Journal of Nuclear Medicine, **37**(5 suppl):131P, 1996 (preprint)

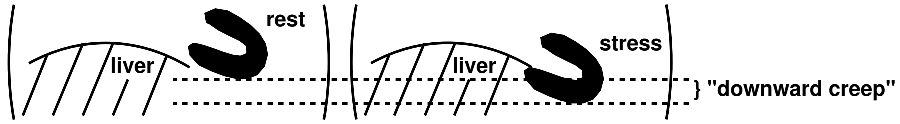
CARDIAC CREEP DURING REST/STRESS MYOCARDIAL PERFUSION STUDIES — PATIENT MOTION AND LUNG AIR REDISTRIBUTION. **BW Reutter, S Lim, RH Huesman, PG Coxson, GJ Klein, and TF Budinger. Center for Functional Imaging, Lawrence Berkeley National Laboratory, University of California, Berkeley, CA.**

To evaluate the impact of cardiac motion on attenuation correction and rest/stress comparisons, we developed 1) a method for spatially registering multislice PET transmission with emission data, which corrects for rigid translational motion and axial rotation of the patient, and 2) a method for evaluating the relative motion of the heart in the chest. The constraint of axial rotation leads to an efficient implementation for computing a compensated transmission sinogram.

Dynamic Rb-82 emission data were acquired on 16 patients on a Siemens/CTI ECAT EXACT HR (47 slice) scanner, with the patients first at rest and then under pharmacologically induced stress (dipyridamole).

Two observers independently registered transmission data with the two sets of summed emission data, using the MPM software package (U Pietrzyk et al., JNM 1994; 35:2011-2018). The summed emission data were also registered with one another. Average motion was 9.7 mm between rest and transmission, 8.5 mm between transmission and stress, and 8.2 mm between rest and stress (average coefficient of variation, 49%).

Anatomical surfaces were segmented automatically from summed rest and stress emission data for three patients, using a differential 3-D edge detection algorithm. Visualization of the surfaces showed that for each patient the motion was largely a "downward creep" of the heart relative to the liver and diaphragm. This was consistent with a subsequent "upward creep" that has observed in post-stress Tl-201 SPECT studies (J Friedman et al., JNM 1989; 30:1718-1722).



Our results suggest that both the motion of the patient as a whole and possible physiological changes in the volume and distribution of air in the lungs can contribute to the gross motion of the heart between rest and stress.

Supported by NIH grant HL25840 and DOE contract DE-AC03-76SF00098.