

THE EFFECTS OF HYPOTHYROIDISM ON SPONTANEOUSLY HYPERTENSIVE HEART FAILURE RAT MODELS

Brenda Simon, Bassel Kisso and A. Martin Gerdes
Mount Marty College and Cardiovascular Research Institute
University of South Dakota and Sioux Valley Hospital
Yankton, SD 57078

ABSTRACT

Recent studies have shown that thyroid dysfunction is an important risk factor associated with heart failure. Clinical case studies suggest that long term hypothyroidism alone can cause heart failure. However, no animal studies have clearly demonstrated this to date. In this study, hypothyroidism was induced in rats with genetic hypertension (SHHF). The major hypothesis being tested is that reduced thyroid function in hypertension will accelerate progression of myocyte remodeling and lead to an earlier onset of heart failure.

Hypothyroid induced atrophy of cardiac myocytes and progression of heart failure was assessed in SHHF (spontaneously hypertensive heart failure) rat models. The effects of induced hypothyroidism through the addition of propylthiouracil, which blocks the production of thyroid hormone, were investigated on lean female SHHF rats. Left ventricular function was determined by echocardiography and hemodynamics. Whole tissue pathology and isolated myocytes size and number were assessed.

PTU treatment caused an increase in LV diastolic chamber diameter of 14%, and an increase in LV systolic chamber diameter of 38%. Heart rate decreased by ~100 beats per minute, left ventricular pressure decreased by ~50mmHG, and there was a decrease in the measurement of contractility over time. Both body weight and ventricular weight decreased. Wall stress increased by 19%.

PTU treatment of SHHF rats resulted in clinical hypothyroidism which resulted in hastening the onset of heart failure. Left ventricular function declined, and myocytes were lost.