased **eNOS activity** changes the 'set point' for the regulation of **wall shear stress** to a new lower value. For the same **wall shear stress**, more **NO** is released, **smooth muscle contraction** will be less, **lumen diameters** will be higher, and **arteriole resistance** will decrease. This not only lowers the set point for **wall shear stress**, but also lowers **total peripheral resistance** and therefore lowers **mean arterial pressure**, assuming cardiac output does not change.