L-NAME treatment in pigs
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$\text{N}_{\omega}\text{-nitro-L-arginine-methyl-ester (L-NAME)}$ inhibits the enzyme nitric oxide synthase (NOS) which generates the physiologic messenger gas, nitric oxide (NO). In addition to its role as a vasodilator NO inhibits inflammation and vascular smooth muscle cell proliferation. It was shown recently that rats fed L-NAME contain evidence of inflammation and increased collagen in their coronary vasculature when compared with control rats. We hypothesized that L-NAME treatment will cause inflammation and an increase in collagen in the coronary vasculature of pigs compared with control pigs. To test this hypothesis we have four pigs, two received L-NAME in their drinking water and two did not. We have samples of the left ventricle (LV), right ventricle (RV), and left anterior descending (LAD), left circumflex (LCX), and right (RCA) coronary arteries of the heart. Similar to studies in the rat, we will stain these samples for monocyte chemoattractant protein-1 (MCP-1), a marker of inflammation; alpha smooth muscle actin ($\alpha$SMA), a marker of vascular smooth muscle, and picrosirius red (PSR) a marker of collagen. We will photograph sections of the coronary vasculature stained with these markers and use a computer image analysis system to count the amount of MCP-1, vascular smooth muscle, and collagen. Preliminary results suggest that we have insufficient statistical power to show differences in the parameters measured and need to examined greater numbers of animals.