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Evaluation of Cardiac Hypertrophy in Spontaneously Hypertensive Rats Using Metabolic Rate of Glucose Estimated From Dynamic MicroPET Image Data

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Alteration in myocardial energy metabolism is believed to be one mechanism involved in the progression of heart failure. Imaging studies have shown that myocardial glucose uptake rates are lower in heart failure and myocardial free fatty acid uptake rates are higher than in the normal heart. Imaging studies have also shown that myocardial fatty acid oxidation begins to be inhibited and that washout of fatty acid increases in the compensated stage of left ventricular dysfunction. In the advanced stage of heart failure the myocardial extraction and retention of fatty acid are further impaired. Therefore, shifts in myocardial glucose and fatty acid use may be an important mechanism for the impaired efficiency of the failing heart and a target for specific therapies designed to decrease overall energy requirements. This study was designed to follow the change in glucose metabolism with the progression of hypertrophy in the spontaneously hypertensive rat (SHR). Dynamic PET data were acquired using the microPET II. A dose of 1 mCi of F-18-FDG was injected into a control Wistar Kyoto (WKY) rat and the same dose was injected into a SHR rat. Each rat was imaged using a dynamic acquisition for 80 minutes acquiring list mode data with cardiac gating of approximately 900 million total counts. Both rats were imaged at 10 week intervals for a total of five imaging sessions. The input function was calculated both using factor analysis of dynamic structures (FADS) and fitting splines from the list mode data. The dynamic data for myocardial tissue regions were fit to a 2-compartment model. Using a single blood sample, the metabolic rate of glucose was evaluated. The study is being used to test the hypothesis that the SHR rat models congestive heart failure and that this model demonstrates changes in metabolism with progression of cardiac hypertrophy.

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