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Muscle metaboreflex-induced central blood volume mobilization in heart failure

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BACKGROUND
During dynamic exercise in normal subjects, muscle metaboreflex activation (MMA) caused by under perfusion of skeletal muscle triggers large increases in cardiac output that occur via increases in heart rate, ventricular contractility, and, marked central blood volume mobilization that acts to maintain ventricular preload. This allows the changes in cardiac function to maintain the increases in cardiac output. In subjects with heart failure, the ability to raise cardiac output during MMA activation is impaired.

OBJECTIVE
To find whether the inability to increase CO during MMA is due to an inability to maintain ventricular preload.

STUDY
Study model: conscious chronically instrumented canines (n=5; weight 21-26 kg, 2 female, 3 male).
Three surgeries conducted with a 10-14 day interval between surgeries to:
1. Implant a 20 PAU Transonic Flowprobe around the ascending aorta to measure cardiac output (CO);
2. Implant a 10 PAU Flowprobe around the terminal aorta to measure hind limb blood flow; to position two perivascular hydraulic occluders around the terminal aorta distal to the Flowprobe to gradually reduce hind limb blood flow; to introduce a 19 G catheter through a ligated lumbar artery and secure it to the terminal aorta cranial to the Flowprobe to measure mean arterial pressure (MAP);
3. Introduce a catheter into the jugular vein and advance it to the arterial-caval junction to measure central venous pressure (CVP).
Flow probe cables, vascular occluders and catheters were then tunneled subcutaneously and exteriorized between the scapula.
Experiments were conducted one week after the final surgery after the animals had fully recovered.
• Animals were allowed to roam freely for 15 minutes before being led to a treadmill.
• There Transonic Flowprobes were connected to Transonic Flowmeters and the catheters were connected to Transpac IV pressure transducers.
• Data were collected during mild exercise on the treadmill with unrestricted blood flow to the hind limbs, and then during increased MMA activation through graded reductions in hind limb blood flow via inflations of the occluders.
• Each dog completed a series of experiments during mild exercise with normal cardiac function; during mild exercise with constant heart rate; during mild exercise with constant heart rate plus limited contractility and during heart failure induction.

RESULTS
MMA in this heart failure model was able to raise central blood volume similarly to normal subjects.

CONCLUSION
The loss of the ability to raise CO during MMA during heart failure is not because of the loss of the ability to mobilize blood volume centrally. Because the MMA reflex was still able to elicit large increases in central blood volume, the limited ability to raise cardiac output likely stems from ventricular dysfunction and not the ability to maintain preload.

Reference