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Ventricular Remodeling of Experimentally Infarcted Rat Hearts

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Abstract—The objective of this study was to look at ways of modifying long term changes in the heart function post myocardial infarction. Specifically, we did this by measuring changes in structure and in function of experimentally infarcted rat hearts. Our main goal was to determine the effects adenosine on the long term ventricular remodeling. Adenosine (an autocooid that can be produced during ischemia or can be administered exogenously) is important because of its therapeutic significance as it is known to reduce the size of an infarct. To test effects of adenosine, particularly as it relates to modifying heart function (and structure), we used an adenosine kinase inhibitor to increase adenosine levels as well as an adenosine receptor antagonist to determine the role of receptors in this process. Thirty eight rats were divided into four groups (1) shams (no infarct) (2) infarct treated with saline (control group) (3) infarct treated with adenosine kinase inhibitor (GP515) to increase adenosine & (4) infarct treated with GP515 + Receptor antagonist (8-SPT). In a span of one month's time all rats were given MI (permanent occlusion) and 48 hours later, were given their respective drug treatments. All rats were given 10 weeks to recover prior to their terminal study. My role in the data analysis was look at the in-vivo hemodynamics (ECG, aortic and left ventricular (LV) pressure, as well as rate of change of LV pressure). After analyzing the aforementioned data, results indicate that the effectiveness of adenosine does not appear to be mediated by adenosine receptors as

the blocking of the adenosine receptors with antagonist did not decrease heart function (as determined by the heart's high LV pressures indicating efficient contractility). Statistical tests, however, will have to be done to determine the significance of results.